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A TEXT-BOOK

ON

NERVOUS DISEASES

BY AMERICAN AUTHORS.

EDITED BY

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PREFACE.

THE prevailing impression that the subject of Nervous Diseases presents peculiar difficulties has but slight foundation in fact. No branch of medicine can be grasped without serious study, and, granting this, the student and practitioner can approach neurology with confidence. Possibly the difficulty is not inherent so much in the subject as in the manner of its presentation, a belief which has led, after careful study, to the somewhat novel arrangement of this work.

In brief, the general affections are considered first, and attention is then progressively directed to those which are more and more special. The advantages of this arrangement are universally recognized in the other sciences, and its applicability to medicine seems hardly to need demonstration. Further, diseases clinically allied have been considered in close connection, and by this means it has been sought to accentuate the practical features of the subject. In building upon this basis attention has also been devoted to the anatomy and physiology of the nervous system, these subjects being incorporated with the consideration of the various diseases. The choice of subjects and the space devoted to each have been arranged with special reference to practical needs, and it is believed that the mode of handling details is conducive to clearness, utility, and completeness.

A glance at the List of Contributors, opposite the title-page, will show that this volume represents the views of writers widely recognized as authorities in neurological science, and especially known in connection with the subjects assigned to them. The work is likewise representative of our great medical schools, and hence it embodies not only high authority, but is likewise illustrative of the best methods of instruction.

Free use has been made of illustrations in black and colors. The series of pictures is largely original, and where authenticity is requisite it has been attained by photographic methods. It is confidently hoped that the other engravings, and especially those which are diagrammatic, will be found full of assistance.

In conclusion, the editor desires to extend his sincere thanks to his colleagues for their recognition of the importance of the work and their earnest support in its production.

F. X. DERCUM.

PHILADELPHIA, September, 1895.

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CHAPTER I.

GENERAL CONSIDERATIONS.

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By S. WEIR MITCHELL, M.D., and F. X. DERCUM, M.D.

For a generation past diseases of the nervous system have attracted an ever-increasing amount of attention. Little by little they have become better understood and have gradually assumed the importance of a well-defined department of medical knowledge. However, unlike such specialties as diseases of the eye and ear, no line of demarcation exists between this department and general medicine. Indeed, the two touch at almost every point and can never be entirely separated. At the same time the field of nervous diseases has become so vast and the details so intricate that special study is absolutely demanded.

Before disease can be intelligently considered, a knowledge of normal structure must first be acquired. Especially is this true of diseases of the nervous system. Here a knowledge of morphology is of supreme importance. Not only must the student be familiar with gross relations and minute details, but also with general principles. To this end his studies should not be confined to man, but should, if possible, include general biology. An understanding of nervous structure is hardly possible without a knowledge of embryology and comparative anatomy. The lessons taught by these sciences make clear much that is otherwise inexplicable and give to the student a foundation for a broad and philosophical grasp of the subject.

The study of the diseases of the nervous system embraces of necessity a study of its pathology. The pathological processes at work are many of them common to the body as a whole. Such, for instance, are the various forms of inflammation, some forms of sclerosis, and several forms of tumor. Again, as in diseases of other organs, morbid processes may affect pri-of nervous diseases heredity frequently plays an important part. This fact lends a peculiar value to the family history. As far as possible, facts bearing upon any diseases from which the parents or other relatives may have suffered should be brought to light. Special attention should, of course, be paid to those which bear directly upon the nervous system. In this connection it is not sufficient merely to inquire whether a grandparent, father or mother, uncle or aunt, brother or sister has been insane, has had epilepsy or other nervous affection, but also to determine whether there be a history of alcoholic excess, of dissipation, of gout, of rheumatism, of syphilis, and even of tuberculosis in the ancestry.

The nervous features presented by family histories are of three kinds. The *first* is illustrated by a class of patients in whom we obtain merely a history of general nervousness, of irritability, of invalidism, or of chronic headaches as existing in this or that member of the family. In other words, we obtain a history which is suggestive, other things equal, of a neurasthenic strain in the ancestry. The *second* is illustrated by a class from which we learn that one or more relatives have suffered from insanity, epilepsy, or organic nervous disease; or there may be a history of suicide, of an occasional feeble-minded or idiotic child, or of strange personal eccentricities. A patient with such a history is more or less liable to serious nervous or mental disease; he is apt to have what is known as "the neuropathic constitution" or the "insane neurosis," as it is also called. There is in such a patient an inherent tendency to certain forms of nervous degeneration. The *third* is illustrated by a class of patients in whose family histories nervous features are apparently lacking, but in their stead various elements are present also potent for ill; among them are alcoholism, the various diathetic diseases, such as gout and rheumatism, and infectious diseases, such as syphilis and tuberculosis. That alcoholism in the ancestors is of grave significance is shown by two facts: first, the admitted physical inferiority of the children of alcoholic parents, and, secondly, the transmission of the alcoholic habit from one generation to another. Diathetic affections, in their turn, are not infrequently related to neurasthenia, migraine, and other functional nervous troubles in the offspring. The importance of syphilis in the ancestry is shown either by the direct transmission of the disease to the fœtus, causing, among other things, an arrest of development, or by the production of a markedly neurasthenic and feeble stock.

As in all other classes of affections, the personal history is also of great importance. The method of eliciting it differs in no respect from that used in studying other diseases. Our inquiries should be especially directed to determine whether the nervous system of the patient has been normal in its development, whether it has shown itself prone to disturbances, and, finally, whether it has been exposed to certain deleterious influences. Thus we inquire in certain cases, as to the time when the child began to talk, what progress it made at school, whether or not at the age of puberty nervous symptoms were present, and whether or not during adolescence the mental and emotional life of the individual was normal. Further, we should inquire as to the history of convulsions in early childhood, of frequently recurring headaches, of neuralgia, or of excessive irritability and nervousness. Finally, we must question our patient as to a history of grave illnesses, of exposure, of injuries, of alcoholic and other excesses. In this connection, too, it is of the greatest importance to determine the existence of syphilitic infection, or, if this be denied, a history of promiscuous exposure. In no other class of diseases does syphilis play so great a *rôle*. Questions directed to determine the presence or absence of this malady are, in organic nervous affections, absolutely essential.

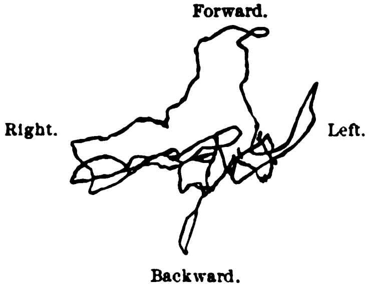
The part played by alcohol is second in importance only to the part played by syphilis. The abuse of tobacco, tea, coffee, and of the various narcotics should also be carefully inquired into. Further, in addition to the toxic agents which the patient voluntarily ingests, there are others which, by being accidental ingredients of food or drink, may profoundly affect the nervous apparatus. This is especially true of the various metallic poisons—*e. g.*, lead and arsenic.

The history of the sexual life of the patient is also of great importance. Sexual excess, and especially abnormalities in the performance of the sexual act (see Chapter II.), are definitely related to various functional and organic nervous diseases. Further, disease of the sexual apparatus, such as strictures of the urethra in men or organic disease of the ovaries and tubes in women, react profoundly upon the nervous system.

Various other facts in the personal history relating to deleterious influences acting on the nervous system could be here enumerated; they are, however, considered in detail in the following chapters.

Next in order is the direct examination of the patient. First, in a general way, we notice peculiarities of posture, of gait, and of general movements; the presence of any gross deformity, of marked paralysis, or the wasting of a limb. We note also the patient's method of talking, his manner of explaining his case, his gestures, and his facial expression. By this means we are often enabled roughly to classify the case with this or that group of nervous affections. Of course, a detailed examination is invariably necessary, and must be made in a systematic manner. Inasmuch as the nervous system is extremely complex, the symptoms produced by its derangement are manifold. They may present themselves as anomalies of motion, of sensation, of reflex action, of nutrition, of intellectual and emotional activity, or as disturbances of various visceral functions.

FIG. 1.



Tracing of the sway in a healthy man. (HINSDALE.) In taking the above tracing a piece of blackened cardboard was fixed in a horizontal position above the head of a normal man standing in the position described in the text. The subject wore a cap, upon the centre of which was mounted a small stylus.

In our detailed examination, we note first whether the patient is able to maintain an upright position without swaying to either side or forward or backward more than normally. In other words, we determine his *station*—*i. e.*, his power of maintaining his equilibrium. As is well known, a healthy man standing erect sways a little to and fro. No one can stand absolutely still. The sway taking place in health has been studied by S. Weir Mitchell and Guy Hinsdale.[1] They found that a man standing with his feet close together, heels and toes apposed, sways forward, backward, and laterally, but that this sway is usually more pronounced in certain directions. "The first movement is almost invariably forward, then there is a counterbalancing effort, bringing the body backward and usually to the right." The more pronounced swaying is in the antero-posterior line. Normal men and women have an average sway of one inch in the forward and backward direction, and about three-quarters of an inch laterally. As is shown in the accompanying tracing (Fig. 1), the *forward sway* is largely in excess of the *backward sway*, and, further, that movement *to the right* is largely in excess of movement to the left; in other words, the normal sway is distinctly *antero-dextral.* As Hinsdale has shown, in left-handed persons the sway is often antero-sinistral. Closing the eyes increases the amount of sway about 50 per cent.

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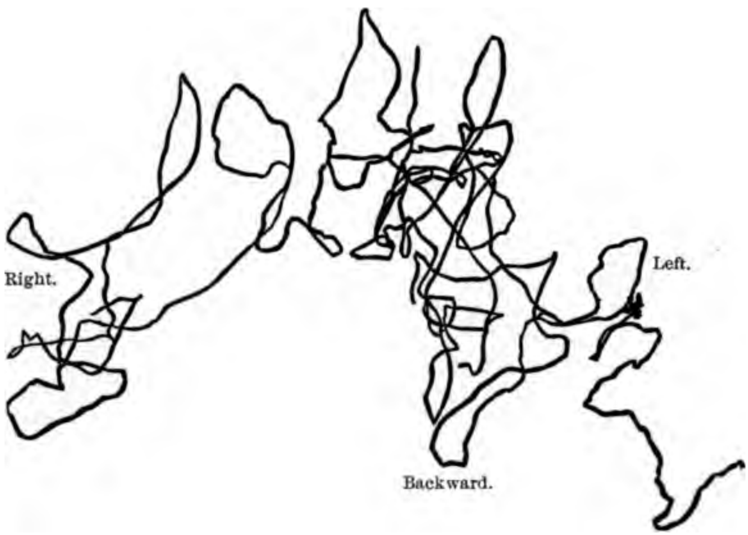
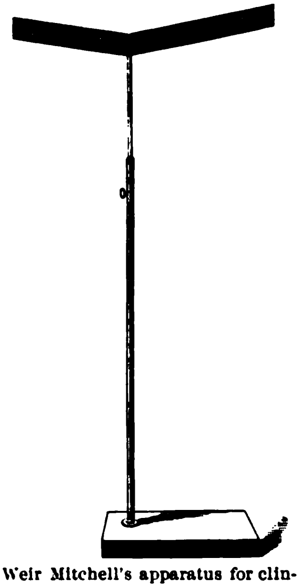
[1] American Journal of the Medical Sciences, April, 1887.

FIG. 2.



Tracing of the sway in a case of locomotor ataxia. (HINSDALE.)

FIG. 3.



Weir Mitchell's apparatus for clinical observations of station.

If the power of maintaining the equilibrium be impaired, swaying becomes exaggerated (see Fig. 2). Further, if the eyes be closed, so that the patient is denied their guidance, this symptom becomes still more marked. In excessive swaying, such as occurs in locomotor ataxia, the patient may even fall while this test is being made. This symptom—*i. e.*, the increased sway upon closure of the eyes—is technically known as the *Romberg symptom.*

We next direct our attention to the other motor symptoms. We begin by studying the *gait.* We note whether the limbs are used as readily and as freely as in health, whether they are weak, whether they are held rigidly and scraped along the ground, or whether they are awkwardly thrown from place to place. The typical abnormal gaits naturally group themselves into the *ataxic gait*, in which the movements are not properly co-ordinated; the *spastic gait*, in which the legs are held very rigidly; and, lastly, the various *gaits of simple weakness*, in which one or both legs are partially paralyzed. It will be best to study briefly the elements of the normal walk, so that the differences between it and the various abnormal gaits will be more readily comprehended.

In walking a series of oscillations are performed by the trunk, and these oscillations take place in three different directions: First, an oscillation in the horizontal direction, the trunk swaying from side to side; secondly, an oscillation in the vertical direction, the trunk rising and falling; and, thirdly, an oscillation in the forward direction, the trunk being propelled forward by a series of regularly recurring impulses. The curves described have been studied in detail by Dercum.[1] In Fig. 4 is traced the oscillation of a point upon the head in a vertical direction. In Fig. 5 is traced the oscillation of the same point in a lateral direction. Fig. 6 represents the rise and fall of the superior spinous process of the ilium, while Fig. 7 represents the lateral oscillation of the same point. These curves are interesting as demonstrating slight differences in the character of the movement in various portions of the body; thus it will be seen at once that the amplitude of the vertical movement of the hip is decidedly greater than that of the head. At the same time it is noted that the lateral sway of the hip is much less than that of the head. These facts are in keeping with what we would expect on *a priori* grounds. The pelvis is relatively fixed and not

FIG. 4.

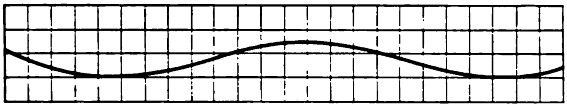


FIG. 5.

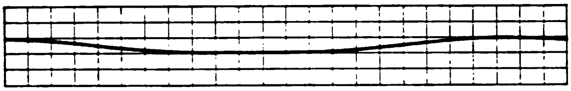


FIG. 6.

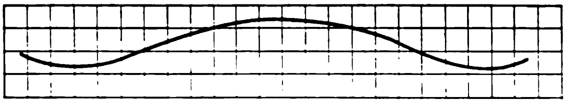
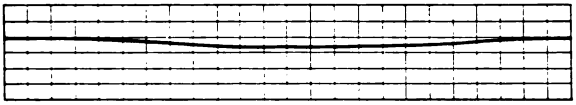


FIG. 7



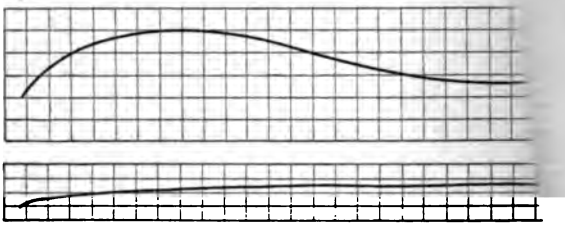
Illustrating the vertical and lateral oscillations of the head and pelvis in the normal walk. Fig. 4 represents the vertical oscillation of the point upon the head. Fig. 5 represents the lateral oscillation of the same point. Fig. 6 represents the vertical oscillation of the superior spinous process of the ilium. Fig. 7 represents its lateral oscillation.

[1] Dercum, F. X.: Transactions of the College of Physicians, Philadelphia, 1887.

nearly so free to move in a lateral direction as t
the trunk; and, again, the tilting of a column is
upper and free end. As a matter of fact, also
more pronounced toward the right than towar
mony with the more pronounced sway to the righ

It is, however, the movement of the leg and f
In Fig. 8 are represented the curves described by

FIG. 8.



Representing the vertical and lateral oscillations of the foot
the external malleolus being taken as a fixed

the course of a single step. The upper line represents
malleolus. It is seen at a glance that this curve is m
elements. The curve begins by the malleolus sweepi
the radius of which centres in the ball of the great toe.
the centre of rotation is transferred to the *tip* of the grea
of the curve up to its highest point is the result of the fi
of the forward movement of the pelvis; thence to the
heel upon the ground the curve is the resultant of a co
which three principal elements are distinguishable: firs
ment; second, a *fall;* and, third, a *forward movement,* th
the movement forward of the body as a whole. The fir
those of a cycloid, and the foot therefore falls to the gr
equal, *along the line of swiftest descent.* A fourth element
slight secondary rise occurring in the curve just previous
The significance of this secondary rise is as follows: We not
the passive leg in swinging forward in its cycloid-like desce
diately strike the ground, but that just previous to the imp
a slight ascent. It is relatively slow in the beginning, but
in velocity until the step is almost completed, when a slow
again takes place. This slowing of movement is coincide
ondary rise (see Fig. 10). The impact of the heel upon th
made without any waste of force and with a minimum
When, in addition, we reflect that the heel is but a pa
lever, and that the muscles attached to this lever are elastic,
the jar of impact is indeed reduced to a minimum.

It is further of interest to note that the weight is rapidly
the outer edge of the foot, and thence again to the ball of
preparatory to the next propulsive effort. A complex rotatory
the foot therefore takes place.

As seen in Fig. 9 and Fig. 10, the rate of motion is not uniform.

With a clear conception of the normal walk before us, the
motor ataxia and of lateral sclerosis, which can be taken as
various ataxic and spastic gaits, become easily understood. In

we will consider first, the lateral sway of the trunk and head is much increased. It is also very irregular and is enormously exaggerated by closure of the eyes. When we come to study the movements of the legs we find that they also are very irregular. When, however, they are closely studied, either in the living subject or, better still, in serial photographs (see Chapter XX.), we note that two factors are constant, namely, an increase in the outward sway of the foot, and, secondly, an increase in the height to which the foot is raised from the ground. The curves or trajectories of the movement of the foot differ therefore strikingly from the corresponding trajectories of the normal walk. This is readily seen by a comparison of the curves of Fig. 11 with those of Fig. 8. The altitude of the vertical trajectory is seen to be very much greater, while the outward sway is also much exaggerated. The vertical trajectory is made up of the same elements as the corresponding line of the normal gait, namely: first, a short curve depending upon the upward rotation of the malleolus upon a radius centring in the ball of the great toe; secondly, a curve depending upon the centre of rotation being transferred to the tip of the great toe. This curve again passes insensibly into one caused by the flexion of the leg on the thigh and the onward movement of the hip. A reference to Fig. 12 will show that the flexion of the leg is much more marked than in the normal walk, and, consequently, the corresponding por-

Fig. 9.

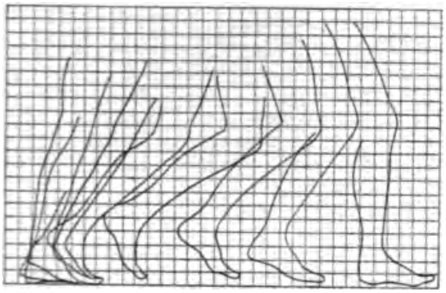


Fig. 10.

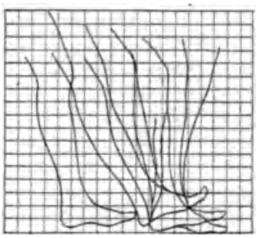
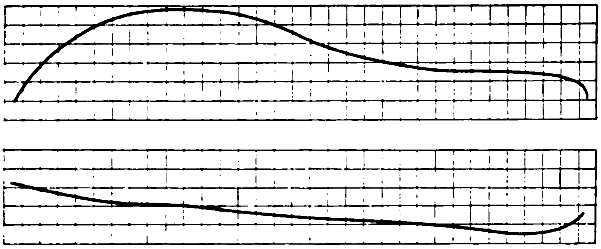


Fig. 9 illustrates the variations in the position of the leg and foot, as also the varying rate of movements, the time interval between any two tracings being always the same.

Fig. 10 illustrates a slight secondary rise and the impact of the heel on the ground.

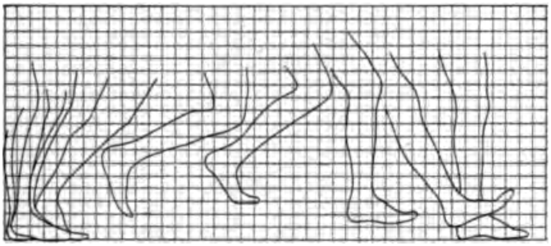
Fig. 11.



Represents the vertical and lateral oscillations of the foot in the case of locomotor ataxia, the external malleolus being taken as a fixed point. The direction of the movement is from left to right.

tion of the curve is higher. Not only is the altitude of the c but the fall of the foot is irregular. Especially do we notice fails to make the slight secondary rise which occurs in the nor before the impact on the ground is made; in fact, the curve su

FIG. 12.

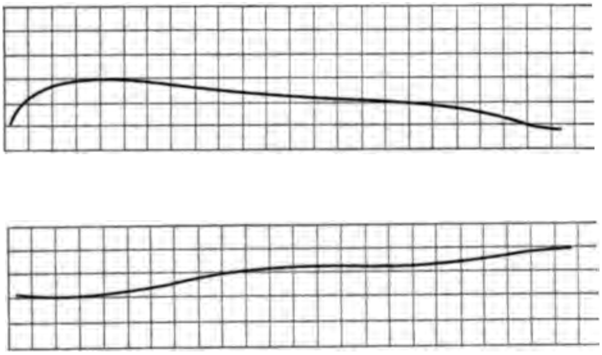


Illustrates the movement and the varying positions of the leg and foot in locomotor ataxia, and also the impact of the heel upon the ground.

as though the impact were made by a sudden descent. Furth note that the outward sway is also increased, we note again th irregular.

When we come to the spastic gait, such as is typified in late we find that the amplitudes of the curves described by the foot This is readily seen by reference to Fig. 14, as well as the curve

FIG. 13.

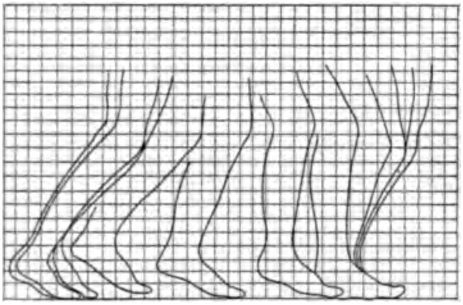


In Fig. 13 are represented the lateral and vertical oscillations of the foot in the sclerosis, the external malleolus being again taken as a fixed point. The directio from left to right.

When we compare the vertical trajectory in this instance with normal walk, Fig. 8, we note at once that the altitude of t much diminished, and we also find that the outward sway to a minimum. In other words, in this gait the leg is held There is but little action at the knee, and the foot is barely, if at from the ground. At the same time, instead of being well abd really adducted. Indeed, the advancing foot at the completion of ment may be brought so far within the median line as to get in its fellow. This crossing of the feet is every now and then seen in

gaits of hysteria. As regards the lateral sway of the trunk in the spastic gait, it is grossly exaggerated, and it always takes place toward the side opposite the advancing leg. It is only by means of this grossly exaggerated sway that the leg can be advanced at all in many cases. The sway tilts the pelvis, and thus assists in raising the foot from the ground. This exaggerated lateral sway seen in the spastic gait differs, however, radically from the excessive sway of ataxia in that it is neither irregular nor spontaneous, but distinctly associated with the effort of moving the limbs. The spastic gait is, further, relatively slow—certainly much slower than the normal walk, and decidedly slower than the gait in ataxia. When spastic paralysis is confined to one side of the body, the peculiarities in the movements of foot and leg, just described, are limited, of course, to the paralyzed side. Occasionally in such cases the foot, instead of being closely approximated to the median line, is well thrown out by the tilting of the trunk toward the sound side.

FIG. 14.



In Fig. 14 are seen the relative positions of the foot and leg, as also the varying rates of movement in the case of lateral sclerosis.

In the gait of simple weakness, such as that seen in partial loss of power in both legs, it will be noticed that the feet are thrown forward in a passive or pendulum-like manner, and that the weight is not trusted to the leg resting upon the ground until the knee has been thrown far back—*i. e.*, has become slightly retroflexed or locked. This expedient the patient unconsciously adopts in order to make a collapse from sudden flexion impossible. This is in marked contrast with what is seen in the normal leg. When a simple weakness is limited to one leg the latter is carried forward in a helpless or pendulum-like manner, and the weight of the body is not thrown upon it until, as in the previous instance, it has become well locked at the knee. Special gaits due to the paralysis or contractures of special muscles or groups of muscles are described whenever necessary in the text.

At times it is important to study the movements of the legs not only in the erect position, but also when the patient is lying flat upon his back, upon his side, or even prone.

It is evident that the various tests for muscular co-ordination are directly related to the tests for station. It follows that a patient whose station is below normal is apt to reveal lack of co-ordination or *ataxia* in his gait.

Next we examine the arms. Here again we determine whether movement is modified, whether it is impaired so as to indicate either weakness or rigidity, or whether there is lack of proper co-ordination. We test the strength of the arm in various ways. A fair idea can be obtained by asking the

NERVOUS DISEASES AND THEIR TREATMENT.

CHAPTER I.

GENERAL CONSIDERATIONS.

JA 27

By S. WEIR MITCHELL, M.D., and F. X. DERCUM, M.D.

For a generation past diseases of the nervous system have attracted an ever-increasing amount of attention. Little by little they have become better understood and have gradually assumed the importance of a well-defined department of medical knowledge. However, unlike such specialties as diseases of the eye and ear, no line of demarcation exists between this department and general medicine. Indeed, the two touch at almost every point and can never be entirely separated. At the same time the field of nervous diseases has become so vast and the details so intricate that special study is absolutely demanded.

Before disease can be intelligently considered, a knowledge of normal structure must first be acquired. Especially is this true of diseases of the nervous system. Here a knowledge of morphology is of supreme importance. Not only must the student be familiar with gross relations and minute details, but also with general principles. To this end his studies should not be confined to man, but should, if possible, include general biology. An understanding of nervous structure is hardly possible without a knowledge of embryology and comparative anatomy. The lessons taught by these sciences make clear much that is otherwise inexplicable and give to the student a foundation for a broad and philosophical grasp of the subject.

The study of the diseases of the nervous system embraces of necessity a study of its pathology. The pathological processes at work are many of them common to the body as a whole. Such, for instance, are the various forms of inflammation, some forms of sclerosis, and several forms of tumor. Again, as in diseases of other organs, morbid processes may affect primarily either the parenchyma or the supporting and protective structures; that is, they may affect primarily the nervous elements, the nerve cells and fibres, or the neuroglia, the connective tissue, and the enveloping membranes. The bloodvessels, also, are subject to changes similar to those which they undergo elsewhere. In general terms, with the exception of certain new formations, such as gliomata, and with the further exception of a special form of parenchymatous degeneration known as tract or Wallerian degeneration, the morbid changes in the nervous system resemble the changes found in other tissues.

On the other hand, in their clinical aspects nervous diseases present numerous special features. This is true not only of symptoms, but also of the family and personal histories. Thus it is noteworthy that in the production the muscle, with a consequent movement of the limb. Much ti have been spent upon this phenomenon. By some observers it still is, regarded as a reflex, the theory being that an impulse tr nerve-endings in the tendon to the spinal cord, and thence is r to the muscle. However, the time elapsing between the blow don and the contraction of the muscle has been repeatedly measured, and is less than would be required for the passage o along the course just described. Therefore it is held by others nomenon is really due to muscle irritability, and that the mu because of the sudden pull upon its belly which the blow upo produces. Whatever may be the correct explanation, the study tion in diseases of the nervous system is of the utmost importan

As might be expected, the quadriceps is not the only muscle when its tendon has received a blow. As a matter of fact, all o of the body whose tendons are mechanically accessible respon manner; but certain tendons, because of their anatomical relati favorably situated for the study of these reactions than others important of them all is the tendon of the quadriceps, the patellæ. The reaction which is obtained by striking this tend as the patellar reflex, or the *knee-jerk.* The term patellar ref tionable because it implies a special theory. The term knee expresses a fact, and is much to be preferred. The other tend such as the elbow-jerk, obtained by striking the tendon of the wrist-jerks, and the tendo-Achillis jerk are of minor importanc valuable in certain cases. To this category should also be ad jerk, described by Morris J. Lewis.

The amount of response to a blow upon a tendon depends, being equal, upon the degree of the muscle tonus. It follo increase in the muscle tonus is accompanied by an increase in reactions. Advantage is taken of this fact in cases in which t are feeble, or apparently absent, by asking the patient to mal voluntary effort with the hands, or with the jaws, at the mome tendon is struck. The voluntary muscular effort causes a rise in all the muscles of the body, and thus the patient is placed under able conditions for the detection of the tendon response. This t the test by motor re-enforcement, while the method by which is elicited bears the name of its discoverer, *Jendrassik.* We and Morris J. Lewis[1] have shown that this volitional re-enforc for a brief interval after the volition itself has ceased, and furthe have been expected on *à priori* grounds, continued muscular exe enfeebles the knee-jerk, and this enfeeblement also lasts for an interval of time.

Mitchell and Lewis have also pointed out that re-enforcem knee-jerk may also occur through sensory stimulation. This has firmed by Bowditch and Warren[2] and by Lombard.[3] If just at t patellar tendon is struck we touch the skin of the patient anyw arm, on the body, or on the leg with a piece of ice or a hot sp denly twitch a hair, or pinch the skin, the knee-jerk becomes marl gerated.[4] Other agencies, such as the emotions, also influence th Lombard has shown experimentally.[5] A familiar example is that by an hysterical girl in whom, as Mitchell expresses it, "fear, excitement, apprehension, and perhaps shame contribute largely to the production of an exaggerated knee-jerk. If in such a girl we strike the patellar tendon, we notice further that not only is the knee-jerk exaggerated, but with it, also, the other leg flies up, and also one arm—usually the left. This is the combined result of the blow on the tendon and of emotions." In like manner volitional efforts which are unaccompanied by actual movement, such, for instance, as volitional efforts directed to a lost part—*e. g.*, an amputated limb, re-enforce the knee-jerk. Again, if a part of the body is faradized during the time the knee-jerk is tested, the latter is also increased. Galvanism also, when passed through any portion of the body, and especially when passed through the head, increases the knee-jerk excessively.

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[1] Mitchell, S. Weir, and Lewis, Morris J.: Medical News, 1886, February 13 and 20,

[2] Bowditch, H. P., and Warren, J. W.: Journal of Physiology, 1890, vol. xi., Nos. 1 an ditch, H. P.: Boston Medical and Surgical Journal, May 31, 1888.

[3] Lombard, Warren Plympton: American Journal of Psychology, October, 1887.

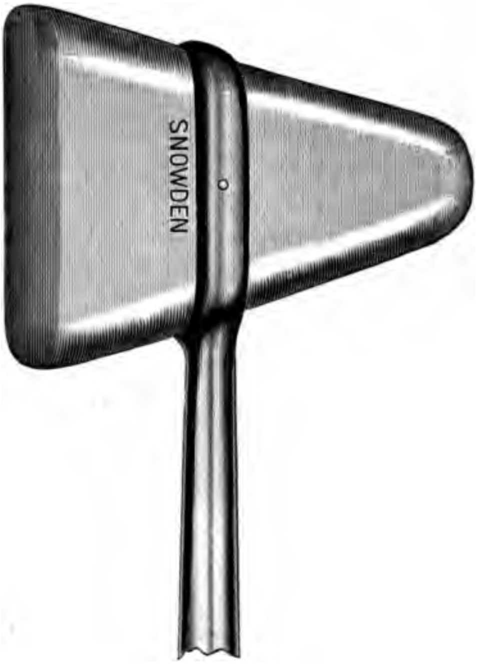
[4] Mitchell, S. Weir: Lecture on Muscle Reactions, etc., Medical News, 1888, June 23.

[5] Loc. cit.

In cases in which it is important to determine whether or not the knee-jerk is really exaggerated, the patient should be seated in a comfortable chair, so as to have all his muscles relaxed, and his eyes should be gently closed.

Finally, in certain diseased conditions, the knee-jerk is inhibited by stimulation of the skin or of the peripheral nerves, as has been observed by Nothnagel, Erb, and others. It was noted by Mitchell that while slight and sudden pains re-enforce the knee-jerk, excessive pain now and then inhibits it. For this phenomenon of depression and inhibition of the knee-jerk Mitchell proposes the expression "negative re-enforcement," or "deforcement."

FIG. 17.

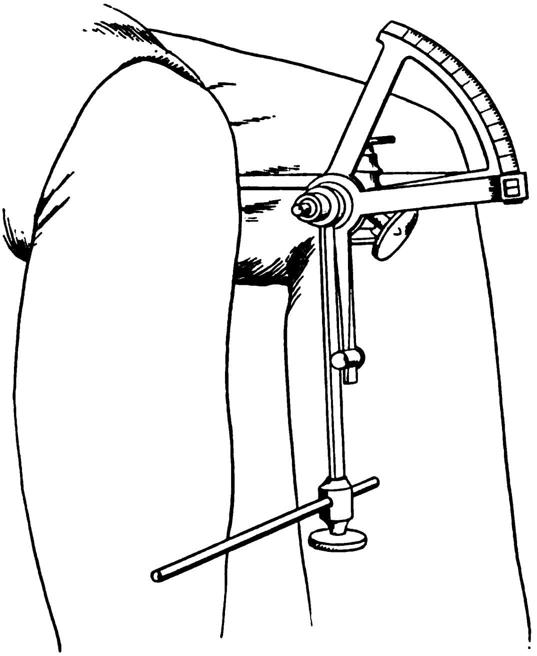


J. Madison Taylor's hammer for examining muscle and tendon reactions. The hammer is made of rubber, while the handle consists of a piece of fenestrated steel.

The detailed method of eliciting the various tendon phenomena is of importance. The practical rule to bear in mind is to place the limb in such a position that the muscle, the tendon of which is to be struck, is slightly stretched. Such a position as is assumed in semi-flexion either of the leg or of the arm answers most purposes. Starting with the knee-jerk, it will be found that the most favorable position is that assumed by a patient when seated on a high chair or table. The thigh and leg are to be at right-angles, the leg hanging and all of the muscles of the limb relaxed. It is often more convenient to seat a patient on an ordinary chair, and to have him cross one knee over the other. This position answers almost every purpose. Occasionally it is necessary to examine the knee-jerk of a patient who is confined to bed. Under these circumstances it is best, when possible, to turn the patient on his side, with the leg to be examined uppermost. The leg should be slightly flexed and gently supported at the lower part of the thigh by the disengaged hand of the physician. The amount of flexion is of considerable importance. It should be decidedly less than when the patient is seated.

If the leg be too much flexed, the muscle will be overstretched, be no response. If, on the other hand, the leg be too little fle extended, the muscle will be too much relaxed, and, as before, no response. A convenient position for eliciting the elbow-j operator to grasp the arm, extend it slowly from the shoulder, a the triceps tendon. The biceps-jerk is conveniently tested b arm of the patient, slightly flexed, to rest within the arm of The biceps tendon is then similarly struck a light blow. T elicited by asking the patient to allow the mouth to remain and then striking a light blow upon the chin in a downward d

FIG. 18.

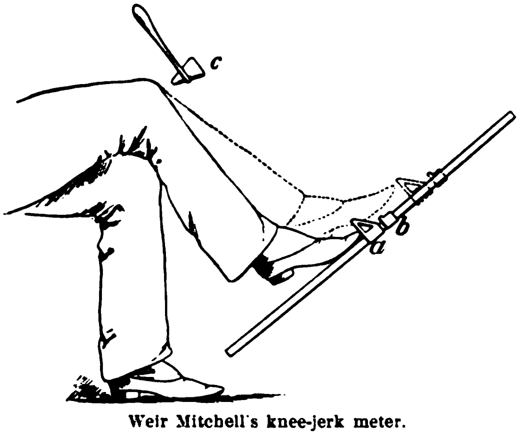


Lombard's knee-jerk meter.

If, under certain circumstances, a forcible and steady pull be the tendon of a muscle, a number of rapidly recurring contra This reaction is most frequently observed in the gastrocnemius, instance is termed the *ankle-clonus*. It is elicited as follows: T seated, and the leg, almost but not completely extended, is supp disengaged hand of the physician. The foot is then seized and steadily flexed upon the leg. If the patient suffers from orga the motor pathways, or if there be any cause that greatly i reflex excitability of the cord, or that decidedly raises the musc foot may vibrate rapidly to and fro. As the student has doubtl this phenomenon is not present in health. Other clonic tenc may also be observed, but only when associated with abnormal c

In describing the various tendon phenomena in a given case instance, whether the knee-jerk be normal, whether it be exagger it be diminished, or, finally, whether it be absent. If the ankle-clonus be present, it is described as slight, feeble, marked, or excessive. The same applies also, of course, to tendon reactions observed in other parts of the body. For the purpose of measuring the knee-jerk with great accuracy a delicate instrument has been invented by Lombard.[1] In this apparatus the patient lies upon one side, with the leg to be experimented upon fixed in a guttered splint, the foot being suspended in a stirrup by a long string so that it is free to move. The amount of the excursion of the foot is then recorded by means of a system of levers upon the smoked surface of a revolving cylinder. However, this apparatus has for its object experimental studies in the laboratory. For clinical use Dr. Lombard has invented a more simple instrument. The patient is seated on a chair sufficiently high to prevent his feet from resting upon the floor. By means of a strong clamp this instrument is fastened to the chair between the legs of the patient. It consists of a bar which can be pulled out or shortened, and which carries a dial plate divided into centimetres. A slight swinging rod with a cross-bar is set so as to drop on the middle of the tibia. When now the patellar tendon is struck a blow, the leg, in response, raises the rod carrying before it an index (Fig. 18). As this instrument is too costly for common use, Weir Mitchell has devised an exceedingly simple meter, which is constructed of inexpensive materials (Fig. 19). A metre or yard-stick carries a light, very movable arrangement of wire, about four inches in height by five in breadth (*a*). This is set or fixed so that the toe, rising with the tendon-jerk, will push it up the scale. As it moves it pushes up, and leaves behind it as a marker, a double loop of wire (*b*).

FIG. 19



Weir Mitchell's knee-jerk meter.

In attempting accurate studies of the knee-jerk in a given case, we should bear in mind the observations of Lombard,[2] that "the extent of the normal knee-jerk is continually undergoing change. So great are the variations, even when the subject is at rest, that a correct idea of the activity of the process can be gained only by averaging the results of twenty or more experiments. The average knee-jerk varies in amount at different times of day, being, as a rule, greatest in the morning, soon after breakfast, and being very much less at night. The decline which occurs as the day advances is very irregular, but, in general, the knee-jerk is larger after each meal. Finally, the extent of the knee-jerk may differ greatly on different days.

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[1] Loc. cit.

[2] Loc. cit.

"The causes of these variations of the knee-jerk are not only alterations in the muscles and nerves involved in the process, but, to a still greater degree, changes in the activity of the central nervous system, either as a whole or in part. Thus fatigue, hunger, enervating weather, and sleep, conditions which decrease the activity of the whole central nervous system, decrease the average knee-jerk; while rest, nourishment, invigorating weather, and wakefulness, influences which increase the activity of the central nervous system, increase the average knee-jerk."

In order to facilitate note-taking, Weir Mitchell has introduced a series of symbols which are so readily understood as to need little explanation, thus: K J + means that an exaggerated knee-jerk is present; K J + + means that it is excessively exaggerated; K J — means a diminished knee-jerk; A Cl means an ankle-clonus; E J an elbow-jerk, and so on. The following formulæ, with the accompanying explanation, are taken from Weir Mitchell's Lecture.[1]

Arms:	R. E. J. 0.	Rt. 0.	L. E. J. 0.	Rt. 0.
	R. M. J. + +	Rt. 0.	L. M. J. +	Rt. 0.
Legs:	R. K. J. 25°.	Rt. 40°.	L. K. J. 27°.	Rt. 36°.
	R. M. J. quadriceps, n.	Rt. n.	L. M. J. n.	Rt. n.

Station: $\frac{2,}{A}\ \frac{1\frac{1}{2}}{D}$ · Eyes shut.

Explanation. Arms: right elbow-jerk, none. Re-enforcement, none, etc. Right muscle-jerk, excessive. No re-enforcement, etc. Legs: Right knee-jerk, 25 degrees Lombard's meter. Re-enforcement sends lever to 40 degrees, and these records mean maximum of several trials. As concerns muscle-jerk, n means normal, + excessive, and — less than normal. Station $\frac{2}{A}\ \frac{1\frac{1}{2}}{D}$, means the anterior sway is 2 inches, and the dextral 1½.

A point of practical value in relation to the tendon reactions remains to be stated, and that is, that each tendon reaction bears a definite relation to certain levels of the spinal cord. These levels correspond to the origins of the nerve fibres supplying the muscles concerned. Thus the anterior crural nerve, which supplies the quadriceps muscle, is derived from the second, third, and fourth lumbar nerves. These arise from the lumbar cord at a level opposite the body of the twelfth dorsal vertebra. The knee-jerk is therefore directly affected by lesions of the cord in this position. Similarly the ankle-clonus is related to the lumbar and sacral portions of the cord at a level opposite the upper portion of the body of the first lumbar vertebra.

In some individuals, especially those of an hysterical temperament, local muscular contractions may come on spontaneously. At times the contraction involves only a portion of a muscle, the swelling produced resembling a tumor. The latter, by reason of its not infrequent spontaneous disappearance, is known as a *phantom tumor.* This condition has been especially studied by Weir Mitchell. It is not accompanied by any displacement of the limbs of a part, but simply by the appearance of an apparently foreign mass beneath the integument. It is occasionally noted in the muscles of the calf, sometimes in the pectoralis major, and not infrequently in the muscles of the abdomen. In some cases similar local swellings in the muscles may be produced by slight blows with a percussion hammer or with the tip of the finger.

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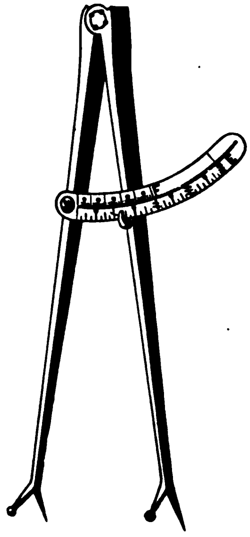
[1] Lecture on Muscle Reactions, etc. Loc. cit.

When a blow upon a muscle is made with sufficient force the muscle responds as a whole, the phenomenon being often attended by a movement similar, but less marked, to that which is seen when the tendon is struck. Muscle-jerks, as they are called, were first studied by Weber, but their best clinical studies are by Buzzard and Weir Mitchell and Morris Lewis. When a blunt percussion hammer strikes on a muscle, two phenomena follow to a variable extent. First, the muscle responds by a long contraction, the breadth of fibre struck rising, and highest near the point struck. This reaction may move the limb, as when the extensors of the hand are thus excited. Also, it is usual to see or to feel, at the point struck, a small, firm contraction, which remains twenty seconds or more, and is called the short or hump contraction. Both of these responses vary, and are increased or even lost in certain forms of disease.

The muscle-jerk, as Mitchell and Lewis showed, is identical with the so-called tendon-jerk, but is a coarser phenomenon. The tendon-jerk is the reply to a quick pull on the muscle, and is a delicate symptom easily lost or increased. The direct muscle-jerk, unlike it, survives for a time nerve sections, but is also, like it, re-enforcible by motion and sensation.

FIG. 20.



Carroll's æsthesiometer.

We next direct our attention to the sensory symptoms. These resolve themselves into those of the various general and special senses. We begin, as a rule, by investigating the *tactile sense.* This is tested in various ways, such, for instance, as touching a part lightly with the finger, with a feather, or with a piece of cotton-wool. If the cutaneous sensibility be normal, the patient responds to comparatively slight impressions. A ready method for determining actual loss of the tactile sense is to prick the skin with a needle, which for this purpose is conveniently mounted on a cork. More accurately, the same thing is accomplished by means of an æsthesiometer. This, in its simplest form, consists merely of a pair of draughtsman's compasses to which a scale is so attached as to enable us to separate the points to specified distances (Fig. 20). Instead, however, a graded rod bearing at right-angles two points, one of them movable, such as originally devised by Sievking, may also be used. Persons in health are able to separate within certain limits the two impressions made by the points of the æsthesiometer. If, however, the tactile sense be impaired, the distance at which the points can be separated is much increased, so that when even widely apart they may give rise to but one impression instead of two. In health, we should be able to separate the points about as follows:

	Mm.
Tip of tongue	1.1
Red part of the lip	4.1
Tip of nose	6.8
Under the chin	33.8
Eyelid	11.3
Forehead	22.6
Occiput	27.1
Vertex	33.8
Third phalanx of finger, volar surface	2.- 2.3
Second " " " "	4.- 4.5
First " " " " "	5.- 5.5
Third " " " dorsal "	6.8
Second " " " " "	11.3

	Mm.
Ball of thumb	6.– 5.7
Ball of little finger	5.– 5.6
Centre of palm	8.9
Back of hand	31.6
Lower third of the forearm, volar surface	15.
Plantar surface of the great toe	15.8
Forearm and leg	45.1
Knee	36.1
Neck	54.1
Back at the fifth dorsal vertebra, lower dorsal and lumbar region	54.1
Middle of the neck	67.7
Upper arm, thigh, and centre of the back	67.7
Sacrum, gluteal region	44.6

The above data have been culled from Landois and Stirling's *Text-book of Human Physiology.*

Absolute loss of the tactile sense is termed *anæsthesia.* It is often associated, as we will see, with other phenomena. Occasionally we find that our patients react excessively to a very slight touch. This condition is known as *hyperæsthesia.* In various nervous affections in which we fail to discover actual loss or impairment of the various cutaneous sensibilities, the patient notwithstanding complains of obscure subjective sensations. Thus, in locomotor ataxia it is not uncommon for the patient to complain of velvety or cushion-like sensations in the feet. At other times, for example in hysteria, the patient may complain of prickling, creeping, or other anomalous sensations. These phenomena are technically termed *paræsthesias.*

In studying the tactile sense it is further of great importance to note whether the patient refers the impression accurately to the point that is touched. Not infrequently, it is found that a patient correctly appreciates contact, but refers it to the wrong finger or to the wrong segment of a finger or limb, and sometimes, indeed, to the opposite side of the body. Thus, a patient may refer a touch upon the proximal phalanx of a finger to the metacarpal bone of that finger, or a touch upon the back of the hand to the back of the forearm; that is, the error may be made on referring the point touched to a segment of the limb immediately above. When the impression is referred to the opposite side of the body, the condition is known as *allocheiria.* For this phenomenon Weir Mitchell proposes the term "*touch squint.*" Occasionally a patient will distinguish two impressions when but one point of contact has been made; that is, there is a condition in which the impression is reduplicated.

Closely allied to the tactile sense is the *pressure sense,* so-called. By it we are enabled to determine the relative amount of pressure which is exerted upon this or that portion of the body. For a knowledge of the amount of pressure upon any part of the surface, we are indebted first to the nerves of the skin, and secondly to those supplying the deeper structures. Every now and then, in various morbid conditions, a light touch is not recognized, and the patient reacts only when considerable pressure is made. The pressure sense may be lost hand-in-hand with the tactile sense. Not infrequently, however, it is merely diminished when the tactile sense is comparatively well preserved. Various forms of special apparatus have been devised for studying the pressure sense, but clinically they are unimportant. A convenient way for testing it, is to have the patient seated with the backs of his hands resting upon a table, and then, having blindfolded him, to place various objects of similar bulk, but varying weight, upon the two palms or fingers. Two vials of the same size containing different amounts of mercury answer this purpose very well.

When we make our studies of the tactile sense, we should notice whether, when the needle is thrust into the skin, it also causes *pain.* The sense of pain, we should remember, is entirely distinct from the tactile sense and may indeed be lost, while the latter is preserved. In such a case the impression made by the needle is promptly recognized and accurately localized, but at the same time the patient feels no pain, no matter whether the needle be thrust deeply into the skin or even be moved about in the rete mucosum. Another convenient method of testing the pain sense is to touch the parts with a pair of thin metallic electrodes, connected with a rapidly interrupted faradic battery, and held about half an inch apart. By this means the area in which the sense of pain has been abolished can be accurately mapped out. In testing for the pain sense in cases in which it is apparently lost, parts that are especially sensitive should be selected, such, for instance, as the pulp of the finger immediately beneath the nail, or the nipple. If the sense of pain be altogether lost in a part, the condition is termed *analgesia*. It is observed in hysteria and in certain organic diseases of the cord, more especially syringomyelia.

Next in order is the study of the *sense of temperature*, or the *thermal sense*. As is well known, normal individuals can readily distinguish hot from cold objects. For practical purposes, this sense can be readily tested by two test-tubes or vials of equal size, one of which is filled with hot and the other with cold water. It is sometimes found that, though the tactile sensibility is well preserved, the patient is unable to distinguish differences of temperature even when these differences are marked. Sometimes he will simply say, "I cannot tell," and at others will call hot cold or cold hot. In the latter instance the temperature sense is, of course, not abolished, but merely perverted. It is best, in making this test, to use temperatures that are decided. For instance, water from a temperature of 40° to 50° F. for the cold test, and water at a temperature of 110° to 115° F. for the hot test. It is not infrequently found that patients who have suffered from frequent exposure to inclement weather are unable to distinguish small differences of temperature. However, most persons will pronounce objects at a temperature of 60° F. as cold or cool, and at a temperature of 85° F. and upward as warm. Some are able to distinguish between a few degrees or even a fraction of one degree, but the ability thus to distinguish varies greatly within the limits of health. For practical purposes, therefore, as has been already stated, decided temperatures should be used. The thermal sense is every now and then abolished in organic disease, as, for instance, syringomyelia.

Every area in which an impairment of the tactile, the pressure, or the pain sense is discovered, should be accurately outlined upon a chart or upon the skin of the patient by means of an aniline pencil. This is of importance, inasmuch as we desire to learn whether the impairment is in relation to the nerve-supply of the skin of a certain part, or whether it is in relation to a limb as a whole or to a segment of a limb. Thus, a lesion destroying the ulnar nerve affects special regions of the hand and special fingers. In certain affections, however—*e. g.*, hysteria—we may have an anæsthesia which involves the entire hand like a glove or a portion of the leg like a stocking. Such an anæsthesia is known as *segmental anæsthesia*.

In certain cases it is important to test the *muscular sense*. We judge largely of the positions of our limbs and of the posture of the body by the sensations received from the muscles. These keep us informed as to the relative degrees of contraction of the various groups. It is obvious that in ataxia and in various forms of motor inco-ordination this sense is much diminished. Further, the sensations received from the muscles are correlated with the sensations received from the pressure-sense already considered, and are probably assisted by the impressions received from the tendons and joints. The muscular sense can be tested very readily by asking the patient to estimate the weight of various objects placed in his hands. We may con-

veniently make use of a handkerchief, tying together the
and suspending the loop thus formed from the patient's hand
semi-flexed and the patient blindfolded. Various objects are
in the handkerchief, and the patient asked to estimate their

Important for us to study also are the cutaneous reflexes.
upon the fact that when the skin of certain portions is irritat
cles respond by contracting. Thus, if the sole of the foot
foot and leg is suddenly jerked upward by an involuntar
flexor muscles. This phenomenon is known as the *plantar re*
if in very young children the palm be irritated, the hand is p
If the walls of the abdomen, especially the epigastrium, b
rectus and other abdominal muscles respond. Similarly, if t
tated, the group of the erector spinæ muscles contract.
reflex is the name given to the contraction of the cremaster
about by irritating the inner surface of the thigh in its upp
testicle on the side irritated is promptly retracted. This refle
active in children than in adults.

Examination of the special senses is next in order. In this
of the eyes is of the greatest importance. It must be made from
of the neurologist. The various methods to be pursued are d
subjoined section by Dr. Charles A. Oliver. Next, the hearin
amined. This should be tested by means of a watch or tunin
amounts of deafness are of comparatively little significance to
It is important merely to determine first whether marked deaf
and second whether the latter is due to disease of the middle-ea
auditory nerve, or of the cortical auditory centres. The af
auditory nerve are described in the chapter upon diseases
nerves. It is of importance here only to state that should a
explainable upon a nervous basis, the case should be referred t
examination and report. The methods of testing the sense
smell are also described in the chapter upon diseases of the cr

In studying the various phenomena of cutaneous sensibil
a given case we should note, at the same time, whether the su
handle is unusually warm or cold. When the surface temp
widely from the normal the fact is readily perceived by the hand
in the legs in infantile paralysis. For accurate purposes, ho
studying the surface temperature of the head in cases of su
tumor, a surface thermometer should be employed. Slight de
the normal are only important when constant. Various prec
be used to examine a part always under the same conditions, as
has shown that so simple a fact as change of posture in a limb
influence the result.[2]

Lastly, it is of the utmost importance to study the various
nutrition presented by the patient. These may be general a
body as a whole, or they may be limited to special parts or str
turbances of the general nutrition often possess a profound signi
is true of general emaciation, of excessive increase in bulk, of
the features, and of change in shape of the extremities. Furth
departures from the normal standard of weight, taking hei
other elements into consideration, are likewise of importance
this true if the patient presents a history either of rapid loss or

Special disturbances of nutrition are such as are presented by

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[1] Clinical Lessons, Medical News, 1894, vol. lxiv. p. 6.

[2] For an account of normal surface temperatures the reader is referred to Land
Physiology.

its appendages, the muscles, the bones, and the joints. The phenomena presented by the skin vary greatly. Thus it may feel unusually dry or it may be excessively moist; in the one case due to diminished, in the other to excessive perspiration. At another time it will be observed that the epithelium is shed more readily or more profusely than in health, and that the hair covering the skin, especially the fine hair on the extremities, is dry and brittle. At times unusual growth of hair may be noticed in special regions, or the hair of a certain area of the scalp, face, or other portion of the body may be entirely lost. The nails, too, may present evidences of change in nutrition. Thus the nails of a paralyzed limb often grow more slowly than those of the normal side. Often, too, they present longitudinal ridges, are rough and unusually brittle. In some cases it is important to test their rate of growth, and for this purpose it is convenient to make a slight stain with nitric acid at the root of the nail. The mark is indelible, and enables us to note the amount of growth from time to time.

Under special conditions the skin may become excessively smooth and glazed, or local sloughs, small blebs, or purpuric spots may occur, all due to disturbances of nutrition having a nervous origin. To this category also belongs a vesicular eruption known as herpes zoster. (See Neuritis.) Redness and burning (causalgia) are also occasionally met with. Again, the skin sometimes presents a condition which can only be designated as a "diminution of nutrition resistance." It is every now and then observed in persons who have suffered injuries of nerves. The skin of a part is no longer able to withstand slight blows, bruises, or changes of temperature as well as formerly; thus a patient immersing both hands in warm water far below a hurtful temperature may have the hand of a damaged limb severely scalded, while the sound hand escapes.

Finally, the skin may undergo slow trophic changes, such as are seen in scleroderma, morphœa, and hemi-facial atrophy; or it may be unusually soft, swollen, and yielding, as in myxœdema.

The nutritive changes in the muscles are revealed especially by changes in bulk and consistence. Thus in many organic palsies the muscles become wasted and flabby to the feel. Under certain circumstances local and irregularly recurring contractions of bundles of fibres, *fibrillary tremors*, are observed. At other times, instead of being atrophied, they may be much enlarged and much firmer and denser than normal.

Changes in the bones are most frequently made known to us by a history of unusual fragility. Thus a patient will tell us that some slight and simple movement, such as stepping over a gutter, tripping over a mat, or even throwing one thigh over the other, has produced a fracture. Change in the shape and size of bones is also now and then observed, as in acromegaly. The joints, also, in various nervous affections, reveal most extraordinary changes called arthropathies. These are manifested in painless enlargements, distortions, and atrophies. Their occurrence should be especially sought for in locomotor ataxia and syringomyelia.

Very often electrical examination of the nerves and muscles gives us most valuable information. Indeed, this is frequently the case when no gross physical changes are present.

Both muscles and nerves when diseased respond to electrical stimulation in a manner different from that in health. The changes in the "electrical reactions," as they are called, are of two kinds. First, the response may vary in amount, and, secondly, it may vary in character. The first is termed a *quantitative change*, the second a *qualitative change*. When a nerve or muscle reacts excessively *quantitative increase* is present. When it reacts in a lessened degree *quantitative decrease* is present. Quantitative increase is,

therefore, observed when a nerve or muscle reacts to a feebl
greater extent than in health. Quantitative decrease is obse
muscle reacts only after a current of decidedly increased stre
applied. When quantitative changes are slight, they are ofte
to estimate or measure. However, if the disease be limited t
one arm, the nerves and muscles of the corresponding limb
or healthy side serve as ready standards of comparison. I
disease be bilateral—that is, if it involve both arms or both leg
is much more difficult. Here the researches of Erb come to
He has shown that the frontal, the spinal accessory, the ulnar,
neal nerves respond almost equally to the same minimal curre
these nerves may, therefore, be selected as a standard in s
titative changes. The degree of quantitative increase or q
crease may, of course, vary considerably. Quantitative de
ample, is in some instances comparatively slight, while in
become so great as to amount to a total loss of response. In t
it is necessary to state that this loss of response occurs first
current, and only at a subsequent period to the galvanic curr
it in other words, "faradic extinction" occurs first, "galvan
subsequently.

The *qualitative changes* are characterized by the so-called r
generation. This embraces a diminution or loss of response
supplying a given muscle to the faradic and galvanic currents,
or loss of response of the muscle to the faradic current and
response to the galvanic current, with the following change
response to the galvanic current is in the beginning increased.
response relative to the closure of the negative and of the p
radically changed. This can be best understood by reviewi
sequence to galvanic stimulation in the normal nerve and musc
or muscle be stimulated by a minimal galvanic current, it will
a response first occurs to negative closure; that is, the posit
(Anode, An), being placed over some indifferent area—*e. g.*, the
the negative pole (Cathode, Ca) over the nerve or muscle to be
will be noticed that a very weak galvanic current is not follow
sponse whatever, but that on gradually increasing the strength
a response is finally obtained when the current is closed—*i.*
upon negative (or Cathodal) closure. If the poles be now revers
negative pole rests upon the sternum and the positive pole upon
muscle, no contraction is obtained either upon closing or opening
However, if the current be gradually increased in strength, we
contraction occurs upon *opening* the current; in other words, th
with a strong current a *positive opening contraction.* It is now furt
that upon *closing* the current there is again a response; that is,
ent a *positive closure contraction.* This positive closure contrac
ever, decidedly weaker than the negative closure contraction; it i
tically, to the positive opening contraction. If, with the elec
reversed, *i. e.*, the positive pole upon the sternum and the negati
the muscle, the current be still further increased in strength, it
when the current is broken that a contraction of the muscle a
though it is notably weak. In other words, there is now prese
opening contraction. If, with the electrodes in the same position
be closed, the muscle responds by a tonic contraction, that is, a
nus (Te) is produced. Briefly restated, the normal reactions t
the following sequence:

CaClC (Cathodal closure contraction).
AnOC (Anodal opening contraction).
AnClC (Anodal closure contraction).
CaOC (Cathodal opening contraction).

In the reaction of degeneration these phenomena are changed, as follows: It is first noticed that the anodal closure contraction is more pronounced. If the condition be more marked or further advanced, this anodal closure contraction equals the cathodal closure contraction, and in the more marked cases, or at a later period, it more or less exceeds the latter. In other words, in the established reaction of degeneration the anodal closure contraction is the first response obtained to a minimal current. It is now also observed that the cathodal opening contraction is more pronounced, and that, as the condition becomes more marked, it soon equals the anodal opening contraction, and may even exceed the latter.

In the reaction of degeneration, therefore, we have a reversal in the sequence of the responses to the current. Further, we also observe that the manner of contraction of the muscle is different from that of health. In health the response is short and almost immediate. In the reaction of degeneration it is slow, long drawn, and persistent. The reaction of degeneration may, of course, be present in varying degrees. Occasionally, also, the phenomenon appears to be limited to the muscle, while the nerve which supplies the latter escapes, or reveals but a doubtful change.

For convenience in note-taking the symbols already indicated are used. Thus, AnClC = CaClC means that the anodal closure contraction is equal to the cathodal closure contraction, which is indicative of the reaction of degeneration. When the reaction is more pronounced the formula may read AnClC > CaClC, which means that the anodal closure contraction is greater than the cathodal closure contraction. The symbol De R is used briefly to indicate that degeneration reaction is present.

The reaction of degeneration, as the student has doubtless surmised, is principally of diagnostic value. It is never present in lesions limited to the upper segment of the motor tract; that is, in lesions occurring either in the cortex or in the descending motor pathways in the internal capsule, the crura, the pyramids, or the lateral columns of the cord. On the other hand, it is present, other things equal, in lesions involving the *lower segment of the motor tract;* that is, the anterior cornua of the gray matter of the spinal cord, the cerebro-spinal nerves, or the terminal distributions of the latter in the muscles. In gradual and slowly progressive myopathies simple diminution of electrical response alone is present.

The above brief outline of the methods to be pursued in studying causes of nervous diseases are only intended to be general in character. Individual cases may demand special methods of investigation.

THE EXAMINATION OF THE EYE FROM THE STANDPOINT OF THE NEUROLOGIST.

By CHARLES A. OLIVER, M.D.

Practically, for the student's purpose, the examination of the eye from the standpoint of the neurologist resolves itself into several separate inquiries.

I. The Study of the Condition and the Movements of the Muscles which Govern the Motion of the Eyeball and the Lids. The muscles in and around

the eye are so delicately balanced and so intimately associate
movement that may be given to the two organs and the lids, th
ancy in their actions is often at once apparent to both the p
observer. For instance, if the external rectus muscle be paral
will be turned outward by the unopposed action of the he
rectus muscle, thus producing a condition known as *paralytic co*
or *abducens paralysis.*

Should the muscle be merely paretic, the incompleteness of i
not be manifest by ordinary inspection. Here the patient will
plain of double vision—*diplopia*—when he looks toward the p
In this case the fault may frequently be brought into view b
gaze steadily at the upraised finger whilst the finger is mov
directions. If this be done repeatedly and carefully, the ey
found to lag behind its fellow when the weakened muscle has
into play. The reason for this is that, with the same degree of n
the weakened muscle accomplishes a less result than its healthy

Should the paralysis be limited to certain related movements,
eyes are brought into definite action with one another the faulty
be found only when the patient is induced to make efforts to acc
movements. For example, the two internal rectus muscles may
act simultaneously so as to permit the two eyes to follow the up
tip when it is moved directly in toward the patient's nose—a con
as *paralysis of convergence,* and most generally dependent upon
the nuclear centres.

When several of the extrinsic muscles of the two eyeballs
affected the condition produced is generally that of almost im
ready total cessation of the ocular movements both in attempt
and conjoined actions. The most probable explanation of this
is, that as the trunks of the various nerves of the implicated mus
apart in their various passages back to their associated nuclear
condition must be dependent upon a sudden hemorrhage into
region. Here the appearance of the external symptom, which
known as *acute nuclear palsy,* or *ophthalmoplegia exterior acuta,*
easily understood, rendering the diagnosis unmistakable.

In this type of the affection the iris and the ciliary muscles are
Should they, however, be affected, dilatation of the pupil (*irid*
inability of recognition of small objects, such as printed or writte
at short distances without the employment of proper correcting
plegia), will be superadded to the list, giving the term *total oph*
Should the iris and the ciliary muscles be alone affected, produc
dilatation of the pupil and inability to accommodate, *interior oph*
is said to be present.

In the chronic form of the same disease the vertical muscles a
those which are primarily affected, the other oculo-motor nerv
sooner or later following, as shown by involvement of the ot
muscle series.

When the paresis is not manifest and the patient asserts t
double, dizziness and inability to move about properly are the
nent symptoms. Here the faulty muscle may be discovered b
of means. One of the simplest plans is to have the patient gaze
faint light alternately with his two eyes. If a slight disturbanc
will assert, after a moment's explanation, that the light appears t
a new position the moment that the covered eye is exposed to the
degree of the fault in the muscle can be determined by the use of
do this properly the prisms should be placed with their bases

position from which the light appears to jump. They are then to be gradually increased in power until the movement ceases. The strength of the prisms that is necessary to stop the movement of the light will give a measure for the degree of the fault.

Another plan that is appropriate for the same character of cases in the sick-room, and one that does not require any special apparatus, is to have the patient gaze at a dim light at about five metres' distance with both eyes open. A prism of ten degrees, with its base up, is then placed in front of the patient's left eye. This will produce an immediate doubling of the light in the vertical meridian. If there is any discrepancy in muscle-tone or action, the second light will not only be situated below the original light, but will be placed to one side of the light. The amount of the lateral deviation, which represents in great measure the degree of fault, can then be estimated by placing correcting prisms with their bases toward the side where the second image seems to be situated.

If the fault be resident in one or more of the vertical muscles, the test is practically the same, except that the prism which is used to produce the doubling must be much stronger and placed upon its side. Especially is the former so if the test be tried upon some object that is situated but a few centimetres in front of the eye.

If desired, resort can be had to one or more of the many devices that are made for studying the various positions of artificially produced double objects. For example, the Maddox rod and its modifications, which, when placed before one eye, cause a small flame to be transformed into a long line of light, may be used. This, which gives such an object to the patient that prevents any fusion with the untransformed image of the flame as seen with the other eye, is a most valuable form of apparatus. Here, if the line of light bisects the undisturbed image of the flame, muscle-balance in the meridian that is tried is obtained. If the line of light fails to divide the flame into two equal parts, correcting prisms may be placed in front of the rod. Phorometers, revolving prisms, etc., of the many varieties now in common use, may be employed just as time, fancy, and inclination impel.

Should the muscular insufficiency be more marked, its presence may be determined by simply placing a plain red glass before one eye of the patient, and then have the patient gaze at a distant light. If any pronounced error exists, he will ofttimes declare the presence and relative position of a double image of the light, the one being properly tinted and the other appearing reddish. Just as before, the mere superimposing of correcting prisms will give the degree of any muscular error. If desired, these discrepancies may be noted upon appropriate registers placed at definite distances by the patient himself, thus saving the time and trouble of correcting the error by prisms.

In all of this work it must be remembered that the different muscles have marked differences of strength, so that the same amount of nerve power that is lost in any two muscle-groupings may produce different results of loss of muscle action. Thus the internal rectus muscle is by far the strongest of the external series, whilst the superior and inferior, with possibly the obliques, are the weakest.

To estimate roughly the relative powers of any one of these muscles, increasing strengths of prisms with their apices pointed toward the muscle that is desired to be studied may be placed before the patient's eye whilst both of his eyes are open and gazing at a distant light. These are to be continued until the light becomes doubled. The amount of prism strength that is necessary to be used can then be gotten and compared with the amount that the muscle is said normally to overcome.

In some of the irritative forms of spasmodic contraction of both the extrinsic and the intrinsic groupings of the ocular muscles diagnosis of the local condition is comparatively easy from concomitant symptoms and the behavior of the muscle-groupings.

Care must always be taken to note the conditions under which the testing is done. The position of the prism, its strength, and the eye before which it is placed should be registered. Whether the test is employed whilst the eyes are gazing at a distant or at a near object, the exact distance of the test-object if it be nearer than five metres, and the form of the apparatus employed should all be taken into account.

In some varieties of nerve disease, as, for instance, in disseminated sclerosis and in many congenital defects of the eyes, such as cataract or faulty development of the retina and optic nerve, the eyeballs are in a state of constant motion. This, which is known as *nystagmus*, is in some cases, especially in the congenital form, rapidly increased when the patient is excited, or when he is made to gaze intently at any object. Rarely, it may be voluntary in type. Its presence, together with the rapidity and direction of the oscillatory movements, should always be noted.

If the paralysis be situated in one or more of the lid-muscles, the want of action of the paralyzed muscle and the over-result of the action of the opposing muscle will be plainly apparent at a moment's glance. One of the most frequent forms of this variety of affection is known as *ptosis*, or drooping of the upper eyelid. It occurs as the result of paralysis of the levator palpebræ muscle. It is both congenital and acquired. If of the former variety, it is generally due to nuclear disturbance and is bilateral in type. It is of frequent occurrence in certain forms of hysteria and in congenital ataxia. It is also often present in anæmic and badly nourished women with uterine disorder, and whilst passing through or approaching the period of menopause. This form, by reason of its generally appearing upon awakening from a long sleep, and hence, as a rule, in the morning, is known as *matinal ptosis*.

The upper lid droops to varying degrees over the eyeball. Thus, for example, if there be paralysis of the cervical sympathetic, the fibres of Müller are affected, causing but a slight drooping, with but little impairment of movement of the lid. If, in pronounced cases, attempts are made by the patient to raise the upper lid, the brow and forehead become wrinkled, showing the compensatory action of the occipito-frontalis muscle.

Lagophthalmos, or drooping of the lower eyelid, is readily recognized by the undue prominence of the eyeball, the exposure of the lower portion of the sclerotic, the eversion of the inferior lachrymal punctum, and the consequent *stillicidium*, or flow of tears. The patient is unable to close the eye. The condition is a frequent accompaniment of both peripheral and central nerve disease, and depends upon a paralysis or a partial loss of action of the orbicularis oculi muscle through the disturbance of its governing nerve—a branch of the seventh pair. It forms one of the most prominent symptoms in Bell's palsy, and is frequently of traumatic origin.

In some cases where there are other clinical evidences of so-called exophthalmic goitre, the upper lids will be found to lag when downward movements are given to the globes. This, which is known as the *von Gräfe sign*, is best obtained whilst the patient is lying upon his back and his head resting upon a pillow. While in this position the finger-tips are to be extended at about seventy centimetres from the patient's face and in the median line on a line with the forehead. After having the patient fixedly gaze with his two eyes at the finger-tip, the finger is to be slowly moved in a curved direction toward the patient's breast, when, if the sign be present, the eyeballs will follow the finger-tip and the upper lids will lag behind.

II. The Study of the Pupillary Phenomena. The size of the pupil is modified by many conditions of the nervous system. Independent of local changes, as in nearsightedness and glaucoma, where the pupil, as a rule, is large, it is more or less dilated in *amblyopia*, or deficient vision from nerve disorder, and *amaurosis*, or loss of vision from nerve degeneration. It is also found quite large in the earlier stages of aneurismal pressure upon the optic nerve, in paralysis of the sphincter muscle of the iris, in neuroses of functional type, and in peripheral irritation. Cerebral compression also produces it. During childhood and youth it is large; and, lastly, it must be remembered that the local application of certain drugs known as mydriatics renders it greater than normal.

The opposite condition, contraction, is found in farsightedness, in old age, and in retinal hyperæsthesia. Meningeal inflammation, cerebral irritation, and certain stages of convulsive seizures frequently provoke it. Certain drugs, known as myotics, when either locally applied or internally administered, produce it.

For proper study, the relative sizes and shapes of the two pupils should be carefully estimated during exposure of one or both of the eyes to light-stimulus. During this test the patient is to face a window or a light.

In many cases of nerve disease of degenerative type, as in epilepsy and general paralysis of the insane, the pupils are frequently unequal in size, irregular in outline, without any signs of local inflammation, and constantly varying. These changes can be satisfactorily determined by either looking at the pupils through an ordinary magnifying lens; or, better, by looking through a convex lens of twelve-to-sixteen-diopter strength placed in an ophthalmoscopic mirror.

Having determined the *equilibrium of the pupil*, as it is termed, whilst the eyes are in a passive state, as it were, the actions of the sphincter muscle of the iris under the various forms of stimulus that are usually applied to it are next to be tried.

As is well known, the sphincter muscles of the irides respond and the pupils become smaller when light-stimulus is thrown upon the retina. This, which is known as *iris response to light-stimulus*, is frequently affected in various forms of intracranial disorder. For example, in degenerations and pressures upon the optic nerves, chiasm, and optic tracts, in disturbances in the region of the floor of the fourth ventricle, and in changes in those strands of the third nerve which are intended to innervate the sphincter muscle of the iris. To obtain the response, the patient should be made to keep both eyes open. Then, whilst he gazes directly ahead into space, the eyes are to be alternately covered and exposed. If the muscle responds properly, the corresponding pupil will be immediately contracted when the eye is brought into view.

Where, although there is free movement of the iris to the so-called accommodation-reflex (see below), the pupil remains fixed at some definite area whilst light is thrown upon the retina, the condition is known as the *Argyll Robertson pupil*. This peculiar want of response of the iris to this form of stimulus is generally seen in disease of the posterior columns of the spinal cord, and is believed by many to be produced by innervation of the radiary fibres of the iris, upon which as a lever, as it were, the sphincter muscle of the iris membrane plays. As just explained, the iris is able to respond only when the patient makes efforts to look at near objects. The symptom is readily gotten by alternately exposing and covering the two eyes to sudden flashes of strong light-stimulus whilst the eyes are made to gaze into darkness.

Iris-reflex to accommodation, which is rendered manifest by a contraction of the pupil when the eye is made to look at a near object, is the next most

important sign. If broken, as it is in cases of nuclear origi
rare instances of spinal disturbance, the iris muscle will fail t
lens is sufficiently increased in strength by the action of the ci
allow small objects at short distances to be properly focused u
(known as the act of accommodation). To determine whethe
in proper working order the size of the pupil is to be accura
whilst the patient is made to gaze at a distance. A pencil or
to be suddenly placed directly in line with the distant objec
about 35 to 40 centimetres' distance from the eye. If the re
the pupil will immediately contract and remain so as long as th
ing of the eye upon the near object remains.

A third and most important reflex-act of the iris is that
reflex to convergence. In reality, it is a mere addition to the
Here two eyes are simultaneously acted upon instead of one, th
third factor—the associated impulse of the two internal rectus
play. To obtain it properly, the two eyes are made to gaze sim
a distant object. The pencil-tip is suddenly brought in the mid
face before the two eyes. If the impulse be correctly applied, t
take place; correct focusing of the two eyes, with simultaneou
of the pupils and inward deviation of the two eyes. It is s
turbed in some rare cases of beginning or ending nuclear palsy
mencing isolated paresis of the internal rectus muscles.

A fourth reflex of the iris consists in a peculiar dilatation
which is produced by irritation of the sympathetic nerve. It
the *sympathetic iris-reflex.* It can readily be produced in man
pricking or pinching the cutaneous fibres of the cervical sympa
back of the neck. Its absence in some cases is designative of
ance in the reflex act. In other neuroses, especially of a degen
it will be noticed that the pupil becomes alternately larger and
out any seeming impulse being given to the organ. This pe
movement may occur several times before the pupil becomes fi
be easily recognized by an ordinary magnifying glass whilst t
made to gaze out of a window. It is of great value in the recog
earliest degenerative stages of posterior spinal sclerosis, and mi
termed *pupillary,* or, better, *iris-innervation sign.* At times, it
prominently brought into view by having the eye turned as fa
as the patient can possibly move it, when the ataxic movements
appear.

In some types of nerve disease where there are lesions of
nature, as in the earlier stages of some of the more common form
eral neuritis, it will be found that a spasmodic contraction fo
momentary dilatation of the pupil, known as *hippus,* takes
symptom can be best studied by the aid of a magnifying lens
patient is made to look out into diffuse daylight. It has been seen

In some cases of hemianopsia (*vide* page 47), where the condu
of the reflex arc between the retinæ and the sphincter muscles o
are affected, as in pressure upon and in degeneration of certain po
optic chiasm or of one of the optic tracts, the pupil fails to bec
when light is thrown upon the blind area of the field of vision.
tom, which is known as the *Wernicke sign,* or, more properly, *hem*
inaction sign, can be gotten in various ways. The easiest and the
method found of use by the writer is, first, to illuminate faintly
the patient's eye by a beam of light obtained from a piece of pl
glass that has been illuminated by a light placed over and back of t
head. This amount of feeble illumination does not seem to dist

ting of the sign. Whilst the eye is illuminated a concave mirror, as found in one of the common forms of ophthalmoscope, is held so as to bring a strong focus of light from various points in the periphery of the field of vision to play upon the pupil. In this experiment it is always best to commence at the blind halves of the field. If the sign be present, the pupil will not contract until the stray beam of light has reached the borders of the remaining fields of vision—showing that the break in conduction has been in the sensorimotor loop of the visual apparatus, which, as we know, is anterior to the region of the corpora quadrigemina. This fact, in association with the hemianopsia, therefore locates the intracranial lesion either in the optic chiasm or in the optic tract.

At times many important data as to the question of action of the ciliary muscle in grave nerve cases, especially where the pupil is dilated, might be obtained by resort to the so-called *catoptric test.* The method is quite easy. A narrow beam of light from a candle or a taper should be allowed to fall obliquely upon the pupillary area. If this area be magnified by a strong convex lens, three images of the candle-flame will be seen. By now asking the patient to attempt to look at a near object, the more deeply situated images of the flame will, if the pupil does not contract too much, be found to separate, showing that the lens has increased in convexity, which must have been produced by contraction of the ciliary muscles.

If the pupil be too small for this procedure, the plain mirror retinoscope may be employed to study the presence or absence of the reversal of movement of the reflexes as the instrument is gradually approached to the eye. If a reverse movement takes place when the mirror comes close to the organ, accommodation exists, thus proving the presence of ciliary muscle action.

III. The Study of the Condition of the Interior of the Eye by Magnifying Lenses and the Ophthalmoscope. Practically, nearly the whole of the interior of the eye, with the exception of a small zone around the ciliary body and muscle, can be laid bare for examination by one of these two methods. The first plan consists in brilliantly illuminating the anterior part of the organ by a convex lens and looking at the illuminated portion through a magnifier. By it, the layers of the cornea, the aqueous humor, the iris, the greater part of the lens, and the anterior portion of the vitreous humor can be brought distinctly into view. The second exhibits, in addition, the greater part of the vitreous humor, the posterior two-thirds of the choroid and retina, and the optic nerve-head. Congenital anomalies, signs of active and chronic inflammation, venous and arterial engorgements, depositions and atrophies, all can be revealed and recognized by these methods. The former plan, which soon becomes comparatively easy of performance by constant repetition, is known as *lateral, oblique,* or *focal illumination,* and should be carefully employed whenever thought necessary.

The latter plan, known as *ophthalmoscopy,* is much more difficult, and, in fact, constitutes an art in itself. For this, two methods are usually employed. One is known as the *direct method,* as the object within the eye is looked directly at, and the other is described as the *indirect method,* because an aërial image of the object is what is looked at. In the indirect method the instrument is held at some distance from the patient's eye and the organ gazed at through an intervening convex lens. In the first, the object is seen erect and in its true position, thus giving rise to the term *erect* or *upright image.* In the second, or indirect plan, the aërial image of the object in the eye is both inverted (thus giving the term *inverted image*) and reversed.

In practice it is always best to study the various layers of the eye, commencing at the cornea. To do this quickly with the direct method, strong convex lenses are to be employed first. These are to be gradually weakened until the level of the fundus or background of the eye is reached. The disk or optic nerve-head is first to be sought for, and then the surrounding retina and choroid are to be carefully studied. If the indirect method is employed, the positions of the ophthalmoscope and the convex lens are to be changed in relation to the patient and the observer.

In health the disk appears as a pinkish-whitish-gray plaque. Its borders are distinct, and it is bounded by a whitish ring which is edged with pigment. From or about its centre a series of light-colored arteries escape and subdivide as they pass out into the retina. Into this same area about the same number of dark-tinted veins which have arisen from much finer stems can be seen to pass. The general color of the nerve-head is of a dull gray-red. The eye-ground, which is stippled, becomes darker and more compact as it gets nearer to the region of the most distinct vision. The macula itself often appears, especially by the upright image, as a longitudinal oval ring of a faint yellowish tinge, whilst the fovea glimmers at the bottom of the macula as a pale straw-colored dot of reflex.

Should there be inflammation of the nerve-head, as in descending optic neuritis from meningitis, the disk edges will become hazy, the surface slightly and irregularly elevated, and its circulation will be rendered visible, giving it an angry, swollen, and over-vascular appearance. There is turgidity of both the veins and the arteries of the retina, notably the former. The vessels are tortuous and the veins throb and pulsate upon the slightest occasion. The general color of the fundus becomes more rosy in hue, whilst in the graver forms flame-shaped hemorrhages and haze-like areas make their appearance.

If a true blocking and distention of the intravaginal spaces in the optic nerve sheaths just posterior to the globe occurs, as is so common in cerebral tumors and other coarse intracranial lesions of infectious type, the nerve-head will be pushed far forward into the interior of the eye, giving rise to the condition known as *papillitis* or *choked disk*. Here the picture is typical. The nerve-head stands out into the vitreous. The retinal vessels are hidden in many places in the disk and surrounding retina as they pass to and from the nerve-head. Fine capillary and numerous clumplike and fan-shaped hemorrhages are seen irregularly distributed in and around the swollen nerve. The veins of the retina are engorged, twisted, and turbid, with deeply pigmented blood, whilst the corresponding arteries are reduced to mere threads of pallid blood.

Later, degeneration changes, both cicatricial in type and fatty in nature, take place, giving the eye-ground characteristic appearances which in many instances are almost unmistakable as to the former condition.

IV The Study of Vision. This part of the subject naturally divides itself into two parts. The first, that known as *central vision*, or, better, the *vision of fixation;* whilst the second is that which is ordinarily termed *excentric* or *peripheral vision*. The former teaches much as to the transparency of the aqueous, lens, and vitreous humors and their equality of structure and regularity of surface. It immediately shows the power of adaptability of the lens, and thus indirectly gives the condition and working capabilities of the ciliary muscle and its accessories; it registers the physiological power of the most important and most frequently employed sensory elements of the retina; and, lastly, it distinctly states the degree of working power of both the conducting apparatus to the cortical layers and the visual elements of the cortex themselves.

To obtain the proper answer, so-called *test-letters*, which by experiment and study have been found to be visible at definite distances by great numbers of presumably healthy eyes, are arranged upon charts. These cards are suspended upon well-lighted walls, and the patients are requested to name the smallest lines of letters that are visible to each eye separately. If the line of type named be that for which the distance used is intended, the *visual acuteness*, as it is termed, is said to be normal. If vision be less, the degree of the remaining amount is to be noted in an appropriate manner.

When color is employed, care must be taken that the amount of the color area that is exposed is properly gauged for the special color used. This is necessary on account of a vast difference between the visibility of the different colors at the same distance.

In testing near objects a double purpose, as before hinted, is served. Not only is the amount of central vision obtained, but the ability of the so-called accommodation or focusing apparatus of the eye is determined. This is accomplished by means of small ordinary printed matter or reading-types, as they are termed. Trying each eye separately, the nearest and furthest points of distinct vision for the smallest type that is visible are to be registered.

If vision be so low that the letters cannot be deciphered, an outstretched hand can be held up at varying distances from the patient, and request made to state how many fingers are raised. If vision be still lower than this, various forms of concentrated or artificial light may be thrown upon the patient's open eye, whilst he is desired to state the direction from which they appear.

In the second form of study the extent of the *field of vision*, as it is commonly termed, and the general condition of its entire area are obtained. Several plans are pursued, but for the sick-room and in situations where complicated apparatus is not easy of access, small squares or circular areas of color pasted upon large black objects are extremely useful. Whilst the patient is made to gaze fixedly at some object, as, for example, a mark upon a blackboard or even a small square of white held directly in front of the eye that is to be examined, the color area is to be gradually moved in from the periphery until it is first seen. This point is then to be registered. Repeating this at a dozen or more meridians around the central object, the various registries are to be connected together by a series of concentric lines, thus giving a map, as it were, of the boundary of the field of vision for the color that has been tried. After this has been done the color square is to be carried directly inward until it covers the object that the patient is gazing at. Note is then to be made whether it becomes dim or is lost at any place. In the office and clinic-room, where most careful work is necessary, more accurate results can be obtained and quickly registered by one of the numerous so-called *perimeters*.

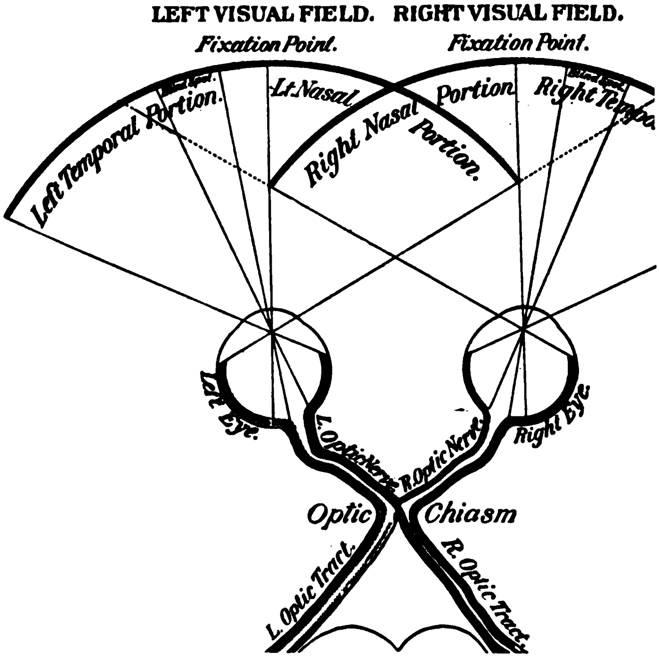
The extent and forms of the fields of vision are subject to many variations; in fact, they become one of the most valuable and certain symptoms in the differential diagnosis of grave nerve disease, and frequently give answer as to the situation and significance of ocular and cerebral disturbance. Irregularly and markedly contracted into a series of indentations in many forms of retinal and choroidal inflammation; ever changing and most confusing to beginners in numerous functional neuroses; dotted with scotomatous areas in coarse changes in the intraocular coats; darkened and even annihilated in the areas of central fixation, as in macular changes of retrobulbar disturbances; and split into almost symmetrical quarters and halves known as tetranopsic and hemianopsic field defects by chiasmic, optic tract, and cortex lesions—it can be readily seen how much information can be gotten by careful work in this direction.

Briefly, the hemianopic defects are subdivided into the following varieties:

(*a*) Right homonymous hemianopsia, which means that the area of either complete or incomplete loss of vision includes all that portion of each visual field that is situated to the right side of the fixation object, thus showing that there is some break in the sensory fibres which are distribu halves of the retina. This condition, as can be easily seen dicates that the disturbance, if not multiple, is situated in the of the optic chiasm.

(*b*) Left homonymous hemianopsia. This is the opposite c first variety. It signifies that the imperfection of vision in portion of each visual field which is situated to the left side fixation, thus showing that there is some break in the senso are distributed to the right halves of the retina. This condi seen by the figure, shows that the disturbance, if single, is in posterior to the optic chiasm.

FIG. 21.



(*c*) Bitemporal hemianopsia or lateral heteronymous hemi is a condition of fault in the two visual fields where the imperfe the temporal or outer halves of each field of vision, thus showi is some break in the sensory fibres which are distributed to the halves of the retina. This condition, as can be seen by the figu the disturbance is situated either directly above, below, in front the optic chiasm itself.

(*d*) Binasal hemianopsia or medial heteronymous hemia means that there is a fault in the two visual fields which includ or medial halves. It shows that the sensory fibres that are dist temporal or outer halves of the two retinæ are not acting. which is very rare, and which means loss of binocular vision t necessarily signifies, as can be seen by the figure, that there mo a multiple lesion.

The superior and the inferior varieties of the hemianopic fiel practically subdivided into the same groupings, except that here the terms superior and inferior are substituted for the terms temporal and nasal.

At times it may be necessary to obtain a gross estimate of the condition of the color sense. This is easily done by any of the simple modifications of Holmgren's series of wools. The skeins being thrown promiscuously upon the table, a test wool is handed to the patient. One eye is to be tried at a time. Without naming the color, the patient is requested to choose the nearest match to the test skein. One or two trials with the test wools of rose, green, and red will soon elicit the character of color vision possessed by the patient.

V. The Study of Trophic and other Changes in the Eyeball and its Adnexa. In addition to the above methods for detecting the motor and the sensory anomalies of the organ, it becomes important to ascertain its trophic condition. Here is where the determination of the degree of deeply seated ocular and cutaneous sensibility are so important. Æsthesiometers of various kinds with especially adapted points for the cornea may be employed. Wisps of cotton carefully twisted and coiled upon the corneal surface without touching the lid-edges often can be made use of to determine the sensibility of this membrane. So, too, local thermometry on and around the anterior face of the organ can be frequently brought into good service in the ready recognition of atrophic and degenerate areas from peripheral nerve disease, such as is so often found in the later stages of herpes zoster-ophthalmicus. Where local inflammation is present, as in chronic irido-cyclitis or in that dread disease, sympathetic irritation, more decided pressure with the finger-tip upon or over the affected parts frequently evokes evidences of tenderness and of pain.

Again, the deep and superficial vascularity of the anterior segment of the globe should be looked at. The presence of the circular rim of fatty degeneration around the edge of the cornea should be sought for. The condition of the lachrymal apparatus; marks of inflammatory thickenings even in the upper and lower culs-de-sac; the degree of tonicity of the globe, which can be best done empirically by pressure with the finger-tips through the overlying lid; the depth of the anterior chamber as expressive, for instance, of lens swelling in the shallowed chamber and low-grade ciliary inflammation in the deepened one—all should be noted.

The relative prominence of the two eyes should always be ascertained. To do this various contrivances have been made, but here as elsewhere visible expressions can be better depended upon than mere mechanical apparatus. Palpation and auscultation for the determination of deeply seated orbital bruit are also of value.

Further, cases arise where it becomes necessary to determine whether the symptoms are real or are intentionally or unintentionally counterfeited. Here there is a large field of investigation which can be multiplied according to skill and ingenuity. Prisms, secretly arranged so as to produce seemingly true double images, or to provoke incorrect positions of previously unseen natural objects, can be used. Lenses so placed and mydriatics so employed as to produce unrecognized changes of focusing power in the patient's two eyes so that he is not made aware of which eye is actually in use, are of great value; whilst interposition of opaque objects and the use of apparatuses by which the patient is unwittingly made to employ his averred bad eye; and even in complete general anæsthesia, carried sufficiently into effect as to render the patient confused when placed in supposed dangerous situations, etc., during the first stage of recovery from its effects, are all useful; all can frequently be successfully applied. The induction of an hypnotic condition might be of value in some of these cases.

In all of this work each and every ocular symptom that may appear of importance to the proper elucidation of the cause of disturbance should be studied. Here the intelligent association of the entire grouping of the ocular conditions are alone useful for correct data. Everything that is relevant should be noted, accurate register of passing symptoms should be taken, and the uncertainties of cases so constantly repeated as to obviate all past difficulties. If this be done most carefully and painstakingly, it will be a source of great happiness and pleasure to realize that all of the findings have reached a plane of certainty that other observers through carelessness and consequent ignorance have sought for in vain.

CHAPTER II.

GENERAL MORBID STATES OF THE NERVOUS SYSTEM.

By F. X. DERCUM, M.D.

NEURASTHENIA.

Among the first of the various morbid states of the nervous system to claim our attention is the one now generally known under the name of neurasthenia, and for which the laity use the expressions "nervous exhaustion" and "nervous prostration." The fact that it is widely diffused through almost every community renders its study of peculiar importance.

Strangely enough, an impression exists not only among the laity, but also among physicians, that the affection which we are about to consider is a comparatively recent one; is one which is the outcome of the high pressure and the great wear and tear of modern civilization. That this is an error a brief study of the subject soon convinces us. Certainly, all peoples ancient, as well as modern, have been subject at various times to the same etiological factors which we know produce neurasthenia to-day; and, while space does not permit us to refer to the pages of history to support us in this assertion, there can be no doubt of its truthfulness. Medical men have recognized, though vaguely, the existence of this affection ever since the sixteenth century, and in the seventeenth and eighteenth centuries allusions to this affection and various descriptions of it continue to appear.[1] However, the early descriptions are obscure and permeated by grotesque pathological theories. Moreover, they make no distinction between neurasthenia and the related states of hysteria and hypochondria. To Robert Whyte, of Edinburgh,[2] belongs the credit of first differentiating between these three affections and of more or less correctly outlining the condition now known as neurasthenia. This he did as early as 1765 in his work on *Nervous Disorders*. He recognized three classes of these disorders, and in speaking of them says: "The complaints of the first of the above classes may be called *simply nervous;* those of the second, in compliance with custom, may be said to be *hysteric*, and those of the third *hypochondriac*." Unfortunately, he did not give the "nervous" condition that he recognized a more specific name. Sandras, in 1851, first used the expression "nervous state" (*état nerveux*), and for which Bouchut later substituted the word "nervousness" (*nervosisme*).

The first use of the word neurasthenia, as applied to this condition, is almost universally ascribed by medical writers to George M. B. Beard. This, however, is an error, the credit really belonging to another American physician, Dr. E. H. Van Deusen, of Kalamazoo, Mich., who first used the word in the supplement to the *Biennial Report to the Michigan Asylum for the Insane for 1867*, under the heading of "Observations Upon a Form of Nervous Exhaustion (Neurasthenia) Culminating in Insanity," and who says, among other things: "Our observations have led us to think that there is a disorder of the nervous system, the essential character of which is well expressed by the term given above, and so uniform in development and progress that it may with propriety be regarded as a distinct form of disease." He further gives a tolerably clear account of the affection. Beard's first paper upon the subject did not appear until 1869, in the Boston *Medical and Surgical Journal*. However, that the term neurasthenia is really older and was not invented by either of these physicians is proven by the fact that it is found in the first edition of Dunglison's *Medical Dictionary*, published in 1833, side by side with its German equivalent, *Nervenschwaeche*. Contemporary English, French and German dictionaries do not contain it. As to who actually invented the word is, therefore, still a matter of mystery.

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[1] For a detailed history of this portion of the subject the reader is referred to—Arndt: Die Neurasthenie, ihr Wesen, ihre Bedeutung und Behandlung, etc. Wien, 1885. Also, Müller, F. C.: Geschichte der Neurasthenie, in Handbuch der Neurasthenie. Leipzig, 1893.

[2] Whyte, Robert: Observations on the Nature, Causes, and Cure of those Disorders which have been commonly called Nervous, Hypochondriac, or Hysteric. Edinburgh, 1765.

Definition. Neurasthenia in its simplest form is a condition in which there is a more or less marked and persistent diminution of nervous energy, together with an increased reaction, mental and physical, to external impressions. In other words, we have two principal symptoms, nervous weakness and nervous irritability. Diminished resistance to fatigue implies diminished resistance to impressions from without. Weakness and irritability seem thus to be necessarily associated.

Neurasthenia, further, is an affection which does not limit itself to any part of the nervous apparatus, but affects it as a whole. Medical writers, it is true, have divided it, clinically, into cerebral and spinal types, according as one or the other group of symptoms predominate, but every case presents overwhelming evidences of a generalized affection. It is extremely probable that brain, cord and peripheral nervous system all suffer, although, of course, in varying degrees. Almost as a corollary it follows that the essentials of the symptomatology are always the same, but that individual cases vary greatly in their details.

Neurasthenia being an affection extremely generalized in character, it is often found associated with other affections, such as hysteria, hyphochondria and other functional and organic diseases. Unfortunately, this association has led to considerable confusion in the minds of medical men, and a few have gone so far as to deny the existence of neurasthenia as a separate affection. The fact, however, that simple and uncomplicated cases of neurasthenia are constantly met with—the fact that cases in which the symptoms of simple nervous fatigue, without any other etiological factors than overwork, present themselves—leaves us no alternative but to accord it a separate place in our classification. Moreover, as we progress in our study of this affection, we will find that the symptoms presented are those of a constantly recurring "symptom group," and that we have a well-marked clinical entity with which to deal. Pathological considerations, as we will presently see, also point in the same direction.

At the very outset of our study we find that neurasthenia resolves itself into two great groups: First, simple neurasthenia, and secondly, neurasthenia associated with other diseases. With these ideas before us let us turn our attention to questions of cause.

Etiology. As in other affections, the causes of neurasthenia can be separated into predisposing and exciting. Among the predisposing is, first and foremost, heredity. In a large number of the cases that come under our notice this factor is present. We frequently find on questioning the patient that either the mother or father, or perhaps both, have been "nervous" or very irritable, or perhaps have suffered greatly with headaches.

Only exceptionally do we find insanity, epilepsy or organic nervous diseases in the family history. Indeed, the occurrence of such a history, especially if it be marked, should make us suspect that the patient is not suffering from neurasthenia, but is in the developmental period of some psychosis or some form of organic nervous disease. The importance of this fact, in view of the prognosis, is such that it should not be overlooked. Neurasthenia, even when hereditary, has no factors in common with the "neuropathic constitution." The hereditarily neurasthenic individual is merely one who makes his start in life with a nervous system in which cell wear and tear take place readily, and in whom the recuperative power is feeble. He has no innate predisposition to insanity, sclerosis or other nervous diseases, as has the hereditarily neuropathic subject. Closely allied to the question of heredity is the occurrence of various diseases in the parents which are calculated to depress vitality, such, for instance, as tuberculosis and syphilis; that is, the children of such parents, without having tuberculosis or inherited syphilis, may readily become neurasthenic. Strangely enough, too, a history of gout in the ancestry is not uncommon. Whether gout directly predisposes the offspring to neurasthenia, or whether the fact that a gouty ancestry is often synonymous with a dissipated and alcoholic, and therefore enfeebled, stock, are questions that we cannot, of course, determine. The clinical relationship of the gouty ancestry to neurasthenia, however, remains.

That the abuse of stimulants by the parents, great age of the father, and, in fact, all enfeebling and depressing influences in the ancestry, tend to produce neurasthenically disposed offspring, is a matter readily comprehended. Consanguineous marriages, it should be remarked, are likely to produce a neuropathic, rather than a neurasthenic, stock.

Next in importance to heredity is education. A child, otherwise healthy, is often brought up so loosely, is so indulged in every whim and caprice, is so pampered and petted, so thoroughly "spoiled," that when circumstances force the grown-up lad or adult woman to face the serious questions of life, energy, will-power, judgment, self-control, nervous strength in all its forms, are sadly lacking. A rude awakening and a succession of painful crises, together with the suffering they entail, are potent factors in the development of neurasthenia. On the other hand, children who are brought up harshly, or too rigidly; who are denied the ordinary pleasures of childhood and youth, and upon whom a too close application to study, and perhaps to physical labor, has been forced, are also likely to develop sooner or later into neurasthenic men and women. Further, an education that provides a child simply with desk instruction, with books to the exclusion of physical exercise; or an education that taxes a child too much in one direction, such as music or drawing, is also attended by grave dangers. The absurd and often cruel custom of forcing prolonged musical training, requiring many hours of daily practice, upon children who have no special or natural musical talent, and who have, in addition, all the other tasks of school, is only too common in this country. The nervous system of the child, like a steel blade untempered, is soft and unresisting; tempered too highly, it is brittle and fragile, and tempered unevenly, is full of weaknesses.

Among predisposing causes we should also mention occupation. Persons whose business is attended by great strain and excitement, by worry and uncertainty, such as inventors, speculators or promoters of great enterprises, are very apt to become neurasthenic. Other occupations, even if they entail a great amount of work, provided there be "emotional rest," do not of themselves entail neurasthenia. Causes relating to the hygiene of occupations are not here considered.

Social position does not seem to exercise a markedly predisposing influence.

Many of our patients are, it is true, of the wealthy classes, a doors of our hospitals is an equally large number of indigent su ing for admission.

The married or unmarried state markedly influences the ten rasthenia. A much larger number of neurasthenics are unma is true alike of men and women. In single men special cau absent in the married, such as masturbation, sexual, alcoholic, cesses, are present. In single women, though excesses and infr moral law are much less frequent, the unaided, and often unequ the world is to blame. Sometimes, as we will see, simple sexual of itself a predisposing cause.

Some writers claim that neurasthenia is more frequent in men. however, remember that a very large number of female neur into the hands of gynecologists, and that therefore statistics o especially from private practice, are not reliable.

Neurasthenia is, as would be expected, most common during most active life—namely, from twenty to forty-five or fifty. claimed by various writers (Bouveret, von Hoesslin) that race is a factor in neurasthenia. It is stated that Hebrews and Slavs liable. Regarding Hebrews, the peculiar conditions under whi lived for so many centuries may in part account for this conditio this explanation, of course, does not apply.

Among the exciting causes of neurasthenia is, first and forem The capacity of every individual for work is limited. It vari different persons, and in estimating the question of overwork t borne in mind. The character of the work also is very impo connection. What one man can execute with ease may be a ver labor to another. Thus, one of the most typical cases of neuras it has been the writer's fortune to see, occurred in the person of who gave up the work of mining for the more pleasant and lucr of a country schoolmaster. In the mine he had been perfectly new position, however, he became rapidly neurasthenic, and thi face of the fact that the character of the work required only t mentary knowledge and that the number of pupils was very limi

It is commonly believed that neurasthenia is the outcome especi tal overwork. Sometimes, however, overwork that is purely phys continued, leads to neurasthenia of the most intractable form. T we will presently return.

It is surprising how much work can really be accomplished by men without neurasthenia being induced. An added factor, ho tional excitement, is very potent for ill; indeed, emotional unrest a cause second in importance only to overwork. Excitement of a character—for example, the excitement of success—acts rather lant. It is excitement of a depressing character, such as wor appointment, vexation and grief, that acts deleteriously. An example of the power of overwork and of depressing emotions neurasthenia is found in nursing. A mother nursing her sick ch a long illness is not only subjected to great physical strain by lo but especially by the prolonged emotional strain entailed by uncertainty regarding the recovery of the patient. Contrasted w paid nurse, who discharges the same duties and loses an equal sleep, experiences but slight strain and remains well. Her emoti secondarily, if at all, called into play.

Various causes that tend to debilitate tend also to produce ne Among these may be mentioned excessive child-bearing and prolo tion. Nervous exhaustion is also apt to follow certain acute illnesses, such as typhoid fever, malaria, and especially influenza.

One of the most powerful and most frequent of the exciting causes of neurasthenia is trauma. Accidents of all kinds, especially if they be accompanied by fright, such as is the case in railway accidents, tend to produce a neurasthenia that is often profound and very persistent. Two factors are generally present in such cases: first, the physical injury, and, secondly, the emotional shock. They are considered in detail in Chapter IV. Among other exciting causes are all forms of dissipation and excess; everything that exhausts the nervous system tends to produce neurasthenia. The excesses of the votaries of Bacchus not only entail loss of sleep, but also the weakness consequent upon over-stimulation. Later, after the alcoholic habit is fully acquired, the symptoms of neurasthenia are complicated by those of chronic alcoholic poisoning. What is true of alcohol is true of tea, coffee, and tobacco. Sexual excess is also a potent factor. This applies, of course, to both sexes. A very profound, bed-ridden neurasthenic, for many months under care of the writer at the Philadelphia Hospital, was a woman who had been at one and the same time the mistress of three different men. Sexual gratification by unnatural means, it need hardly be said, is also very injurious, although regarding masturbation, it must be admitted that the habit is sometimes carried on to a surprising extent and yet the individual remains apparently well. This is, however, the exception and not the rule. An exceedingly dangerous practice is that which, we fear, is only too common among married persons—namely, the interruption of the sexual act by the withdrawal of the male at the moment of ejaculation. An equally harmful practice is the unnatural prolongation of the act.

As opposed to sexual excess, sexual abstinence every now and then produces neurasthenia. The number of instances is, however, we are convinced, very small, and it probably occurs only in cases of long-continued and suppressed excitement, such as sometimes exists in engaged persons, especially when the engagement is of long duration.

The above causes embody briefly the various factors producing simple and uncomplicated neurasthenia, *neurasthenia simplex*.

A moment's reflection will convince the reader that nervous exhaustion is frequently the outcome of various other functional and organic affections. Thus, a person who has tuberculosis may present, in addition to the specific symptoms of that disease, also those of neurasthenia. This is likewise true of anæmia, chlorosis, and other nutritional disturbances. Again, functional or organic disease of the stomach or intestines or of the generative apparatus, may so affect the general nutrition as to produce symptoms of nervous weakness. It is very plain that a neurasthenia arising from any of these causes differs radically from *neurasthenia simplex*, and that its natural designation is *neurasthenia symptomatica*.

Symptomatology. Contrary to our experience in many other nervous diseases, the patient presents no striking physical peculiarities. There is no gross abnormality in the walk, no local palsy, no muscular spasm, no incoordination of movement. There are none of those obvious features that so often enable us at a glance to relegate a given case to this or that group of diseases. Frequently it is only after our patient begins to talk that we gain an idea of the character of his affection. He begins by telling us how he *feels*, and we soon become impressed with the *subjective* character of many of the symptoms. Physical symptoms are present, it is true, but we soon learn that in the majority of cases they are brought to the surface only by systematic examination. The patient is rarely talkative. Only after repeated questioning we learn that he is "nervous;" that he gets easily excited and "upset;" that he can no longer work as he did; that he gets tired before the day is half over; that he can hardly sleep at night; that when he awakes in the morning he feels completely exhausted; that his head aches; that his heart palpitates; that his memory is impaired; that he has to force himself to eat; that his food lies heavily in his stomach; that his bowels are constipated; and so on and on through a long train of distressing symptoms. To these may be added a long list of sexual troubles.

One could hardly expect such a patient to be cheerful. As a matter of fact, he is generally depressed and worried about himself. Often, too, he gets anxious and fearful if left alone; or, again, feels oppressed in company or crowds. Sometimes he feels afraid to stay in the house; at other times afraid to stay in the open street. In other words, he may have, in addition to his general symptoms, those of an unnatural fear.

In outward appearance neurasthenics differ greatly. If marked depression exists it may affect the walk, the attitude, the facial expression, the manner, and, in short, the entire bearing of the patient. If depression be absent or but slight, all of these symptoms may be wanting. Again, it not infrequently happens that the neurasthenic subject has an excellent physical development, and this may give his friends the impression of his having very good health. Men of fine physique who have been the victims of overwork may bear little or no outward trace of their breakdown, at least for a time.

We will see as we progress that the symptoms of neurasthenia are of two kinds: first, *essential symptoms*, and, secondly, *adventitious symptoms*. The first are those which essentially attach to the condition, and always present the phenomena of weakness and irritability. The others are secondary outgrowths of the various disturbances of function, and may or may not be present. This division into primary or essential symptoms, and secondary or adventitious symptoms, we will find very useful. It simplifies a subject otherwise difficult and complicated, and renders our conceptions clear and accurate.

When analyzed relative to their distribution the symptoms resolve themselves into:

Motor,
Sensory,
Psychic, and
General Somatic.

MOTOR DISTURBANCES. The patient almost invariably complains of muscular weakness. In the large majority of cases this sense of weakness is founded upon actual feebleness of the muscles. If the patient be asked how far he is able to walk he will frequently give as answer an absurdly small distance. Very often he will volunteer the information that he cannot stand long, that he grows so tired that he is compelled to lie down. When tested by the dynamometer it is found that his grip is markedly weak. Occasionally, however, to our surprise, the grip is normal; but in this instance, if the patient be made to repeat the grip a number of times in succession, we find that it quickly grows weaker, and in a little while the index of the instrument may even fall to zero. In other words, the patient may be able by sudden effort to simulate a normal condition, but he cannot maintain it. Symptoms of fatigue soon make their appearance, and the weakness becomes very evident. This incapacity for prolonged muscular exertion may be regarded, as we will see, as representing one of the fundamental features of neurasthenia.

Weakness of the legs and back is complained of more frequently than weakness of the arms, but in all cases the condition is found to be more or less general. Decided local weakness is rare, and when found should suggest hysteria or other functional or organic disease. Actual paralysis is never observed in simple neurasthenia.

A phenomenon observed with sufficient frequency to be worthy of note is muscular tremor. In the experience of the writer, it is absent in the majority of cases. It is more often seen when the neurasthenia is the result of trauma or fright. As might be expected, it becomes more pronounced with emotional excitement. It is rather a fine tremor, and is often inconstant and irregular. Occasionally it is betrayed by the handwriting. It may be limited to the hands or may be widely diffused over the muscles of the limbs and trunk—notably in traumatic cases. Allied to this tremor are spasmodic and irregularly recurring contractions of small bundles of muscle fibres, either in the truncal muscles or in those of the extremities. They closely resemble the fibrillary contractions seen in the muscles in poliomyelitis, and, like tremor, they are more frequent in cases of traumatic origin. Slight spasms or twitchings are occasionally seen in the orbicularis palpebrarum, in the fibres of the frontals, and elsewhere about the face in neurasthenic subjects. Occasionally a tendency to twitching or brief spasm exists in some of the muscles of the extremities. It is perhaps most frequently met with in the muscles of the calf.

The tendon reactions of neurasthenics are likewise important and interesting. The knee-jerks are in the majority of cases decidedly exaggerated; but if an attempt be made to elicit the reaction repeatedly and in close succession, marked diminution of the response and even disappearance may be observed. In other words, the nerve centres concerned in the production of this tendon reaction may become exhausted. In some cases, instead of being exaggerated at the outset the jerk may be normal or even less than normal. Should the knee-jerk be altogether absent, and remain so during tests made by Jendrassik's method of re-enforcement (see page 28) we should at once look for signs of organic disease.

Exaggerated tendon reactions are also noted elsewhere. The tendons of the triceps, of the biceps, and the various tendons at the wrist may react in an exaggerated manner. An ankle clonus may also be present, although it is never so marked nor so prolonged as we find in organic disease of the cord. It is generally of less extent and rapidly exhausted. Regarding these tendon phenomena in neurasthenics, it should also be stated that they are much more marked during excitement. This is doubtless the reason why they vary from time to time. Thus an ankle clonus present at one occasion may be absent at another; and two observers examining the same patient in immediate succession may obtain different results because the first has for the time being exhausted the tendon reactions. These points are of importance in view of the medico-legal aspects of the traumatic form of neurasthenia.

At times the muscles also respond in a manner similar to the tendons when tested by percussion. Rarely the nerve trunks are also mechanically irritable, so that tapping will produce slight contractions in the muscles supplied.

SENSORY DISTURBANCES. The sensory disturbances of neurasthenia are exceedingly numerous and varied. Being subjective, many of them are incapable of any but indirect verification. They range from vague, generalized, abnormal feelings, often incapable of description, to others which are definite in character and limited to certain portions of the body. Thus, some patients suffer from a vague and general feeling of distress that can only be described by saying that they "feel badly all over." Others, again, will complain of a general feeling of fatigue, and some of a sense of profound exhaustion. Very often the patient tells us, "I feel tired all the time," and sometimes

adds, "I always feel like lying down, and am not comfortable
This feeling of general fatigue is rarely absent in simple neur
profound cases it may be hidden and obscured by a host of oth
Among sensations less generalized are such as "lightness" or "
the head, or a sense of "constriction" or "pressure" about the
times the patient complains that his limbs feel heavy, as thoug
not be moved, and at other times as though they were withou
Vague sensations of tightness, of pressure, or other uneasiness ma
to this or that portion of the trunk.

At other times a feeling of uncertainty in making voluntary
present. Sometimes associated with this peculiar symptom,
independently, we have a feeling of uncertainty in regard to
objects. In such cases the patients complain of being giddy, and
present the symptoms of more or less marked vertigo. Mild for
ness are, however, more common in neurasthenia than actual ve
this symptom is directly related to the nervous asthenia there can
It appears to be due to the lessened vasomotor control of the cer
so that slight effort or change of posture affects the intra-cranial ci
is not impossible also that at times it is due to an actual weakness o
centres concerned in co-ordinating movement. It not only gives
distressing subjective symptoms, but now and then is sufficientl
disturb the equilibrium. Beard and Charcot both refer this sym
asthentic state. However, in vertigo from simple neurastheni
symptoms—that is, symptoms patent to the observer—are fa
present than in vertigo observed in organic disease of the cer
disease, or gastric disturbances. In the neurasthenic patient, th
sense of uncertainty may be so great that he will grasp surroun
to prevent from falling, but in reality he has comparatively litt
in maintaining his equilibrium. At least actual staggering and i
movements are rarely observed. When the severer forms are
nervous exhaustion, it is very probable that they are due to
gastric troubles. The latter are, as we will learn, exceedingl
Neurasthenic vertigo is sometimes almost continuous, and is t
distressing symptom, and one of the most difficult to treat. More
however, it occurs at irregular intervals. It may follow mental
effort, such as reading, writing, unusual bodily exertion, or even
acts as moving about in bed or the taking of food. It is a sympton
Further, too much stress cannot be laid upon the frequency of its
in neurasthenic subjects. It is extremely probable that the larg
of cases of vertigo, certainly the milder forms, are not due to ea
peripheral affections, but to exhaustion of the nerve-centres.

Among the less vague sensory symptoms are various pains whic
head, back, and limbs. They may be described in general as d
pains or aches, which in the milder forms suggest the sensations
fatigue. It is extremely probable, moreover, that even when
nounced, they are nothing more than exaggerated fatigue sensatio
ache is one of the most common of these symptoms. When pres
milder degree, it is described simply as a dull feeling or a dull a
a rule, it is not diffused over the entire head, but is seated in the o
the upper part of the back of the neck, or over the brow, or just
eyes. Of these regions, the occipital is by far the most freque
situations are occasionally described, but it is probable that in
other factors secondary in value come into play. These we will
discuss. Very commonly the headache is accompanied by the sen
sure or constriction, already mentioned, and the patient feels as

tight band had been placed around the head. Various other anomalies of sensation are occasionally present. For instance, patients sometimes complain of great heaviness, or of throbbing and "whirling" sensations in the head. It is extremely probable that these curious sensations are, many of them, if not all, the results of various intracranial circulatory disturbances, and are not directly fatigue sensations. They must, therefore, be regarded as *adventitious.* "Lightness" of the head is not, as a rule, associated with aching, but rather with giddiness.

Neurasthenic headache, when mild, disappears upon the mere cessation of work. The average statement by the patient is that mental effort of any kind brings it on, or, if it be already present, leads to exacerbations. It may in some cases be so severe as to be practically continuous. The practitioner should be cautioned not to confuse it with attacks of migraine, which so often complicate neurasthenia. Migraine is here, as elsewhere, to be differentiated by the fact that it occurs in paroxysms, that it affects chiefly one side of the head and face, that it is accompanied by more or less marked disturbances of the sympathetic nervous system, such as flushing or pallor of the affected side and contraction or dilatation of the pupil, and still further by the occurrence of various prodromal symptoms.

Backache is another of the more common sensory disturbances. It is most frequently referred to the small of the back, though occasionally to the midscapular region or to the sacrum. It also is pre-eminently a fatigue sensation. It is dull and diffuse in character and varies greatly in degree. Sometimes the patient finds relief by lying down and resting. At others the pain is severe and constant. Like neurasthenic headache, it is brought on or made worse by exertion. Further, we should be careful not to confound neurasthenic backache with the backache associated with diseases of the uterus and ovaries. The latter is to be differentiated by gynecological examination, and also by the difference in the character of the pain. This, it will be found, is described rather as the pain of constant soreness and of irritation, than the aching of fatigue. Frequently, however, a neurasthenic patient is at the same time the victim of pelvic disease, and in such instance a differentiation is neither possible nor necessary.

Aching in one or more limbs is occasionally present in nervous exhaustion, although this is far less common than headache or backache. When present, it is described as dull and diffused, and may affect one or more limbs. Thus, in a case for some time under the writer's care, there was decided aching in both legs. The patient, who was a collector, was in the habit of walking great distances daily. Another instance was that of a young woman who stood behind a counter many hours every day, and who, in addition to other neurasthenic symptoms, developed persistent aching in the left leg. This pain was so pronounced as to be for a time the most prominent feature of the case. It yielded, however, quite readily to rest and massage. Occasionally the aching is referred to a joint, or, more accurately speaking, to the neighborhood of a joint, and in such instance care should be taken that such a case be not mistaken for rheumatism, organic disease or hysteria. An example occurred in the person of a young man of nineteen, who presented himself with an aching pain in the right wrist. Examination failed to elicit increased pain on motion, nor were there other evidences of joint affection. Evidences of hysteria were also wanting, but examination did elicit various signs of neurasthenia. Finally it was learned that in his occupation, that of pocket-book maker, the right hand and wrist were used all day long in folding leather. It is very probable, therefore, that the pain was in reality a fatigue sensation. In addition to aching, or sometimes without it, there may be present in the limbs various anomalies of sensation, such as "throbbing" or "thrilling."

Occasionally the patient complains of tremulous feelings, altho
not really present. It is probable that these sensations are
fatigue phenomena, and that they depend either upon circulato
nervous disturbances. So regarded they merit the term adven
do the various sensory anomalies referred by patients to the he

Leaving now the vague sensations and the diffuse achings
sensory disturbances much more definite in character. First a
spinal tenderness. In many cases of neurasthenia, we find tha
flinches when we pass the finger, even though we do so lig
spinous process. The patient reacts as though the spine and the
were hyperæsthetic or tender in certain places. Patients someti
of this condition previous to examination, having made the
themselves. Most frequently, however, it is discovered by the p
it is revealed by a slight pressure it is probable that its seat
Indeed, some years ago, the writer demonstrated the fact that
neis may be made to disappear for a time by a local injectio
The spine, it should be remembered, is not tender along its e
but only in certain small areas or spots. These are found
certain situations, more especially over the seventh cervical
mid-dorsal region, the dorso-lumbar juncture, the mid-lumba
sacrum or the coccyx. Their area is generally very small.
such a spot can be covered by the tip of the thumb. Occasi
ever, the trouble is much more pronounced and serious. T
not only tenderness, but also spontaneous pain which seems to
deep-seated, as though it involved the vertebræ themselves, and
described as "a burning" or as an intense "soreness." It is
pressure and movement. Like simple backache, it may be so
be relieved by lying down, or it may be so severe as to constitu
cipal feature of the case and may dominate all of the other sym
this condition which has given rise to the term "spinal irritation.

Closely allied to spinal tenderness is cutaneous hyperæsthesia.
exist over the back, the sides of the trunk, the front of the chest,
trium, and the extremities. The scalp may become exquisitely s
the face, the teeth, the gums, the nipple, or the testicle may suffer
noteworthy that in some neurasthenics a trivial wound causes a
pain out of all proportion to the injury.

Anæsthesia is never present in simple neurasthenia. Patien
frequently complain of numbness of the extremities. This
readily provoked by slight pressure exerted either on the nerv
in their immediate neighborhood. Thus, if a neurasthenic remai
position for a short time, such as sitting with the knees crosse
upon a chair with the backs of the thighs resting upon the hard ed
develop numb feelings in the legs. These are comparable, no do
"asleep" or "pins and needle" feelings which prolonged pressur
on in perfectly healthy persons. These numb feelings occur in
asthenics even spontaneously or are attributed by them to appar
quate causes, such as the weight of the clothing or the constriction

The disorders of cutaneous sensibility present in addition vari
symptoms, adventitious in character, and comparable in every way t
alies of sensation already mentioned in discussing the general sens
ances. These patients often complain of formication or prickling
Sometimes these are described as "velvety." Occasionally the
as though a light touch were being passed over this or that por
body or as though water were flowing over the side of the face or
tion of a limb; sometimes, too, there is a subjective sense of heat

between the shoulder-blades, and at other times, though more rarely, there is a subjective sense of coldness of the surface. This may be localized and, rarely, even widely diffused. Sometimes it is combined with the sensation of flowing water just mentioned.

Visual Disturbances. Not only are the disturbances of the general and cutaneous sensibilities of great importance, but so are those of some of the special senses, notably of vision. For instance, it is very common for patients to complain that they are not able to read for more than a few minutes, and that if they persist for any length of time the letters become blurred and indistinct. Frequently they tell us that they cannot sew, cannot write, or, in fact, perform continuously any work which requires close or persistent use of the eyes. Such use, if attempted, is followed sooner or latter by headache, or, it may be, by vertigo, or by a sensation of painful tension in the eyes themselves. Hand-in-hand with this, vision becomes difficult. The first symptom, therefore, which the function of vision in neurasthenics presents is weakness. A moment's reflection will convince us that this weakness may be made up of different elements. First, the cerebral centres themselves may be so asthenic, may be so readily exhausted, as to unfit them for the reception of impressions for any but an exceedingly short period of time. This form of visual weakness is probably connected with the mental confusion, the headache, the vertigo, and other symptoms of intracranial distress that follow in certain cases attempts at reading.

Secondly, there may be a loss of working power in the retina, and also in the accommodative apparatus of the eye itself. As regards the retina, the weakness may make its appearance in two different ways; there may be, in one instance, a loss of power to appreciate continuously a small object held at convenient focal distance from the eye. This is observed every now and then in examining a neurasthenic patient with the perimeter. The point of fixation readily seen at first becomes, as the examination progresses, less and less distinct, until finally it disappears altogether. After a few minutes' rest it may return, but only to disappear again. This phenomenon is undoubtedly a fatigue symptom, and must be referred directly to the macula lutea. It must, of course, be distinguished from the similar fatigue symptom observed in healthy persons by remembering that in the latter it is noted only after a very prolonged effort. Retinal weakness may also show itself by a change in the visual field. The latter may be slightly diminished or contracted, as though the peripheral portions of the retina were less sensitive than they should be. It not infrequently happens in the beginning of an examination that the answers given by the patient indicate a normal field; indeed, this is true of the majority of neurasthenic cases. However, as the examination progresses, or if the tests be frequently repeated, the answers become less and less certain as regards the periphery, until finally the picture of a contracted visual field is produced. It thus appears that the contracted visual field of neurasthenic patients is in reality a fatigue symptom. This is rendered the more probable when the ordinary method of taking the field, that is, moving the test-object from the periphery to the centre, is reversed, and the test-object is moved from the centre to the periphery. The field obtained is apt to be much smaller than by the ordinary method, for by starting from the centre the retina is fatigued long before the peripheral portions are reached.

In some patients the eyes present in addition also slight hyperæsthesia. They seem to be somewhat sensitive to bright light. The hyperæsthesia is apparently retinal, and may be so marked as to lead the patient to remain indoors or to lead him to begin voluntarily the use of smoked glasses.

It appears from the above considerations that the sense of sight presents the same symptoms as are found elsewhere in neurasthenia, ness and irritability. In addition, there is present in a very of cases a lessening in the power of accommodation. This, al symptom, and, like its congeners, inconstant and variable. related, not only to the amount of work executed by the ey but also to the general condition of the patient. The muscle dation reflect more or less accurately the general tonus of the of the body, and especially is this the case in neurasthenics, i bility goes hand-in-hand with weakness. We can understand, independently of the condition of the eye the accommodati rise or fall according to the condition of the patient. Furt probable that the fatigue of the eyes, so easily brought on in is due especially to the weakness of the accommodative appara a slight extent to exhaustion of the retina. Further, inasmu vergence of the eyes, necessitated in accommodation to near ob taxes the internal recti, the latter reveal every now and the sufficiency. This, it need hardly be said, is also a fatigue sym

From what has been said we can readily understand that the frequently be unable to find defects in the eye itself or that he find in its apparatus of accommodation or in its extrinsic mu anomalies to account for the symptoms presented. Indeed, quently happens that the eye examination is practically nega the defects discovered are so slight as hardly to justify correctio

Besides the various eye symptoms already discussed, neura present others not so easy of interpretation. Patients will s that "everything appears misty," or as though objects were a veil; or they may even have attacks during which, for a vision is lost. Sometimes, too, they will say that everything perhaps, exceedingly bright; or that familiar neighborhoods lo familiar faces unnatural; that near objects look as though far they appear excessively small or excessively large. In part, anomalies of sensation are to be referred to the disturbed nu retina, but the greater number are doubtless due to disordered nerve centres. They are clearly adventitious in character.

The pupillary phenomena of neurasthenia are also interestin cant. As a general thing the pupils are rather large, sometin so. They may also react less promptly to light. Hippus, that contraction and dilatation of the pupil, is also frequently observ thenics, especially under excitement. It may occur independe stimulation. When marked it is almost always accompanied by of the knee-jerks. Inequality of the pupils is also observed in but it is always transient in character. Permanent inequality o of course, suggests organic disease. The observer should, how mind the existence of slight physiological inequalities. Fixed o pupils also suggest organic disease. If present the symptoms ataxia, or of grave cerebral disease, such as paretic dementia sought for.

Disorders of Hearing. Comparable to the various symptoms presented by the visual sense, we would expect, perhaps, to me or less disturbances of hearing. Deafness, properly speaking, doe and an examination of the ear is frequently negative, just as i sponding examination of the eye. However, a patient will freque cannot hear right. There is something the matter with my ears." of hyperæsthesia, less often present among the visual phenome frequently met with here. Patients suffer exquisitely from noise the latter are insignificant. On this account they will sometimes isolate themselves in secluded rooms of their homes, and even then may find no relief. Various paræsthesias, comparable to the adventitious symptoms observed in visual sense, are also observed here. They are, however, far more distressing. They consist of various forms of tinnitus, and are described by the patients as "roaring," "buzzing," "whistling," or "ringing," and at other times as "throbbing," "beating," "pulsating," or "ticking" sounds. They are brought on by fatigue, either mental or physical. These symptoms are often among the most distressing and persistent from which the patient suffers.

Disorders of Smell. Disorders of the sense of smell are very uncommon. However, impairment or loss of smell is sometimes noted in traumatic neurasthenia. Occasionally, also, we have symptoms of olfactory hyperæsthesia. In such instances the patient is excessively sensitive to odors. He will complain of the odors of flowers, of the odors of the kitchen, or of various perfumes, even when these are barely perceptible to others. It is not improbable, too, that patients sometimes experience olfactory paræsthesias, and are annoyed by disagreeable odors which apparently have no existence.

Disorders of Taste. Disorders of taste are somewhat more common than those of smell. Patients will sometimes say that they cannot taste anything; that everything "tastes like sawdust," or "tastes queer." More frequently they will complain of bitter, salty, sweet, or acid tastes. The latter are in part actual paræsthesias, and in part due to disordered secretions. Occasionally some one article, like bread, presents an altered taste; or, it may be, that an entire class of foods, *e. g.*, animal foods, acquire a peculiar and often offensive flavor.

PSYCHIC DISTURBANCES. Among the most interesting symptoms of neurasthenia are the psychic disturbances. Considerable confusion has existed with regard to them, but we will find that they are easy of comprehension, and that they arrange themselves into a natural order. At the outset we meet with a marked and characteristic symptom, namely, a diminution in the capacity for sustained intellectual effort. Just as the patient is incapable of long-continued physical labor, so is he incapable of long-continued mental labor. The attempt to do mental work sooner or later brings on symptoms of exhaustion. A certain task may be properly begun, but soon the patient experiences difficulty in keeping the attention fixed upon it. Gradually this difficulty increases until at last, instead of clear and distinct ideas, vague and confused impressions alone obtain. At the same time, painful sensations about the head arise, such as tightness, headache, giddiness, and even vertigo.

The difficulty of concentrating the attention may be so great as to lead to an habitual state of distraction and inattention. Hand-in-hand with this there arises a more or less marked dislike for intellectual labor. The patient finds that he must force himself to his work, and is often tempted to seek temporary, though treacherous, aid in stimulants. Naturally the patient becomes alarmed, and he complains to the physician that he is "losing his memory," or that he is "losing his mind." Indeed, the phrase "loss of memory" is one of the most common expressions used by neurasthenics. However, it is obviously incorrect. A patient will give a most circumstantial history of his case, not omitting unimportant details, and will in other ways show that there is actually no impairment of memory whatever. The difficulty really consists in a lack of the power of concentrating the attention. The patient does not remember, because he has really never comprehended, or, more properly speaking, has never apperceived that which he has read or heard.

Mental fatigue is also evidenced by a number of other symptoms. For instance, ideas do not seem to present themselves as readily, or in the same rapid succession as in health. There is, so to speak, a lack of thought, and this the patient himself well recognizes, often sayi think." There is also in these patients an undoubted less strength of the will. The patient does not feel the stimulus to is even convinced at times that effort is useless. He believ accomplish nothing; he has no courage, no confidence in himsel with this state is also a lack of decision. The patient is frequ lating that it is necessary for his friends to decide the most t for him.

Added to the other symptoms, we have more or less marke The most trifling causes often excite and anger the neurastl He is also apt to be morbidly sensitive, and often feels inte fancied neglect or oversight on the part of relatives or frienc riences changes in the emotions more readily than in health. inadequate causes may provoke marked depression, and at laughter. A play at the theatre or a newspaper account of a provoke him to tears. In other words, his emotional equilibri disturbed. This condition differs from the similar state observ in that the exciting causes for the emotional changes are al would produce the same emotion in health, though to a far less hysteria this rule does not apply.

The patient is also introspective. The various disturbances which he experiences more and more alarm him until, finally he on the alert for suspicious symptoms. He thinks because he h palpitation of the heart that he has incurable heart-disease, o feels the throbbing of a pulsating aorta that he has a tumor or an because his urine is cloudy, possibly from phosphates, that he h rhœa. The patient suffers from *nosophobia*. The associated m sion is sometimes so profound as to suggest true hypochondria. ever, important to remember that the mental depression of neuras from true hypochondria in that the patient can be more or less vinced by the physician that he is not the subject of any org that there is really no organic heart affection, no aneurism, no re rhœa. In other words, in simple neurasthenia the patient still h of appreciating evidence regarding his case when properly pre is not the victim of actual delusions. Unfortunately, however, met with who have so far transgressed the limits of mental hea belief in some special disease is definite and fixed, and who some about this belief a series of more or less systematized delusions. I sary to say that such cases suffer from actual insanity and not neurasthenia. Indeed, we not infrequently find in this class o more or less marked neuropathic family history.

In addition to the irritability and the mental depression exhibit patients, they sometimes show a distinct loss of the sense of the While morbidly sensitive themselves, they do not hesitate to di presence of non-medical persons their most intimate symptoms detail, and often using, in order to make themselves clear, exp are not only offensive to the ears, but often absolutely disgusting improbable that this condition is the result of the constant intros the constant dwelling upon nosophobic ideas.

Among other changes, too, we notice, now and then, a distinct the affection for near relatives, so much so that it attracts the the latter. Sometimes this condition is brought to the attention sician by the patient himself, who may say, "I no longer ca mother," or "I no longer care for my children as I should." A time, if the question be radically tested, the patient will usually react in a normal manner, though perhaps in a lessened degree.

Not only does the patient lack the capacity for sustained intellectual labor, not only is he irritable and depressed, but he frequently presents, in addition, the symptoms of an apparently causeless fear. That fear and weakness should go together is natural. Everywhere in nature they are associated. That a person whose nervous system is exhausted should also be morbidly afraid is, therefore, not surprising. We find, too, as we study our cases, that this emotion varies greatly in character and degree. It may be vague and ill defined, and may consist merely of a general feeling of anxiety. More frequently, however, it takes definite shape, or comes on acutely at certain times. Often it is associated with striking visceral symptoms. One of the most common forms is a fear which in some patients accompanies attacks of palpitation of the heart. The emotion is often so great as to give rise to the most marked outward signs. In some cases it is apparently the direct outcome of the heart symptoms, the patient believing that he has some serious organic disease and that death itself is threatening. Similarly a patient with sudden and strange sensations in the head may fear death by apoplexy, while another with sinking and other distressing sensations in the epigastrium, associated with an attack of acute indigestion, may feel as though he were about to die of utter weakness.

In other cases attacks of fear occur spontaneously and independently of visceral disturbances. In this instance the fear seems to be of purely nervous origin, and while somatic symptoms are frequently present they are altogether secondary. The patient has no idea of threatening death from heart, brain, or other disease, but simply experiences a sense of fear which is uncomplicated, and which may be slight or so intense as to be horrible and overwhelming. If the attack be severe the limbs become relaxed, the patient sinks into a chair, or may even fall to the ground. The face becomes pale, the body moist with a cold sweat, the pulse small and rapid, and the respiration hurried and irregular. As in intense fright from other causes, the sphincters may even become relaxed, and the bladder and the bowels spontaneously voided. Attacks of such severity are uncommon. More frequently they are of such a character that the patient is able to conceal them or to disguise their nature. It should be remarked in this connection that patients rarely mention voluntarily the various symptoms of fear which they experience.

In addition to the two forms of fear above mentioned, namely, the fear having its origin in visceral disturbances and spontaneous fear, we have attacks occur which are associated more or less closely with certain definite or fixed ideas. Thus, a neurasthenic patient may experience a morbid fear when left alone, or he may experience fear in the presence of strangers or of crowds; or he may be attacked by fear in the dark or in his own room or in the open street. These special fears find, as Bouveret truly claims, a fitting analogue in the fear which healthy persons experience when standing at a great height, even though they may be in a perfectly safe position. It is exceedingly probable that in neurasthenia the faculties are so weakened that ordinary surroundings no longer give a sense of security. Fear is therefore a natural consequence. It is doubtless in this way that spontaneous fear arises in the healthy person when the latter looks down from a great height. He misses the security of his ordinary surroundings. As might be expected, a large number of these special forms of fear have been described. Thus, the fear which comes on spontaneously in some neurasthenics when they find themselves in open places is called *agoraphobia.* The fear which comes on in narrow or closed places, *claustrophobia;* the fear of being alone, *monophobia;*

the fear of crowds, *anthropophobia;* the fear of darkness, *nyctophobia;* the fear of storms, *astrophobia;* the fear of special localities, *topophobia.* Certain neurasthenics, perhaps as a result of some paræsthesia of cutaneous sensibility, feel as though they were being contaminated by everything they touch. This fear has been termed *mysophobia,* or the fear of filth. Persons who have become neurasthenic by the shock and fright of railway accidents often suffer from attacks of fear on seeing or hearing a train of cars, and often find it, on this account, impossible to travel on railways. This form of fear has been called *siderodromophobia.* It can be readily seen that these names could be multiplied almost without end. For instance, Bouveret has called the fear which comes on in some neurasthenics on assuming the upright position *stasophobia;* and Beard has even framed such expressions as *phobophobia,* fear of fears, and *pantophobia,* fear of everything. Unfortunately these names, while they define certain morbid fears, often give rise to the impression that all of these various forms are intrinsically different, and also that they are actual psychoses. Indeed, they are often so considered, the error arising from the fact that similar morbid fears also occur in the insane.

Among the most important symptoms still to be considered is insomnia. It is sometimes one of the earliest of the symptoms presented. It varies greatly in degree. A patient may, for instance, find it difficult to fall asleep, or the sleep may be so light that the slightest sound awakens him, and the night may be spent in alternately falling asleep and waking. More commonly, however, the patient falls asleep readily, but finds that he awakes at an earlier hour than usual, and further that his sleep has not been refreshing. If the insomnia becomes more pronounced, the hours of sleep become progressively shorter, the patient awaking earlier and earlier until at last a few hours or a few minutes may constitute the entire sleep; or he may even pass nights and days in succession without any sleep whatever. As a rule, such sleep as is obtained is light; occasionally it is heavy and profound. In every instance, however, the patient feels unrefreshed on wakening; indeed, the various fatigue sensations are more pronounced than ever. He will frequently say to the doctor that he feels completely exhausted on awakening; that he feels as though he had worked hard all night. After taking breakfast the feeling of fatigue is somewhat relieved, and, as a rule, it becomes less marked as the morning advances. Not only are neurasthenic patients troubled with insomnia, but they frequently dream a great deal. They seem to rehearse in their sleep the various experiences of the day. More often the dreams are of an unpleasant character, the patient dreaming of murders, of horrible occurrences and of terrible accidents, and while so doing may wake up suddenly in a paroxysm of fright. Startling dreams, that is, dreams attended with fright, are very common in neurasthenia of traumatic origin. Regarding the amount of sleep and also its character, the physician should be cautioned about always accepting the statements of the patients. One of the most common errors made by neurasthenics is in relation to the amount of sleep actually obtained.

We have in neurasthenics now and then peculiarities of speech, and even of handwriting, to which it is well to allude for a moment. Patients sometimes speak slowly and enunciate their words in a slurred and slovenly manner. There is nothing, however, in their manner of speaking which is in any way comparable to the halting or scanning speech found among the insane. When asked to speak clearly they always prove their ability to do so. We are frequently impressed with the fact that they talk as though they were tired, and that for this reason they do not articulate properly. Even the voice seems to have a tired ring. Patients are also seen who speak too rapidly and run their words, syllables, and even their phrases together.

As regards the handwriting, peculiarities are noticed not so much in the writing demanded by the daily occupation, as in letters to relatives and friends. In the latter it is apt to be hasty, jerky, and irregular. Occasionally both words and letters are incompletely formed, and here and there letters, syllables, and even whole words may be omitted. A letter so written is like the speech, merely slurred and slovenly, and is of itself no evidence of insanity, because the patient himself is fully conscious of the errors that he makes, and because other letters demanding care and accuracy, *e. g.*, business letters, are written properly.

GENERAL SOMATIC DISTURBANCES. These consist of disturbances of digestion, of circulation, of secretion, and of the sexual functions.

Digestive Disturbances. The digestive disturbances of neurasthenia are of peculiar importance. They are among the most common of the symptoms presented, among the most distressing to the patient, and among the most difficult for the physician to treat. They are sometimes, though infrequently, but slightly pronounced; at other times so severe as to be suggestive of organic disease. As elsewhere the first symptom that we meet is weakness. The patient having taken a moderate quantity of food feels at first no special distress; but after the lapse of a greater or smaller interval of time, sensations of weight, of oppression, and of general discomfort about the epigastrium make their appearance. Sometimes they are accompanied by a sensation of distention, and an examination will often disclose an excess of gas in the stomach. Frequently the condition is attended by eructations. If the case be more pronounced, in addition to the mere sense of weight and oppression, pain is referred to the epigastrium, and also, at times, to the back between the shoulder-blades. If we examine the epigastrium we find that it is slightly sensitive to pressure. It is not, however, painful as in inflammation, ulcer, or other organic trouble; nor, do we find that the stomach is dilated, but simply distended. The tongue is clean, or at most but slightly coated. If the gastric juice be examined, analysis may reveal no marked change in the amount of pepsin or hydrochloric acid present. Frequently, however, a more or less decided diminution in the amount of free acid is noted, and at times, indeed, the latter may be entirely absent. Thus far the trouble is still purely functional. Digestion is much delayed, and often the stomach is not emptied before it is time to take the next meal. Nausea may be present, but vomiting is quite rare. This atonic indigestion is not always limited to the stomach, but may involve the intestine. In such cases there is more or less meteorism. Constipation is the rule.

Patients suffering from even this mild form of gastro-intestinal neurasthenia, or nervous dyspepsia, as it is more commonly termed, are subject to attacks of palpitation of the heart, the attacks coming on when the indigestion is at its height. If the trouble continue for some time, mental depression with nosophobia sooner or later appears. Occasionally during an attack of indigestion the patient feels heavy and sleepy and entirely incapacitated for intellectual or physical effort. Frequently the indigestion is accompanied by giddiness. The appetite is apt to be capricious. If there be marked mental depression it is diminished, but every now and then it is increased. Further, the patient, after taking food, feels unsatisfied, or, after the lapse of half an hour or an hour, feels as though he had not had his regular meal. As a rule, neurasthenic patients consume but little fluid. Infrequently, however, thirst is increased.

In neurasthenia of long standing digestive disturbances are far more pronounced. Shortly after ingesting food, the patient experiences a sense of weight and discomfort, together with marked distention and oppression in the epigastrium. Frequently it happens that eructation is grossly insufficient or does not occur at all, or only after very prolonged and distressing intervals.

At times it would seem as though there were a veritable spasm
opening which prevented the escape of the gas. When maki
examination of such a case we sometimes find that distention i
to approach actual dilatation. During eructations portions of
the stomach may be carried into the œsophagus, and thus gi
familiar symptoms of heartburn or pyrosis. While in a lar
cases the indigestion is marked by a greatly diminished qu
absence of hydrochloric acid, there are instances in which thi
be in excess, and others, again, in which butyric, lactic, and
present as the result of fermentation. The tongue in cases of this
and there is more or less tenderness of the epigastrium. We no
dences of an actual gastric catarrh. In other words, the aton
noted in simple cases of neurasthenia becomes gradually more an
vated, and finally leads to more or less marked structural chang
tinal distention and meteorism present are also apt to be ac
transient pains, and, in addition, the abdomen may also be deci
to pressure. At times patients suffer from attacks of pain in cer
the ileo-cæcal region, and also at the junction of the transverse
ing colon. These attacks are evidently due to collections of ga
to be associated with paralytic distention in one part of the b
with spasm in another. Hand-in-hand with this, constipation
very marked, the contents being often completely dry and expel
great difficulty. At other times, instead of constipation, there
diarrhœa of relaxation. Frequently, too, in these patients, evac
bowels is attended by a sense of nervous exhaustion, the patient
completely prostrated and compelled to lie down after each mo
casionally mucous colitis, in which the patient passes more or l
masses of fibrinous or mucous exudation from the bowel, is pres
contents of the bowel are voided covered with this material, or i
this substance is passed in large quantities during attacks simulati

Circulatory Disturbances. The disturbances of the circulato
consist in modifications of the force and rhythm of the heart's a
character and frequency of the pulse, and in more or less m
tions of vasomotor tonus. Perhaps the most striking, if not ind
common, symptom of circulatory disturbance in neurasthenia, i
tion of the heart, already mentioned. As pointed out, this sym
often associated with digestive disturbances, though it is not n
pendent upon the latter. It commonly occurs at that period of di
the process seems to have been slowed or arrested and when ma
distention of the stomach has taken place. It may also be as
marked gastralgic pains. During an attack the patient is ofte
tressed, and frequently suffers from fear. The heart beats vi
with increased rapidity against the chest-walls, while the arteri
they can be seen, throb excessively. Sometimes the face is pal
quently it is flushed. The patient complains of oppression o
After a longer or shorter interval the attack passes over. Th
of the heart, which may have increased to 120 or 130 a min
fall to near the normal. Such an attack is the most common of
cardiac disturbances noted in neurasthenia. However, every no
the patient has a seizure which resembles an attack of true angi
Charcot, who has minutely described this condition, separates su
into three periods. The first period is purely prodromal, and m
eral hours, or even an entire day, or may recur several times
actual onset of the crisis. During its continuance the patient
sensations of pain about the heart, a feeling of oppression, of fu

left half of the body, sensations of choking, and difficulty of swallowing. In addition, he has a cough, is irritable, has insomnia, and loss of appetite. Suddenly, when the second period has arrived, the patient feels an intense griping in the præcordial region, accompanied by extreme nausea and indescribable terror. The præcordial pain radiates through the chest to the left brachial plexus and down the left arm. Sometimes it is felt even in the left leg. At the time that the paroxysm occurs the face is pale, cold, and livid. The respiration becomes slow and seems to stop during inspiration, which is gasping. The pulse and the heart-beats can scarcely be felt. The pupils become contracted, equally so, on both sides. The agitation of the patient is extreme. He grasps in all directions for support. He feels as though he were about to faint or about to die. The second period lasts from three to six minutes. The most important points in the clinical picture are the paleness of the face, the smallness of the pulse, and the feebleness of the heart-beats. There is apparently a universal peripheral vascular spasm, involving probably also the arteries of the heart itself. The third period makes its appearance by a change in the color of the face, which becomes red and hot. A similar change is also observed in the left half of the body. The pulsations of the heart are now very appreciable, and become more and more energetic. The pulse-rate is from 130 to 140 a minute, with intermissions every ten or fifteen beats. The respiration is very frequent, but always panting, with a tendency to arrest during inspiration. This period lasts from ten to fifteen minutes. It is characterized by an intense vasomotor reaction. The arterial spasm of the preceding period is succeeded by dilatation of the peripheral vessels. In the last and final period the pulse-rate falls. At the same time the oppression diminishes. The patient, however, trembles all over; his face, his lips, his limbs, and even his teeth may chatter. This trembling may last from half an hour to an hour. Fortunately, attacks of such severity are not frequent, but it is well to be aware that such attacks do occur in neurasthenia, and that, though very distressing, they are far less serious than true angina.

The pulse-rate, which is temporarily increased in attacks of cardiac palpitation and also in attacks of pseudo-angina, is every now and then more or less *permanently* increased. In other words, there is present in some cases of neurasthenia a more or less permanent form of tachycardia. (This tachycardia is unassociated with any symptoms suggestive of Graves' disease. There are no prominent eyeballs and no increase in the volume of the thyroid gland.) It would appear that through frequently recurring attacks of palpitation the pulse-rate is more or less permanently raised, and that it often remains for long periods of time at 100 or 120. During a period of unusual quiet the rate may fall, say, to 95 or 90 in a minute. However, when there is a variation, it is more frequently that which is produced by fatigue and excitement, and the pulse-rate shows a still further exacerbation. It is not uncommon for 150 to 160 beats to be counted in the minute in such an attack. Tachycardia is sometimes associated with a history of shock and fright.

Bouveret describes a malignant form of tachycardia, but it is doubtful whether this can properly be claimed as a symptom or even an outcome of neurasthenia.

In a few cases, a heart murmur, evanescent in character, has been noted. It is mentioned by Bouveret, Richter, and others. Of late it has been again studied by John K. Mitchell.[1] It may be rendered audible by requesting the patient to make a change in posture, such, for instance, as sitting up in bed

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[1] Mitchell, John K.: Transactions College of Physicians of Philadelphia, 1892. xiv 132.

after having lain still on the back for some time. In additio
accentuation of the first and second sounds, a soft but di
murmur is heard near the apex, systolic in time. It disapp
seconds, but may be again provoked by additional change
other slight physical exertion.

More interesting, however, than either palpitation, spurious
tachycardia thus far considered, are the symptoms of loss of gen
tonus. These are present in the larger number of cases.
neurasthenic patients often suffer from involuntary flushing o
of other portions of the body. Not only is this loss of vasomo
able to the smaller vessels, but it is even manifested in the l
One of the most common symptoms, indeed, observed in n
aortic pulsation. The patient feels a deep-seated throbbing i
trium, and this is so marked that it can readily be verified b
the physician. It is a most distressing symptom, for the p
to believe that he has some serious organic disease. Less often
sensation, instead of being felt in the aorta, is felt in the limbs.
it is excessively persistent and distressing.

In order to convey to the reader a proper idea of the con
vasomotor apparatus, it is well to relate the experiments of Mos
Mosso, it will be remembered, demonstrated by means of a plet
that in a healthy man whose arm is fixed in the apparatus, ar
himself up to some intellectual effort, the arm shows a distinct d
size. In other words, a reaction takes place through the vasom
tus, by means of which the vessels of the limb become slightl
while at the same time those of the brain become dilated. A
with this result, demonstrated that in neurasthenic subjects a sim
takes place, but that it is *evanescent*. There is at first a sudden
reaction, but it almost immediately disappears; this may be foll
other reaction and again by a sudden fall. Thus we have th
and the weakness of the vasomotor apparatus absolutely demons

Feebleness of the circulation is further shown by coldness
lividity of the extremities.

Disturbances of the Secretions. In a large number of neurastl
is, as Beard long ago pointed out, an insufficient ingestion of li
patient really suffers from deficient thirst. In keeping with thi
is a diminution, not only in the secretions of the stomach and in
also a deficient secretion of saliva and a consequent abnorma
the mouth. In addition there is unusual dryness of the skin
and there can be no doubt that in some instances premature lo
directly traceable to this cause. Not only is the secretion of
diminished, but there is also a diminution in the quantity of u
case of neurasthenia of traumatic origin recently under the
writer the amount of urine voided in the twenty-four hours wa
over four ounces. Hand-in-hand with this, the patient ingeste
fluid. In keeping with this general diminution of secretion we
have unusual dryness of the joints, so that when the fingers o
forcibly moved crackling sounds are produced. Occasionally t
are noticed in the back of the neck on suddenly rotating the head
such instances to be referred to the joints of the articular processes

As opposed to the condition of diminished perspiration, we som
more or less marked hyperidrosis. This hyperidrosis may be
local. Frequently it is transitory in character, the patient swe

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[1] Angel: Archiv f. Psych., 1884, xv. 618.

upon slight emotional or intellectual excitement or upon slight physical exertion. Thus many neurasthenics on attempting to read or to write find that their head and neck become moist with perspiration. In sudden and transient sweatings the perspiration is distinctly thin and watery. Very often, instead of being transient, it is continuous, and is especially noticed in the hands. In some cases, this sweating of the hands is so marked that the patients will wet various objects which they touch, as in a case cited by Beard, where a bookkeeper was forced to put a blotting-pad under his hand before attempting to write, in order to avoid soiling his book. The moist hand of the neurasthenic is nearly always a cold hand. Again, the perspiration in the continuous or constant form, instead of being watery, as in the transient form, is decidedly sticky and often unpleasant in odor. This is more especially the case when the sweating occurs in the axilla and the groin. Occasionally sweating occurs only when the patient lies down, and when the muscles and the vasomotor apparatus are relaxed. Sometimes it is so marked at night as to cause veritable night-sweats.

Not only is the perspiration affected, but the oil glands also suffer, and as a consequence acne is a very common occurrence.

Instead of a simple diminution in the quantity of urine voided, the latter may be decidedly increased. In such cases the symptom is of necessity concomitant with unusual thirst. More often there is an apparent increase only. Thus a patient under a slight emotional excitement passes an unusual quantity of urine, and is obliged to empty the bladder at comparatively short intervals. It will be found that when the period of excitement has passed away a long interval occurs, during which a small quantity of urine only is voided. We must, therefore, distinguish between a temporary or spurious polyuria and a true polyuria. Temporary polyuria occurs especially after attacks of fear with cardiac palpitation. It may also occur after physical exertion. Cases in which there is marked increase in the frequency of micturition are frequently traumatic in origin. This sympton may be so marked as to suggest, at first sight, cystitis or other bladder trouble. Pain is, however, rarely complained of. When questioned, the patient will simply say that when the desire is felt he must go at once lest he soil his clothing.

As opposed to this, patients will sometimes complain that they cannot pass their water readily or freely. Examination fails absolutely to disclose any local trouble. The condition is evidently psychic and similar to that which occasionaly occurs in normal individuals who cannot void the urine in the presence of strangers.

While the urine is sometimes actually increased in quantity this symptom is rarely so striking as to suggest a genuine diabetes insipidus. Even if this should be the case, it must be remembered that the symptom disappears when the treatment is directed to the neurasthenia itself. It should be remarked, in this connection, that transient glycosuria has been noted in neurasthenia. This symptom, however, should always be regarded with suspicion, and other signs pointing to the existence of a true diabetes mellitus should be carefully sought for. Similarly albumin has every now and then been detected in the urine, and the remarks just made in regard to glycosuria apply equally to it. In this connection it is important to remember the cases of albuminuria in lithemic states recently so well described by Da Costa.[1]

The most common condition of the urine observed in neurasthenia is an excess of uric acid and urates. It is very common for the patient to pass urine which almost immediately afterward becomes cloudy, and which on being allowed to stand shows an excessive deposit. In many neurasthenics

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[1] Da Costa, J. M.: American Journal of the Medical Sciences, January, 1893, p. 1.

this condition is a transitory one. It should also be distinguis
which occurs in normal individuals who are exposed to grea
whom, as a consequence of sweating, the urine is naturally con

It occasionally happens that, instead of the above conditi
voids a urine which is cloudy while it is being passed, but beco
cooling. Such a urine becomes cloudy again on being heated
tion is that known under the name of phosphaturia. It is no
and then in persons who are otherwise healthy, but its freque
in neurasthenic subjects, and the facts that it is sometimes c
irritation of the bladder or of the urethra, and that it is so often
the patient's mind with sexual disorder, make it of importanc
patient, already predisposed to nosophobic ideas, imagines that
from spermatorrhœa.

It is probable that in a large number of cases the excess of
simply apparent, the condition really depending upon a dimin
of the urine. In some instances, however, the phosphates are s
excess by actual analysis. Similarly oxalic acid in the form
lime is occasionally present in large quantity. This is especially
case when dyspeptic symptoms are marked. Oxaluria, probabl
the sharp-pointed octohedral crystals so often present, is occasio
panied by symptoms of bladder and urethral irritation.

Sexual Disturbances. Symptoms referable to the sexual ap
in a large number, if not in the majority, of neurasthenics.
both sexes. Occasionally they are so marked as to dominate the
picture, so much so that many writers term the condition "sex
thenia. It is commonly supposed that masturbation and sexual
necessary factors in their production, but this is an error. They
least in their less marked forms, in neurasthenia pure and simple.
we have already seen, masturbation and sexual excess are every
the principal causes of a nervous exhaustion, and we should not
to find in such a case sexual symptoms most prominent.

In men these symptoms are commonly as follows: If the
unmarried, he complains of frequent nocturnal seminal emissio
numerous nosophobic ideas upon this symptom. He attribute
physical depression to this cause, and is frequently very despond
patient be married, he complains that the ejaculation occurs prem
also that it is not accompanied by the usual pleasurable sensations.
if the condition becomes more marked, he complains that the
longer be satisfactorily accomplished, because erection is incon
further, that the genitals seem more or less anæsthetic. This con
deepen into one in which no erection whatever can occur, and in
or less marked diminution of sensation is demonstrated by examina
in the history of his case the patient is apt to imagine that he
from spermatorrhœa, especially if there be present phosphaturia,
the victim of a gleet or other chronic urethral irritation. How
these circumstances, the microscope fails to demonstrate spermat
later on, when the loss of sexual power has become actual and m
matozoids may be found. We should remember in this connectio
many neurasthenics indulge in nosophobic ideas with regard to t
apparatus when no sexual disturbances whatever exist. Many a
in whom seminal emissions occur with but normal frequency belie
is losing, or has lost, his sexual power. This, as has just been poi
very common in those who suffer from phosphaturia.

Associated with the weakness and irritability of the sexual ap
have also various paræsthesias. These consist of pricking, creepin

bing sensations referred to the urethra and penis or to the testicles. They are at times extremely distressing and are most difficult to relieve. Occasionally there is excessive hyperæsthesia of the glans and testes.

In women sexual disturbances similar to those observed in men also occur. Not infrequently it happens that a patient complains of orgasms occurring during sleep, and further states that these are accompanied by voluptuous or unpleasant dreams, and that she feels very much prostrated by them. If she be married she is less likely to complain of nocturnal orgasms, but rather of some abnormality of the sexual act; the orgasm may be delayed, deficient, or absent. The genitals are in these cases in a condition in which they respond less readily. Contact or friction fails to produce pleasurable sensations. Earlier in the history, on the other hand, we occasionally have marked hyperæsthesia. Various distressing paræsthesias resembling those occurring in the male may also be present. It need hardly be stated that female patients are less ready in their admissions in regard to sexual symptoms than men. Occasionally, however, they evince a morbid desire to dwell upon them, the mental condition being similar to that existing in male patients.

Very frequently neurasthenic women complain of pelvic pain, and this upon investigation proves to be ovarian. Associated with this condition there is often marked ovarian tenderness. Here, however, we must be upon our guard not to confound actual organic disease with a symptom purely indicative of hyperæsthesia or irritability. In this connection, too, we should remember that large numbers of neurasthenic women suffer from actual pelvic disease. In these cases the neurasthenia is almost always the outcome of the local affection, and must therefore be regarded as a neurasthenia symptomatica.

Symptomatology in General. In the clinical picture presented by neurasthenia the most varied symptoms are aggregated. Because of this apparent complexity, it has sometimes been maintained that in neurasthenia we have not a single affection to deal with, but really a great variety of diseases. However, it is evident that in their essentials all of the symptoms are the same. They are simply various ways of expressing the two cardinal facts of the weakness and irritability which affect the nervous system as a whole. It must not be expected that every case should present all of the sensory and motor disturbances, or all of the other anomalies of function above described. While it is true that there are some cases in which fatigue symptoms appear to be distributed evenly over the entire nervous system, there are others in which this or that particular group of symptoms is accentuated. For instance, the form in which the symptoms are widely diffused, that is, are both cerebral and spinal, has been called cerebro-spinal neurasthenia. Again, the form in which cerebral symptoms predominate has been termed encephelasthenia; the form in which cord symptoms predominate (*i. e.*, spinal irritation, great weakness of the legs, etc.) has been called myelasthenia. Inasmuch as neurasthenia is an affection of the nervous system as a whole, the predominance of this or that group of symptoms cannot be considered as sufficient ground for its division into separate forms. In its essentials the affection is always the same.

Pathology. Numerous writers have indulged in more or less vague speculations regarding the pathology of neurasthenia. These it would be useless to consider. Let it simply suffice to say that neurasthenia does not stand in any relation with anæmia, chlorosis, or other affection of the blood. Neither does it bear any direct relation to the condition of the other tissues. That it has a pathology of its own involving the nervous system directly is rendered more than probable by the remarkable researches of C. F. Hodge,[1] and the equally remarkable discoveries of A. Mosso. The former investigator studied the nerve cells of various animals both before and after fatigue. In some instances he exhausted the cells by prolonged electrical stimulation; in others his results were based upon fatigue induced by the normal exercise of function. Among the animals studied by him were the frog, the cat, the dog, the English sparrow, the pigeon, the swallow, and the honey-bee. The spinal ganglion cells of the cat, for instance, were studied in such a way that a ganglion which had been subjected to prolonged electrical stimulation was compared with the normal ganglion on the corresponding nerve root of the opposite side. Again, the spinal ganglia and brain cells of birds and of bees

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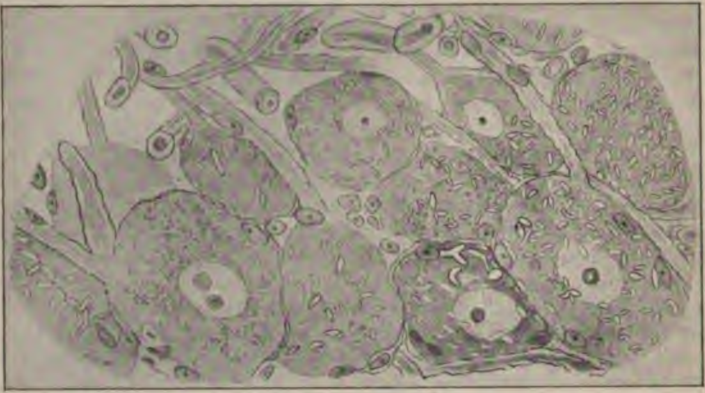
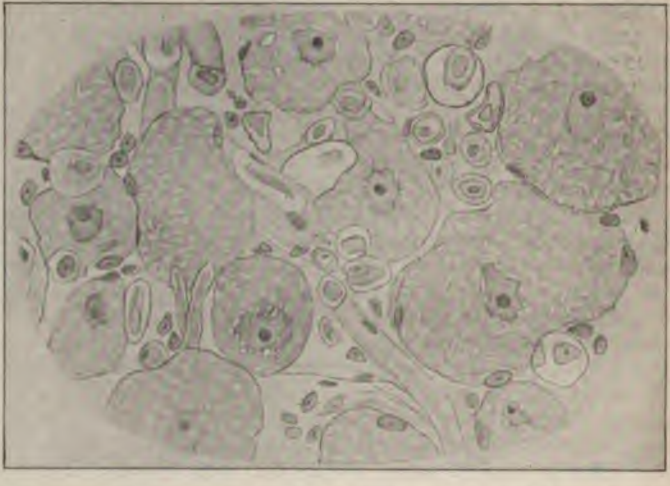
[1] Hodge, C. F.: A Microscopical Study of Changes Due to Functional Activity in Nerve-cells. Journal of Morphology, 1892, vol. vii. p. 95.

FIG. 22.



Portion of field from the third brachial ganglion of an English sparrow killed at 7 A. M. (HODGE.)

FIG. 23.



Field from the corresponding ganglion of an English sparrow killed on the same day (as in Fig. 22) at 7.30 P. M. (HODGE.)

were studied by securing various individuals before and after prolonged flights, the examinations being in each instance conducted under precisely the same conditions. Thus, for instance, the nerve cells of bees that had worked all day were compared with those of the same hive that had been at rest during the same period. In all instances, irrespective of the animal selected, changes were discovered in the nerve cells; further, these changes were always the same. They involved the nucleus, the cell protoplasm, and even the cell capsule when present. They are summarized by Hodge as follows:

A. For nucleus: 1. Marked decrease in size. 2. Change from smooth and rounded to a jagged, irregular outline. 3. Loss of open reticulate appearance, with darker stain.

B. For cell-protoplasm: 1. Slight shrinkage in size, with vacuolation for spinal glanglia; considerable shrinkage, with enlargement of pericellular lymph space for cells of cerebrum and cerebellum. 2. Lessened power to stain or to reduce osmic acid.

C. For cell capsule, when present: Decrease in size of nuclei.

D. Individual nerve cells, after electrical stimulation, recover, if allowed to rest for a sufficient time. The process of recovery is slow; from five hours' stimulation, being scarcely complete after twenty-four hours' rest.

FIG. 24.

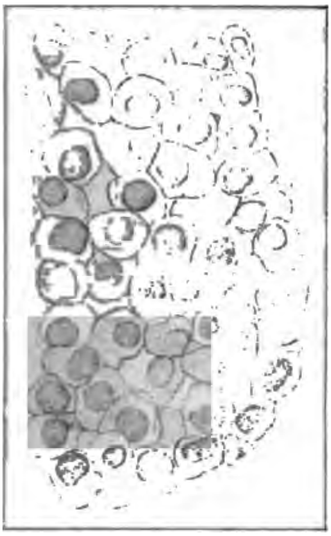
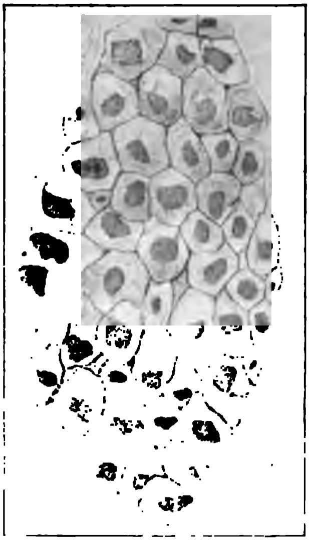


FIG. 25.



Median subdivision of the antennary lobe of the brain of the honey bee. Fig. 24, morning, 6 o'clock Fig. 25, evening, 7.30 o'clock. Both of same day. (HODGE.)

Certainly these experimental results are more than suggestive, and are of the utmost value. Clearly, we have here facts upon which a rational pathology of neurasthenia can be based. When we consider that the primary symptom of neurasthenia is loss of power, and when we learn that the lesion of fatigue is loss of cell substance, it remains but to add one fact to the other. When we learn further that the process of recovery of the exhausted cell is slow we can readily frame a hypothesis to account for the persistence of the average neurasthenic symptom. We must also reflect that in the neurasthenic patient fatigue has exceeded the normal limits, and further that the proper opportunity for repair to take place does not present itself.

Before, however, a complete conception of the pathology of
can be formed another factor must be considered. It is exceedin
where there is such an excessive waste of nerve substance as we
to infer is the case in neurasthenia, that various substances, the
metamorphosis, are circulating in the blood—substances some o
be normal to the blood but now present in excess, and others perl
new and foreign. At any rate, some of the phenomena pres
vanced and chronic cases of neurasthenia suggest the presenc
blood of substances having a toxic action. Mosso,[1] on observing
presented in the evening, after a day's march, fatigue in the ar
well as in the leg muscles, conceived the idea that fatigue altere
tution of the blood, and he afterward discovered that the blood c
animal when injected into an animal at rest, produced in the latte
teristic symptoms of fatigue. That in purely nervous exhaus
modifications of the blood ensue seems a reasonable conclusion.
our knowledge of the substances produced in the blood under t
stances is exceedingly limited, such evidence as we possess prov
have a decidedly toxic action. It is well known to physiologists t
muscle, which has been completely exhausted by electrical stimul
fuses longer to respond, is washed out by injecting into its artery
solution, the exhaustion disappears and the muscle again reacts
trical current almost as well as before. Evidently the muscle e
fatigue suffers not only from loss of substance, but also from the
various toxic agents which strongly inhibit its physiological acti
equal experimental proof is not at hand as regards the nerve c
can be no doubt that the physiological problem presented is the
chemical nature of the various fatigue substances is still a matter
inquiry, but whatever their nature they are the direct result of tis
ism, and in some aspects this fact is exceedingly interesting and
For instance, we are all familiar with the relation existing betwe
neurasthenia and with the fact that the urine of neurasthenics ver
tains uric acid in great excess. These facts must have a special s
The suggestion acquires additional force from what we know at
day of the relation of uric acid to the nucleins. Uric acid is not,
so long taught, a substance standing in a complementary relation t
have reason to believe from the researches of Horbaczewsky that i
the chemical disintegration of the nuclein constituent of cells, an
true its presence in excess in neurasthenics and the relation of gout
thenia acquires a new significance.

In addition to the above considerations, other factors secondary
ter, but of great practical importance, present themselves. It is w
that prolonged and persistent derangement of function may be f
actual tissue changes. Thus a heart which is constantly overactin
subject to frequently repeated and violent attacks of palpitation
dergo hypertrophy; or the walls of the bloodvessels may become
and, if the case persist sufficiently long, may even undergo at
change. That actual organic changes may supervene in the dig
is more than probable. That such changes also occur in other struc
as the muscles and even the bones, there can be very little doubt.
curring in the last mentioned structures they are like the chan
bloodvessels, suggestive of senescence, and are best seen in cases of
persons who have overtrained physically and in whom neurasthe

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[1] Mosso, A.: Sulle leggi della fatica. Rendiconti della R. Accademia dei Lincei, 188
Ermuedung (German translation). Leipzig, 1892.

pressive of a breakdown, the direct result of physical excesses. These secondary changes observed in neurasthenic subjects are of course terminal in character, and their recognition is of the utmost importance with the view of prognosis. When they are present the symptoms are necessarily persistent and little if at all influenced by treatment. The picture presented is that of a profound and practically hopeless nervous exhaustion to which the name *terminal neurasthenia* seems to be especially applicable. Further, the presence of toxic substances in the blood as an outcome of simple though long-continued and profound neurasthenia furnishes an added explanation for those cases which eventuate in terminal forms, and renders more readily comprehensible the occurrence of such changes as general arterio-sclerosis and sclerotic changes in the kidneys and other viscera.

Diagnosis. The simpler and milder forms of neurasthenia are readily recognized. The same symptoms are so frequently grouped together that the recognition of the affection is comparatively easy. However, the physician must be cautioned to eliminate absolutely all evidences of organic or special functional diseases. This is especially true of such affections as are accompanied by general loss of strength, as beginning phthisis, the various diseases of the blood, and some of the more obscure affections presently to be mentioned. At times we have associated with neurasthenia hysteria, and it therefore becomes important in many cases to decide whether our case is one of simple neurasthenia or pure hysteria, or whether we have neurasthenia present with hysterical complications. The differences between simple neurasthenia and hysteria are well marked. It will be remembered, for instance, that in the former affection we have no true paralysis. On the other hand, paralysis in various forms occurs in hysteria. Again we have seen that true anæsthesia never occurs in neurasthenia. It often occurs in hysteria; for example, in the form of hemianæsthesia. Convulsions, too, are not symptoms of neurasthenia; on the other hand, they are common symptoms of hysteria. Contractures of the muscles never occur in neurasthenia; they frequently occur in hysteria. Further in its course and aspect simple neurasthenia differs markedly from hysteria. Its beginning is very gradual, and the symptoms become steadily more and more marked with time. The onset of hysteria, however, may be sudden, and the symptoms presented often vary greatly from day to day. Again, such hysterical stigmata as the globus hystericus, the form of head pain known as clavus, and the reversal of the color-fields, are never present in neurasthenia. Other points of minor importance might also be cited, but enough has been given to indicate the line of thought that is to be followed in differentiating between the two affections. We must not forget, however, as already hinted, hysteria is every now and then engrafted upon neurasthenia. Indeed it may be said that under certain circumstances neurasthenia will predispose the patient also to hysteria.

We have already pointed out, in discussing the symptomatology, the differences existing between the pseudo-hypochondria of neurasthenia and true hypochondria. In the former condition it will be remembered the patient, who for the time being believed that he had serious disease of the heart on account of frequently recurring attacks of palpitation, readily permits himself to be set at right. The ideas, however, formed in true hypochondriasis are fixed and amount to actual delusions. We have a true psychosis to deal with. It will hardly be necessary in this chapter to point out the differences between the depressed mental state of neurasthenia and melancholia. Suffice it to say, that the evident psychic pain present in the latter affection, together with the self-accusatory ideas or the delusion of the unpardonable sin, at once settle the question. The following, however, should be borne in mind; that is, that every now and then the patient in whom the diagnosis of neurasthenia has been made, may many months or years afterward suffer from true hypochondriasis or true melancholia. The question naturally arises whether in these cases a natural transition into these psychoses has taken place. It can be safely said that this transition, if it occur at all, is exceedingly rare. Almost always, if such transition has taken place, we will find that the patient presents a history pointing clearly to a neuropathic ancestry, or we have symptoms presented by the patient himself which are incontrovertible evidences of a neuropathic constitution.

Occasionally there is danger of confounding some of the psychoses, when in their earlier stages, with neurasthenia. At times, indeed, it is a matter of difficulty to distinguish between a profound encephalasthenia and a *beginning* paretic dementia. In neurasthenia, it will be remembered, we may have tremor, alterations of speech, of handwriting, and there may even be transitory inequality of the pupils. As regards the speech and handwriting, we should remember that it is always possible for the neurasthenic to speak and write correctly and legibly when his attention is directed to it; and, further, that he is conscious of his errors as he makes them. There is, also, this important point of distinction. In neurasthenia the change in the mental faculties is one of *quantity* and not of *quality*. We must remember that the cardinal feature of cerebral neurasthenia is the inability for sustained intellectual effort. The patient is capable of appreciating and correctly reasoning about various matters that are brought to his attention, but soon becomes exhausted. In paresis, however, there is a distinct blunting of the mental faculties. We soon realize, on studying such a case, that the intellectual as well as the emotional faculties are distinctly obtunded. It is only in the earlier stages, when the change is still very slight, that difficulty may arise, but it is just under these circumstances that errors have occasionally been made by reputable physicians. See also Chapter XXIII.

Prognosis. Neurasthenia is essentially chronic in its course. It does not immediately threaten life. In the simple and uncomplicated form the prognosis is very bright, and this is the case no matter how profound the affection may be. It is truly marvellous how much can be accomplished. As we will presently see, patients presenting the symptoms in the most pronounced degree, who have suffered great loss of weight, and who, in outward appearance, look as though their hold upon life was extremely small, often make, under favorable conditions, a most wonderful recovery. If neglected, however, the affection is apt to progress steadily from bad to worse. Complication with hysteria offers no material drawbacks. Sometimes the difficulties are very great, but rarely insurmountable.

While the above sanguine view is justified by a large number of cases we should always be guarded in our statements to the patient; we should remember that there are many circumstances which make a complete recovery impossible. A number of important factors must be taken into consideration. They are: First, a history of neurasthenia, or general nervousness in the ancestors; secondly, a history of any affection in the ancestry which suggests the existence of a neuropathic constitution, such as epilepsy, insanity, or organic nervous diseases; thirdly, any trace of neuropathic or degenerate constitution in the patient himself; fourthly, the age at which the neurasthenia begins; and, lastly, the length of time for which it has existed. As to the age at which neurasthenia begins, it is known that persons in whom the affection makes its appearance in childhood are with difficulty brought to the average level of health, and even then are prone to frequent and prolonged relapses. Again, in neurasthenia making its appearance toward the close of the middle period of life, or in old age, the results of treatment are only partially satisfactory, and often disappointing. The reasons are obvious. The tissues in general, the bloodvessels, and other organs exhibit more or less the effects of the wear and tear of life. As to the length of time during which a neurasthenia has lasted, we should remember that long-standing cases are often very intractable. In discussing the symptomatology and pathology of the affection we have pointed out how, in long-standing cases, secondary and terminal organic changes in the tissues arise.

If a temporizing treatment be employed in a given case, and the original etiological factors allowed to remain at work, several possibilities as to the future present themselves. First, the patient may suffer day in and day out from the various distressing symptoms of the affection; and, finally, if the case persist long enough, and if it be of sufficient severity, terminal neurasthenia supervenes. A second possibility is the formation sooner or later of some toxic habit. Among men the alcoholic habit especially in that form known as periodic inebriety, may sooner or later be established. In women the morphia or chloral habit may be formed. A third possibility is the transition of the neurasthenia into one of the psychoses. In persons of a non-neuropathic heredity this outcome is very improbable. The remarks that have been already made in this connection in reference to hypochondriasis and melancholia apply also to paretic dementia, paranoia, mania, moral insanity, etc.

Treatment. When we consider the etiology and pathology of neurasthenia the indications for treatment become very clear. It is evident, first of all, that rest is an imperative factor. Certainly, if the waste be rapid and repair be slow, the diminution of function—the securing of as complete a rest as possible—is the object to be aimed at. We all know that absolute rest, physiologically speaking, is an impossibility; but, at the same time, that the degree of relative rest which is practicable is very great. The first question in a given case is naturally, How much rest does this patient require? It is frequently impossible for persons actively engaged in the pursuits of life to take absolute rest; nor, in fact, is absolute rest always a necessity. Very frequently the most astounding changes can be brought about by relative rest. In the high pressure of modern civilization, especially as it exists in this country, the temptation to overwork is extreme, and in very many cases of neurasthenia, if the excess of work be stopped, recovery will ensue. In many cases we need only adopt the scheme of "partial" rest, instituted by Dr. Weir Mitchell, in which the patient, often an active business man, is directed to prolong the hours of rest in bed, to rise not earlier than nine or ten o'clock in the morning, and to retire with the onset of evening. A man following these directions must necessarily curtail the hours devoted to work, and very often this simple expedient is sufficient to bring about a most favorable result. Let us remember that if our case of neurasthenia be profound and of long duration, that the rest must be as nearly absolute as it is possible for us to make it. Dr. Weir Mitchell has already pointed out how this is to be accomplished; how in very bad cases the patient is not even allowed to feed herself, to turn in bed without the assistance of the nurse, nor to leave the bed to void the bowels or urine. Now, while rest is undoubtedly a factor of prime importance, rest of itself, as Dr. Mitchell has shown, is not without its attendant evils. (See Seguin Lecture and "Fat and Blood.") It is well known that a joint, if not moved, will stiffen and finally become ankylosed; it is well known that a muscle which is not exercised will waste away, and it is probable that analogous changes take place in other tissues. How to combat these evils is the problem which presents itself. If we exercise our patient we expend his strength. Evidently the solution of the problem is to obtain the effects of exercise without this expenditure. Ordinarily we obtain these effects by massage and by electricity. To Dr. Weir Mitchell we owe the introduction of these expedients into the treatment of neurasthenia combined with rest, and it is the combination of these forms of "pas with prolonged rest in bed that constitutes the "rest cure." To massage and electricity we will presently return.

The diet in neurasthenia next claims our attention. The i raise the nutrition of the patient to as high a level as possible, end in view, first, to administer the most readily digested foods, to administer them in as large a quantity as possible. These best illustrated by the methods pursued in severe cases, such as r atic rest treatment. Almost of necessity milk constitutes a la the diet; the patient is placed almost habitually upon milk at f on other food is added. The neurasthenic is, as we know, almos a dyspeptic; he lacks both the desire to eat and the ability t properly. He presents the train of symptoms so familiar un "nervous dyspepsia." Frequently he objects strenuously to the ing over and over again that he cannot digest it, that the milk wi or that it gives rise to pain. The custom under these circumst some way modify the milk, either by the addition of some dil tea or one of the carbonated waters; or peptonized milk or k ministered. Most often, however, it is found that the inability is very much exaggerated, and it is best never to ask a patient "Does milk agree with you?" but simply to order it. We shou however, to order it in small quantities, beginning with about every two hours, and excluding absolutely all other food. This course, insufficient for the needs of the body, but we find that, e gust for milk is present, the patient being placed upon a very s of food, and becoming in a day or two very hungry, becom grateful for the milk, and takes it eagerly. We should next milk very slowly, being careful at first to keep the patient a l all of the time. Finally, in the course of a week or ten day increase the amount to eight, ten, or even twelve ounces every t the case may be. If we find that the patient is quite hungry b or fifth day, it is well to add a small slice of stale bread with or twice a day, and later on three times daily. The diet is the creased by a soft-boiled egg, or perhaps by a mere fraction thereof Finally, a small chop or steak is given at noon, and a small thoroughly boiled rice may be given at supper. Upon these b substantial diet is finally built up, until the patient eats three la day; such, for instance, as a breakfast of fruit, cracked wheat, one boiled eggs, or a good-sized steak or several chops, bread and butte a dinner of a good slice of roast beef, with vegetables and boiled r of potatoes). The supper should remain as a light meal of br fruits, light pudding, and milk. It will be noticed that in this di chocolate, tea, and cocoa are absolutely omitted.

Soups, beef-tea, and broths possess relatively little value; the cupy space which can otherwise be given to milk. Certainly th a far higher nutritive power. The same holds true of tea, ch cocoa, while coffee is exceedingly objectionable, inasmuch as the n is an individual who has, in the vast majority of cases, already exha lants—not only coffee, tea, and alcohol, but also the various narco vain hope of finding relief. The writer believes that coffee and a in moderate use, should be avoided.

Another element of importance in the treatment is, as Mitchel fair have both pointed out, the *isolation* of the patient. Not patient the victim of a neurasthenia, but in very many instances teria. Neurasthenia and hysteria are, as we have seen, often i intertwined. Under these circumstances isolation, the withdrawal of the patient from the influences of relatives and friends is of the utmost importance. How deleterious home surroundings are under these circumsances need not be dwelt upon. In cases of nervous prostration which are sufficiently pronounced to require rest in bed, isolation is imperative, and it should be absolute. No exceptions should be made in favor of any relative, nor should any communication ever reach the sick-room, except through the mouth of the doctor, and then even should be most guarded and most general in character. The writer's experience accords with that of Drs. Mitchell and Playfair, that even slight infringements upon this rule are sometimes followed by the most unfavorable results.

We find, then, that our resources for combating profound neurasthenia comprise rest, artificial exercise (namely, massage and electricity), a special diet, and isolation.

The success which attends our efforts in a given case depends largely upon the way with which the various means at our disposal are utilized. No doubt every one who has essayed the rest-cure has developed certain methods of his own which he finds give him the best results. The writer's experience has led him to adopt the following: The patient is placed in bed. Most often it is a woman, and as a rule she is extremely nervous, and perhaps hysterical. Frequently she is a stranger amid strange surroundings. She is left by her friends in the care of a physician whom she knows only by reputation, and of a nurse of whom she knows less. It is my custom, therefore, to begin treatment in the most gradual manner. It requires time, in the first place, for the patient to become accustomed to her bed, for lying in bed is in the beginning quite a task to even neurasthenic people, and, in the second place, it requires time to become acquainted with and acquire confidence in her nurse. The writer, therefore, at the first visit is in the habit of examining the patient thoroughly, if he finds that the examination is well borne and causes no excitement, but only in part if she be very nervous. Frequently he does not finish the examination until the next or even the third visit. He simply orders a small quantity of milk, as already explained, and instructs the nurse that she give the patient that evening a light and rapid sponge bath. Generally massage should not begin until the second or third day, and then should only continue for a short time, and should be very gentle and superficial in character. The reasons for beginning the massage in so gradual a manner are: first, that the patient may become accustomed to the touch of the nurse, and, secondly, because the gentle, superficial stroking soothes the patient. We should take advantage of this quieting effect and direct that the massage be given in the evening, At this time it strongly favors sleep, and is indeed one of the most powerful agents for combating insomnia at our command. Just as the diet is very gradually increased so should the massage be very gradually increased, both in depth and vigor; finally, the duration of the massage should be increased to at least an hour. Dr. Playfair recommends that the patient be masséed for even three hours. It is doubtful, however, whether anything is gained, if indeed something be not lost, by this prolonged rubbing.

Another point which the writer regards as important is that the massage be performed by the nurse; this of course makes it necessary that the nurse be also a masseuse. If the patient be treated by a regular masseuse at certain intervals in the day, the visit of this third person, with whom the patient has also to become acquainted, acts as a disturbing factor; to use an everyday expression, the patients are apt to be "upset" by it.

Electricity is not of the same value or importance as massage. However, it is a remedy which the writer almost invariably utilizes, but generally as follows: In the first place, almost all that can be gained by artificial exercise can be gained by massage, and we must remember that most excessively afraid of the battery. However, as in the case of application must be begun in a very gradual manner. A scar ible current is at first used, and the nurse, who has been previous in the points of Ziemssen (see Chapter XXXIV.), is made to use t terrupted current in such a way that each group of muscles cont number of times. Until the patient becomes accustomed to this o ant sensation the application may be limited to the forearms and it may be applied to the thighs, arms, and trunk. Electricity is useful adjuvant to the rest-cure, but it is only an adjuvant. At th its utility cannot be questioned. The writer never uses it early or i ning of a case. He is fearful, and in fact such has frequently been ence, that the excitement and the irritation consequent upon its use ously upon the patient. Moreover, the exercise that it gives frequently tires and exhausts, and it occasionally retards the increa which otherwise takes place. We should begin with it only several the treatment has been well under way, and sometimes in the latt case, preparatory to getting the patient out of bed.

After our treatment has been continued for some time the que How shall we determine whether we are making satisfactory pr the first place, if the patient is taking a large amount of food, a is having its proper effect, the color of the patient should imp patient should, as the masseuse expresses it, "pink" readily under The muscles should gradually become firmer to pressure. Howev and more important guide than this is the change in weight sho patient. Occasionally it is noticed that in the first few days the gressive loss of weight, but soon the patient begins to gain, a average case, gains rapidly; sometimes in the course of from eigh weeks as much as twenty-five, or even thirty-five, pounds. The confirm from personal observation and personal experience muc been said upon this subject by Drs. Mitchell and Playfair, and he h to regard the progressive increase in weight as the most valu attainable regarding the progress of a case. It is far more valu the persistence or non-persistence of such symptoms as backache or or general nervous feeling. Some of the subjective symptoms disa tively early, others persist; but even the latter, in the majority of c fainter and fainter, until at last they no longer impress themselves consciousness of the patient. In those instances in which obscure sensations seem to be permanent, it is not improbable that more o nite changes, "the terminal changes," have taken place, and t persistent symptoms are due to the latter.

It will be noticed that in the above plan of treatment drugs find However, it is occasionally judicious to use a few remedies. Not infr as we have seen, the indigestion of our patients is complicated by a gastric catarrh. Indeed, this is more frequently the case than writer is in the habit of prescribing nitrate of silver, say one-fourth o combined with a fourth of a grain of extract of hyoscyamus, to half an hour before meals. Sometimes, also, at the beginning of ment we find that the patient's tongue is coated and that the b loaded. In such cases it is well to prescribe small doses of cal bicarbonate of soda until the desired effect is obtained. In other w eral principles must guide us in the use of medicines in these cases as far as possible, medicines are to be avoided. In a number of cas tive of some sort becomes necessary. The choice of this is largely of personal judgment; the simpler the remedy the better. The w the habit of using the fluid extract of cascara, given at night, and, if possible, in gradually diminishing doses.

Occasionally special symptoms require special interference. It may be that the headache is so intense as to demand active medication. In this case phenacetin or antipyrin may be used, and sometimes moderate doses of bromide of ammonium may be given at the same time. Frequently, too, the insomnia is so profound that it does not yield to the general treatment. We find, however, as a rule, that patients who are taking a large amount of milk sleep a great deal. The excess of food seems to have a soporific or sedative influence; and, therefore, narcotics are rarely indicated. The massage, too, if given in the latter part of the day, favors sleep. Sometimes, though not always, a wet sheet, followed by a gentle rubbing, or a hot sponge bath, rapidly given, act as sedatives. Occasionally, however, the insomnia is so profound that we are driven to the use, for a time at least, of drugs. The milder drugs, small doses of sulphonal or trional, or possibly of bromide, should be given (see also p. 85). The stronger narcotics should practically never be used. Fortunately, in the average case we can get along without them.

Strychnin, so much vaunted in neurasthenia, is a drug rarely used by the writer. In a large number, perhaps the majority, of cases the treatment can be conducted successfully from beginning to end without the use of any other medium than an occasional laxative. However, in those cases in which the neurasthenia is complicated by anæmia, iron or arsenic may be used with advantage. If the analysis of the urine has revealed an excess of uric acid, as is so frequently the case, special remedies applicable to the condition may be given. The alkalies are indicated, and the urine often clears up rapidly under their use. However, the writer has been especially successful with the administration of piperazin. About one gramme should be given daily for a time. It should be administered largely diluted. One gramme having been dissolved in about a pint of water, the solution should be given in divided doses at intervals throughout the day until the entire amount is consumed. If, on the other hand, oxaluria be present nitro-hydrochloric acid is indicated.

Let us suppose now that the patient is progressing favorably, when is the rest in bed to terminate? How are we to know when the maximum amount of good has been obtained? In neurasthenic cases of long standing it is probable that our best guide is the change shown by the body-weight. If a decided increase has taken place, and it then ceases, it is probable that the maximum increase has been reached—*i. e.*, the maximum increase possible under the treatment. If, at the same time, the patient's symptoms have become progressively less marked, we have probably reached a period when the patient should be gotten out of bed. In young neurasthenics, however, and in others in whom the neurasthenia has not been so profound, the increase in weight is not of itself a sufficient guide, inasmuch as these patients will sometimes grow needlessly fat. In such cases we are to consider whether the weight is about normal to the height and age of the individual. In getting the patient out of bed we must remember that, though well nourished, she (or he) is weak. We must remember that, though the muscles have been thoroughly rubbed, and though they have been toned up by the battery, the patient has not exercised for weeks and months. She is in the condition of having accumulated an enormous amount of *latent* energy. This energy must now be mobilized—made potent by gradual exercise. She is allowed, therefore, to sit up for five or ten minutes in a day. While in bed passive movements of the legs and arms are made. Gradually the length of time for sitting up is increased, so that the patient sits up twenty minutes to forty-five minutes or an hour a day. Little by little the time is increased end of ten days the patient is up from four to six hours.

Passive movements, which until now have been practised, are di For them Swedish movements and light calisthenics are subst patient is also made to walk about the room a little. Finally, a s of the house or a carriage ride follows. Next comes a trip to th some ten days or three weeks. During this time the patient is ma in the open air. As a rule, she walks little at first, but gradually amount until two or three miles at a brisk gait is attained. Th little by little, discontinued, and during the stay at the seasho immersion in a hot salt-water bath, say twice weekly, is ordere mersion should be very brief, because a prolonged bath in some be followed by a sense of fatigue rather than exhilaration.

Gradually the patient is permitted to renew her relations with and friends. Finally she is returned home, and, in order to ins relapse, which under proper precautions rarely occurs, she is t some ten hours in bed out of the twenty-four, to still take her bed, and to still keep up a moderate quantity of milk in add regular diet. Daily exercise is also insisted upon. Little by little break in upon the rules laid down by the physician at parting course of a number of weeks adopt the lives of the people about

In the rest cure time is a necessary element in achieving a du The patient should have the benefit of from six to twelve we ment, and in some cases even more.

One of the most powerful therapeutic adjuvants in the treatme thenia, and one that is much neglected in this country, is hy This implies both the internal and external use of water. We h pointed out that the large majority of neurasthenics have defi and, in consequence, consume too little liquid. There can be no some of the benefit derived from a prolonged course of milk die ordinarily used in conjunction with the rest-cure, is to be attrib increased ingestion of liquid thus necessitated. In most cases, in the milk that is added to the diet, it is necessary to urge the pati freely of water, and, in order to secure compliance with this advi to prescribe a certain amount of some special water daily—fo Poland Spring water, or a carbonated water, as apollinaris or selt we have pointed out in discussing the pathology of neurastheni toxic substances circulating in the blood, the free ingestion c urgently indicated, and we need only recall the experiment haustéd frog-muscle, in which washing out the fatigue substan completely restores the electrical excitability, to realize the valu measure.

Water, when applied in the form of douches, showers, or spr surface of the body, acts in two ways: first, by the impact of the the surface of the body; and, secondly, by the stimulating effect perature of the water. Cold water applied suddenly and for sho acts as a powerful stimulant. It is at once followed by a reaction culation of the part. This reaction, after the shower-bath or afte bath, may be enhanced by vigorous rubbing with a towel. It is a ter to attach to the water-pipe in a bath-room a section of a garden-hose with a sprinkler, and this, for practical purposes, an well. Elaborate apparatus can be found in only few public instit is rarely at the disposal of the practitioner. The manifold uses of rapy in neurasthenia at once becomes evident when we learn that of "tender spine" yield more rapidly to vigorous douching than t should be a part. As before, tea, coffee, tobacco, and alcohol should be excluded. The exercise should consist of walking in the open air and of light gymnastics performed under the eye of a physical instructor. Little by little, as the case progresses, the amount of exercise and also its character may be modified. Whenever practicable, out-door exercise, such as horseback riding, should be substituted for in-door work. Running and swimming ought not to be indulged in, unless the patient has made very marked progress toward the goal of health. Running is very apt to tax the heart, while swimming is often followed by depression and exhaustion instead of exhilaration. These remarks apply to the after-bed treatment of profound neurasthenia as well as to the milder cases. Great judgment is required to prescribe the amount and character of the exercise for each individual case, and upon it the success of the treatment often largely depends.

It is often advisable to send our patients away from home. Some small hotel or cottage at the seashore at which the life is quiet and tranquil should be selected. Here, with a certain amount of exercise, such as walking, appetite and digestion rapidly improve. One of the principal advantages of a stay at the seashore is the securing of a wholesome sleep. Indeed, it constitutes one of our most effective means of combating insomnia. After awhile the patient may bathe in the surf, and, with a view to this, the place selected should have a beach with a very gentle slope. A steep beach with a rough surf may be followed by unfavorable results. The bath, of course, should last for a few minutes only.

Occasionally it is of advantage to send the patient on a sea-voyage. As a rule, the longer the voyage the better. After the first few days of seasickness have passed away an increased appetite follows, and there is the same soporific influence of the sea-air as before. Further, on board of a vessel the patient experiences all of the beneficial effects of isolation. The monotony of the daily life also predisposes to a calm and even frame of mind.

In sending out patients other than to the seashore we should remember, in our choice of climate, to be especially careful not to send them to high altitudes. The air of high plateaus and of mountains may increase many of the nervous symptoms. It is apt to increase the headache, and especially to produce insomnia, or to make it worse if already present. As patients improve, however, a gradual change from a low to a high sea-level is often on the Insanity of Overexertion of the Brain. Tuke, among o for combating the insomnia of grave brain exhaustion, has u freely for the last three years. He states that in doses of 15 gr hours it usually produces sleep, and that the general conditio proves, and also that he has "frequently given antipyrin to the to 100 grains per diem for a fortnight at a time," and that h noticed any of the ill-effects ascribed to it, except occasionally nau ing. It may be that patients in the condition we are speaking ity of overexertion of the brain) may be more tolerant of th those in whom temperature runs high. As in all nervous cases, at first a matter of experiment; but it is quite safe to begin above indicated, raising it somewhat, and administering it mo according as the patient's condition improves." Of course the w urge the use of such large doses of antipyrin in every-day forn thenia. In his experience much smaller doses suffice. Howeve of Batty Tuke in the use of antipyrin in the insanities of ove exceedingly important as demonstrating that we have here a d value. The writer, however, wishes here to repeat the staten made, that whenever possible the use of medicines is to be avoid

In the foregoing remarks upon the treatment the writer h merely general principles. Of course special forms of neurasth special methods. This is particularly true of that form in w symptoms predominate, namely, "sexual neurasthenia." Here it first to correct the sexual life of the individual. Abstinence sho ble be enforced. At the same time, advantage should be taken ures at our command for raising the general level of the pati Among these, active exercise in the open air, outdoor sports, the ship of healthy comrades, together with wholesome reading, a important. The special symptoms, however, usually compel us various forms of medication. The sexual apparatus, as we have is in a condition of irritable weakness, and one of the most distre toms to the patient is the frequency of the nocturnal emissions. mides are again of value, but their usefulness is secondary to tha hydrobromate, which should be given in doses varying from 1-80 a grain at bedtime. In some cases it is expedient to administer b hyoscin together. If the weakness of sexual power be accor markedly diminished cutaneous sensibility, the use of urethral together with a dry wire-brush, may be resorted to. Hot and c to the spine may also be employed.

In many patients who complain of sexual weakness, the affectio rather than physical. In such instances, true sexual neurasthen exist. Many such cases require only a few words of good advice. however, the condition amounts to one of actual hypochondria. class which taxes our resources to the utmost.

Before closing it is necessary to speak briefly of the animal e efficacy of which has been so much vaunted. The results do not, in t opinion, justify the claims. Distinct exception must, however, be n statement as regards extract of the testicle. There can be no doubt when the possibility of suggestion is excluded, the use of this re benefit. It appears to act as a stimulant, and will occasionally e make an impression upon a case otherwise intractable. The nu being introduced to the profession apparently have a similar actio

CHAPTER III.

GENERAL MORBID STATES OF THE NERVOUS SYSTEM.
(CONTINUED.)

BY JAMES HENDRIE LLOYD, M.D.

HYSTERIA.

In the vast literature of the affection we are about to consider a few names only rise conspicuously above the general level. These are the names, first, of those who by reason of their authority in medicine did little more than establish definitions and terms, often erroneous, which for long periods have encumbered the science, and, secondly, of those who have corrected these errors and have advanced our knowledge of this remarkable disease by the accuracy of their description of its symptoms. To the right comprehension of hysteria a brief historical study is more essential than in the case of almost any other affection, because, more than in the cases of most others, the knowledge of it has been obscured by definitions which are of great antiquity. Some of these venerable traditions still confuse the subject in spite of the progress of recent scientific criticism, and their influence in various ways is still wholly pernicious.

In tracing the history of hysteria it is almost sufficient to trace merely the history of one dominant idea. This is the idea that the womb is the seat of the affection. From this one central thought all others rose and radiated for two thousand years. With slight variations of expression this uterine pathology served the purposes of science, and even until to-day has satisfied the requirements of the people and of a portion of the profession. This tradition, probably the most ancient in medicine, has held an ascendency which is not often acquired except by some mystic creed, and unfortunately it has been one of the last of the primitive errors to lose ground. It gave origin to many grotesque and not a few obscene conceptions; and created a prejudice against and a scandal about the very term *hysteria*, that still adhere to it with the persistency of an evil reputation.

Among the earliest writers Hippocrates, and, later, Plato held the fanciful doctrine of the wandering womb. Hippocrates taught that the hysterical globus was caused by the ascent of the womb against the liver and diaphragm. Plato[1] said that the womb is an animal that desires ardently to engender children; when it remains sterile it controls itself with difficulty; it is indignant; it wanders about the body, obstructing the air-passages, arresting respiration, throwing the body into extreme dangers, and causing diverse maladies. Aretæus accepted this incredible doctrine; for him the womb was an animal within an animal, enjoying delicate odors. He said that it was possible to drive it down by presenting fetid odors to the nose, and to drive it up by presenting the same odors to the vulva. This opinion, unworthy of an untutored savage, controlled practice down to the middle ages. He was responsible for the opinions that young girls alone are subject to the disease; that elder, married women escape it—an opinion not altogether banished to this day.

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[1] Timæus, p. 568. Works of Plato. Translated by Taylor, London, 1804.

Galen taught that hysteria was caused either by the suppression of the menses or the retention of the semen (meaning by this the *female* principle). Henceforth this latter idea has great prominence. The retained semen, undergoing putrefaction, became a violent poison, and had especially injurious effects in women who, having been habituated to sexual indulgence, were deprived of it suddenly. To effect the discharge of this semen Galen recommended an indecent practice. Thus the subject became identified gradually with the idea of sexual excesses and perversions.

Aetius and Paul, of Aegina, wrote that hysteria attacked preferably young girls who were lascivious and sterile.

From these authors there is a wide gap down to the early part of the seventeenth century.

The earlier of the modern writers merely repeat the ancient pathology. For them, as for the ancients, the disease was confined entirely to females past puberty. Forestus was the first who attempted to distinguish hysteria from syncope, apoplexy, epilepsy, and lethargy, and described well the phenomena of the disease; and yet, like his predecessors, he exaggerated the sexual and libidinous character of these patients. He even pretended that hysterical women conceived more easily than others, implying that this fact was due to their greater sexual passion. Sennertus apparently believed in the infectiousness of hysteria, for he speaks of the danger to which one is exposed from those dead of the disease.

The first writer to recognize hysteria as a disease not confined to the female sex, and hence not dependent upon affections of the womb, was undoubtedly Carolus Piso. His name ought to take high rank among those who have contributed to the knowledge of the disease. His work should have constituted an epoch. Yet it was not to be so. The old ideas were too deeply rooted to be eradicated by one man. Piso, or Le Pois, was the first who distinctly gave to hysteria a cerebral origin. For him the disease was caused by a serous exudation which compressed the brain.

Willis elaborated this idea. The origin of hysteria is frequently remote from the womb, and is in other organs. He recognized sudden terror as a cause, and thus anticipated the ideas of the present time, especially on trauma.

From this point begins the modern epoch of hysteria, which is characterized by a steady departure from the ancient pathology of the uterine origin of the disease. This has been, however, an extremely slow process, marked by periodic reactions. Even yet it is not complete.

Sydenham's was the first great name of this period, as Charcot's has been the last. Sydenham taught that hysteria and hypochondria were identical, being distinguished by the fact alone that the former occurs in the female, the latter in the male sex. He believed it to be the most frequent of chronic maladies, and explained it by a humoral pathology.

Astruc insisted upon marriage as a remedy for hysteria—a fallacy which exercises an influence to this day. It is based, of course, upon the primitive idea of a predominant sexual element in this disease.

Whyte was the first who approached the subject in the modern scientific spirit. With him the sexual idea falls into the background. He finds a number of causes for hysteria, among which are the neurotic temperament, prolonged illness, as fever, and gout.

Tissot believed that the ancients were wrong in regarding the womb as the sole cause of hysteria.

Dr. Rush[1] placed the seat of the disease in the nervous and muscular systems.

In 1816 Louyer Villermay[2] wrote a treatise which marked the height of the last reaction toward the ancient pathology—a book, of which Briquet said, that it ought to date from 1500 rather than from 1816. This author admits, like the ancients, the existence of a sperm or semen in the female; he reverts to the belief in the peregrinations of the uterus, and consecrates his work to making of hysteria a disease of lubricity, a shameful affection, the victims of which are objects of disgust or pity. It is not too much, perhaps, to say that Louyer Villermay is responsible for not a few of the reactionary ideas that have characterized the treatment of hysteria in the present century. He was refuted by Georget, whose observations were made at la Salpêtrière, in which hospital at a later date it was reserved for Charcot to establish this subject of hysteria upon a basis at once scientific and enduring.

Before him, however, Brodie,[3] in England, wrote his short essay on local hysterical affections, a work that has no superiors for originality. It forecast the whole vastly important subject of traumatic hysteria.

The masterly treatise of Briquet, which may be said to have been the forerunner of the school of Charcot, appeared in 1859. To the latter and his pupils we can refer here but by name. The chapter which follows is based largely in method and spirit upon the scientific foundations which they laid both broad and deep, although for clinical material and illustrations it draws almost entirely from original sources. We are indebted especially to the treatise of Gilles de la Tourette for much assistance in literary reference.

Definition. Hysteria is a psycho-neurosis, of which the physical symptoms are the most conspicuous, tending to disguise the mental phenomena and to simulate superficially, the effects of various organic diseases. It is not a simulated disease, although occasionally, like all diseases, it may be feigned; but it is a genuine and profound affection of the cerebral centres, which presents many clinical and physiological questions of great interest.

Hysteria is not, as so many authors have claimed, a protean affection, aping all other diseases and having no identity of its own. On the contrary, it is a distinct morbid entity, with a natural history, and with a train of symptoms that are subject to well-defined laws and may be submitted to a methodical study. It is the object in this chapter to make such a systematic presentation of hysteria.

It has been too much the custom to regard hysteria as a conglomorate affection made up mostly of fantastic, assumed and purposive symptoms, which are the evidences either of foolish or of designing women. Hence hysteria is still confused by some authors with folly, mendacity and malingering. In this chapter the disease will be demonstrated to be something far other than these.

Finally, hysteria in its major stages has some manifestations that appear to ally it with other grave nervous diseases, such especially as epilepsy, chorea and insanity, and it has received names, such as "hystero-epilepsy," "chorea major" and "hysterical insanity," which have the sanction of very respectable authorities and which are intended to indicate such alliances. Nevertheless it will be maintained in this chapter that such terms are erroneous, because hysteria is a distinct affection, and that where it seems to have such alliances the appearance is delusive and that in reality the symptoms are purely hysterical. By this, however, it is not meant that two or more distinct neuroses, such as epilepsy and hysteria or hysteria and not coexist in the same person, but only that, even if co-exi distinct. Hence the error of attaching to some of the phenon hysteria terms derived from other diseases will be avoided in tl

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[1] Diseases of the Mind, p. 259.

[2] Traité des Vapeurs, ou Maladies Nerveuses, et particulièrement de l'Hystérie et de L'Hypochondrie. Nouvelle Edition, Paris, 1832.

[3] Lectures Illustrative of Certain Local Nervous Affections. 1837.

Etiology. The causes of hysteria have been investigated and classified by Guinon.[1] He shows from the ancient authors developed not infrequently in former times under the influe which were not recognized. The conclusion is forced upon us ical study that hysteria has existed as at present at all times, an error of doctrine has prevented it from being judged at i Some causes of hysteria have been recognized, nevertheless, modern times, and it is probable that even they do not com This is to be explained by varying conditions of life and envi as changes and developments in social and industrial elements. bring some causes more conspicuously forward, as, for instan modern life; or create others, as surgical anæsthesia; or allow o into insignificance for long periods, as political revolution and causes again are peculiar to locality, as earthquakes; others to c as poisoning by mercury.

The causes of hysteria may be divided into six classes as follo

Heredity. This is the most important and extensive cause of is in fact the only one that can be said to be almost universa one great predisposing cause, all others are only exciting causes, the circumstances of time, place and person. So uniformly h recognized that it did not escape even the ancients. Hippocrat heredity in most diseases, as when he said that phlegmatic person phlegmatic, bilious persons bilious, and phthisical persons phthi teria, in fact, may be included in the class of constitutional o psychoses, which class includes at its other extreme the types of insanity. Statistics, as well as common observation, prove the assertion. Thus hysteria has been observed to be transmitted not from an hysterical parent, but indirectly, or by *transformation*, fr afflicted with one of the more grave psychoses, such as epilepsy This is an indication that the neurotic constitution is developmen result of evolution acting through innumerable generations. Geor many years since in the Salpêtrière that hysterical women had, near kindred, hysterics, epileptics, hypochondriacs, lunatics, and t blind. Briquet[2] demonstrated the truth of this fact by a statistic

His first table shows that 351 hysterics had 1103 near relative tories could be traced. Of these relatives there were 214 hysteric tics, 16 insane, 14 with convulsive diseases, 3 somnambulists, a other nervous affections—a result of 25 per cent. His other tabl healthy non-hysterical women had but 2⅛ per cent. of neurotic rel subjects whose hysterical careers began with convulsions had 28 neurotic relatives, which rate was shown also by relatives of those teria had begun before puberty. On the other hand, those pat attacks had come on gradually had but 19 per cent. of kindred neuroses. From all this it appears very clearly that the heredit is in direct proportion to the severity of the hysteria, and that n have only about one-eighth as much of a neurotic element in th as have the hysterics.

Striking instances are given of the prevalence of hysteria in same family. Landouzy[1] saw five sisters attacked with hysteria, and says that Bernutz saw six daughters, born of an epileptic father, the victims of hysteria. I have seen recently an only daughter with hysterical paraplegia, whose only brother is epileptic, whose father is a confirmed inebriate, and whose mother has the marked eccentricities of a mild grade of paranoia.

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[1] Les Agents Provocateurs de l'Hystérie. Paris, 1889.

[2] Briquet: O

Reynolds[2] on the other hand thinks that association and the bad influence of an hysterical mother explain the transmission, which, therefore, is not by heredity proper, but rather by example. When this author states, however, that "hereditary taint has not been shown to exert any marked influence in the development of hysteria" he exhibits the lack of the faculty for judicial criticism and the disregard for literary and clinical evidences which unfortunately characterize many British writers on hysteria.

Trauma. Next to heredity, trauma ranks in importance as a cause of hysteria; not that it is so frequent as some others, but that it gives rise to grave consequences and medico-legal complications. It may cause most or all of the symptoms of hysteria, both paroxysmal and permanent; but it is prone to cause some of the latter class especially. Among these are localized palsies, anæsthesias, hyperæsthesias, tremor, and contraction of the visual fields. These effects are confined by no means to the female sex, but some of them, as tremor and paralysis, are observed especially in men. From the practical standpoint the recognition of traumatic hysteria is of the utmost importance. This is so for two reasons: first, because of the prevalence now of accidents by railroad and by machinery, leading to litigation for damages; second, because recently the subject has been obscured not a little by controversy and by the use of euphemistic terms.

Of all the forms of hysteria this hystero-traumatism has the most modern aspect. This is due to the fact that the vast extension of railroads has greatly increased the frequency of the peculiar morbid entity to which the name applies. It would be a mistake, however, to suppose that cases caused by railroad accidents, or cases that lead to litigation, alone display these symptoms. Any form of traumatism, even the slightest, may give rise to the most obdurate symptoms. Hence, it is not probable that this class of cases is of strictly modern origin; in fact, traces of such cases can be found in medical literature before the present era, but they were not recognized at their full value, because both of their rarity and of the imperfect knowledge of hysteria which formerly prevailed. Brodie's[3] monograph on this subject was far ahead of his time; it is remarkable that it exerted so little permanent influence and seems to be so completely forgotten or overlooked by the followers now of the German school. Brodie, in the simplest and plainest way, described many of the interparoxysmal symptoms of traumatic hysteria, and illustrated the subject with records of many typical cases. Among symptoms observed by him were hysterical contracture, astasia-abasia, paralysis, hysterogenous zones, neuralgias, aphonia, anuria, and characteristic mental stigmata. The works of Erichsen and of Page mark the beginning of the modern interest in this subject; the former explained these cases by the theory of a spinal concussion, the latter gave them a psychical origin. From the time of the appearance of these books the subject has been discussed with acrimony. Various terms have been used to avoid the use of the dreaded term *hysteria*, among the most common of which are "railroad spine," "traumatic neurosis," and "traumatic neurasthenia." In Germany, and later in England and America, under the influence of Oppenheim, the tendency has been to differentiate a special form of neurosis, the result of accident, to which the name of hys-

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[1] Quoted by Gilles de la Tourette.

[2] Art., Hysteria. Reynolds' System of Medicine.

[3] Op. cit.

teria is denied, but which is based, nevertheless, upon many o
well-recognized stigmata of hysteria; while in France, under t
school of la Salpêtrière, a more strict and consistent nomenclat
adopted, and, in consequence, a more scientific presentation o
has prevailed. It seems probable that a more scientific criti
prevail in America, for until more interest and care are shown
the stigmata of hysteria the followers of the German method m
defensive. Their "traumatic neurosis" appears to be hypothet
Moebius[1] says that it is a form of hysteria, and Jolly that it is
voked by trauma.

Apart from litigants it is not denied that trauma causes hy
predisposition of the patient is a more important element than t
the accident. Accidents, accompanied with shock, and especiall
are potent causes, hence the degree of actual physical injury
great. Hence it is, also, that railroad accidents, which excite
cause so many of these cases. Earthquakes have caused invete
traumatic hysteria; one such case was seen by the writer in a n
persistent hysterical tremor for years following an earthquake an
wave, to which he had been exposed in a South American por
case of tremor has been seen recently in a young woman follow
surgical operation under ether. Other causes are dog-bites, lig
tornadoes. This class of causes is closely allied, therefore, t
emotional shocks in non-traumatic cases, which will be considere
age, sex, or condition is exempt from this form of hysteria.
worst cases occur in men.

Still another group of cases is ascribed to so-called internal
such as severe visceral disorders, painful colic, etc. These cases b
erly to the class of hysterics caused by disease, which will be discu
To these belong also the cases occurring after childbirth.

Acute and Chronic Disease. In persons predisposed any pro
ease, or even an acute illness, when accompanied with suffering,
ment, depletion, and too much introspection, may cause one or
symptoms of hysteria. I have seen, and shall report briefly later, so
able instances of this. One was in the case of a young lady who
gone an arthrectomy of the knee-joint, followed by protracted con
she developed hysterical rapid respiration to what appeared to b
ing extent. I have seen a typical hysterical aphonia, with othe
develop in a girl who was bed-ridden with a chronic disease. It
that some of the older cases of so-called aphasia occurring in the
were similar to this one. Churchill[4] collected reports of cases o
after childbirth, as did also Imbert-Gourbeyre[5] and Poupon,[6] am
hysterical aphasia (aphonia?) and hemiplegia are plainly discernibl
occurring after infectious diseases, such as typhoid fever, scarlatina,
and influenza, the diagnosis may remain for a while uncertain
some form of hysterical paralysis and one of the infectious forms

Brodie describes a typical case of hysteria in a woman followi
fever.

Diabetes has produced hysterical symptoms, and we have th thority of Fournier[1] for the statement that secondary syphilis has excited the crises of hysteria in women previously subject to it. Raymond and others have also written on this subject. This potency of syphilis to excite hysteria may be ascribed to the rather sensational character of the disease and to the profound impress that it makes upon the imagination.

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[1] Quoted by Gilles de la Tourette, whose discussion of this subject, both historical a
complete and convincing. The literature of traumatic hysteria in France has gro
proportions, and must be consulted by all who wish to obtain a thorough knowledge of

[2] Guinon has presented this aspect of the subject with clearness and force in his
"Les Agents provocateurs de l'Hystérie," Paris, 1889.

[3] See Rev. Neurologique, October, 1893.

[4] Dublin Quart. Journ. Med. Sci., vol. xvii.

[5] "Des Paralysies puerpérales," Mem. de l'Acad. Imp. de Méd. Tome xxv.

[6] L'Encéphale, 1865, p. 393.

Emotions and Moral Shock. Closely akin to the mental states accompanying traumata are emotions of various kinds acting as causes of hysteria. The depressing emotions, although not exclusively, act thus. The most common are fright, chagrin, disappointment, grief. As said already, fright is an especially active cause in traumatic cases, but it may act with equal force in non-traumatic cases. Chagrin and disappointment are active causes in the young, and in doubtful cases, in which hysteria is suspected, such moral causes ought always to be sought for. Grief probably plays a subordinate part in causing the disease, unless it be accompanied with other emotion, such as sudden shock. It is common to ascribe to love affairs great potency in causing hysteria, and this probably because of the ancient idea of a sexual element in hysteria; but this asserted tendency is probably much exaggerated. Mere sentimentalism and emotionalism do not fall necessarily within the bounds of the modern strictly defined hysteria. Genuine grief or disappointment in love may act as a cause.

A common cause of hysteria in all ages has been religious excitement or emotion. This is seen in this country among the devotees of the more emotional sects. It is not uncommon to observe this cause among negroes in their camp-meetings or revival services. These epidemics of hysteria assumed immense importance and great proportions during the middle ages; their history has been written completely by Charcot and Paul Richer. They depended always upon one principle, which ought never to be forgotten, and which still has importance every day—the principle of imitation. This acts with equal potency sometimes in non-religious epidemics, as in schools, and even in hospitals. Thus, recently in a general hospital ward in Philadelphia, in which several cases of hysteria of severe grade were being treated, the affection threatened to spread to other patients, and required that the hysterics be removed and isolated. Of historic instances, one of the most important was the epidemic of Salem witchcraft, which owed its origin to some hysterical children, and was supported in the perpetration of some of its greatest atrocities by the testimony of the same hysterical perverts.

Defective Education. As said already, it is claimed by some that the effects ascribed to heredity as a cause of hysteria are the effects rather of evil example, and, presumably, of bad or defective education. It cannot be doubted that the two classes of causes are closely allied, but it is a grave error to confuse the two or to identify the one with the other. On the other hand, it is extremely difficult to distinguish the actions of these causes from one another. In general terms it may be said that the training or environment that leaves undeveloped or undisciplined the will-power and the reason, and that permits habits of self-indulgence and the excesses of the emotions and the imagination to go uncurbed, is the one best adapted to foster the hysterical predisposition. This is perhaps a somewhat scholastic statement of a most practical subject, yet even such a practical observer as Brodie ascribed hysteria to a defective volition. Perhaps it is best to permit the mind, after its own laws, to adjust the natural and wholesome mutual play of its own faculties, and not to attempt to analyze them too closely in the hope to find in psychological terms a statement and a remedy for this disease. A wise education, under proper instructors, supplies the best environment for the proper development and mutual adjustment of all the faculties, and th of it are discipline, self-control, and the exercise of the mind i rational pursuits. But the scope of such an education cannot here. On the other hand, it is a grave question whether eve education, even universally applied, could eradicate or suppress grand neurosis. Its roots are too deeply fixed in the constitution to render it probable that it could be controlled by education in some of the existing causes already named. We must be conte general statement that defective training permits a more free acti causes which a wise education would do something to hold in ab

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[1] Leçons cliniques sur la syphilis, 1873, p. 816.

Toxæmia. The relation of toxic states to hysteria presents novel, and a most important, question. The subject is not entir the complete recognition of it in all its aspects remains still to l especially by writers in the English language. The most that w to hear in this country is that confirmed alcoholic topers may pre the hysterical stigmata. This, in fact, is a matter of common because alcoholic intoxication is a common vice. Drunkennes alent that all its effects are sooner or later recognized. Hence long been regarded as an hysterogenous poison. But what is familiar alcohol is also true of the less familiar poisons, such as cury, and sulphide of carbon. Another reason for the confusi about the hysterogenous effects of these poisons is the fact that produce grave organic lesions, especially in the peripheral nerv Hence the tendency is to attribute all nerve-involvement to orga produced by the particular poison, and to confuse the hysterical sti the symptoms of such organic change. It is only within a com recent period, and chiefly in France, that this subject has been i with understanding and profit.[1]

Hysteria in all these cases is due probably to a psychic impressi fright, apprehension, or worry, the direct effect of the consciousn patient that he has become the victim of a poison. Hence the causes in these cases is not unlike that of trauma and acute dise probably suggests the key to the whole problem, so that instead o these cases some special form of symptomatic neurosis we should s merely the display of the classical hysteria, plus the effects of or ease.

As far back as 1859 Briquet, in describing hysteria in the male seven cases, of which four were in workers in lead. He seems to h nized the full significance of this apparent coincidence. Later th analogy of saturnine anæsthesia and hysterical anæsthesia did n French writers, most of whom attempted to explain it away and t tiate the one from the other. The discovery, first published by La Oulmont, that these saturnine anæsthesias could be transferred or e by a magnet, first marked incontestably the hysterical character o least, of them. Debove and Achard first suggested for this class *toxic hysteria,* which has full recognition now in France. Guinon th lead can be placed in the front rank of the causes of hysteria. In try this subject has been little, if at all, investigated, all the phen disease shown by workers in lead being attributed indiscriminat organic changes wrought by the poison.

Mercury has a power, similar to that of alcohol and lead, to exci ical symptoms. Letulle[2] was the first to establish this fact sati f false teeth made by an inexperienced dentist, which caused irritation er gums. She was told by a throat specialist that she was seriously poi- ed with mercury, which he asserted had been absorbed from the vermilion the vulcanite plate of the set of teeth. The patient was much alarmed this statement, and developed at once a variety of purely psychic and sterical symptoms, among them a marked tremor of the facial and some er muscles. She had no symptoms of genuine mercurial poisoning; but, s was just as potent, she believed that she had.

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[1] Gilles de la Tourette, op. cit., p. 101.

[2] Letulle: De l'Hystérie mercurielle (Soc. Méd. des Hôp. de Paris. 12 août, 18

ther poisons, such as morphine, absinthe, and tobacco, have the hystero- nous quality. In fact, there is no reason why any poison could not excite sterical symptoms in persons who are predisposed. It is probable that the eral lowering of nerve tone and of resisting power, which are the results the long-continued use of these poisons, are factors, as well as the mental pressions before referred to.

Hysteria can occur in either sex, at any age, and in every race and coun- y. The assertions made by some writers, notably English and German, at hysteria does not occur in their respective countries in all its classical ses, are erroneous, and are disproved constantly by other writers among eir own countrymen, who occasionally report typical cases; and by ob- rvers in America, who not infrequently find typical cases also in English d German emigrants. Such assertions, therefore, are evidences either defective observation or of a prejudice respecting hysteria that is not re enlightened than the ancient belief that the disease has its seat in the mb, and is confined, therefore, to the female sex. During the past year ce I have seen some of the worst forms of hysteria in young English and rman women. One English girl could be hypnotized readily; she had e grand attacks, just as they are seen in la Salpêtrière, with many interparox- al stigmata. One young German woman had astasia-abasia; her case is orted in this chapter further on.

The recognition of hysteria in children, and especially in boys, has been y slow and almost entirely modern. Le Pois was probably the first note the occurrence. Whyte,[2] in his old book, gives a case of hysteria in oy. Briquet[3] made a statistical study of 87 cases, apparently all girls. ring recent years in France this aspect of hysteria, as all others, has been roughly explored.[4] Clopatt[5] has given the most complete statistics, which s follows:

	Girls.	*Boys.*
In early childhood	19	1
3 years	...	1
4 "	1	1
5 "	4	2
6 "	3	2
7 "	15	4
8 "	16	6
9 "	15	7
10 "	18	15
11 "	24	17
12 "	22	13
13 "	27	16
14 "	12	8
15 "	...	3
	176	96

According to the combined statistics of Landouzy, George Briquet, as given by the last author,[1] hysteria is most common the age of twenty years. Briquet's general conclusions, from statistical study, are that one-fifth of the cases in the female sex puberty; that rather more than one-third of the cases develop ages of fifteen and twenty years; that the frequency of hyste rapidly from twenty to twenty-five years; that it then remai until the age of forty years; and, finally, that the disease is ve forty years.

Batault[2] has made a similar statistical study of hysteria i found that the disease is most frequent between the ages of ten years, after which it decreases in a ratio very similar to that seen sex. Gilles de la Tourette thinks, however, that hysteria dev later in men than in women, and attributes this to the fact that and alcoholism, which are common causes in men, act so mu quently in adult life.

As to the comparative frequency of hysteria in the two se differ. Bodenstein, from the polyclinic of Eulenberg and Menc one case of hysteria in men to ten in women. On the other ha Charcot's clinic, was surprised to find by statistics that hysteri type was relatively more frequent in men than in women, but this applies particularly to the lower classes, among whom hysteria in much more prevalent than it is in the higher classes. According hysteria in the male is twice as frequent in the hospitals as is hy female—a fact that will surprise many in America, who still idea that hysteria is rare in men. I doubt not that carefully ma studies in the hospitals of this city (Philadelphia) would show a proportion of cases of the disease in men than is now suspected. A claim, this preponderance is due to the effects of trauma and alco this country, however, the tendency is to ignore traumatic hyst or to call it by other names. It is common, for instance, for hy mata to be overlooked entirely in surgical cases, and yet some interesting of these, as tremor and anæsthesia, may occur. I ha extensive hysterical anæsthesia involving the upper arm, the shou large part of the trunk in a case of injury to the elbow in a youn

Hysteria, finally, has its *habitat* in every race and nation. It is to claim, especially in England and Germany, that racial characte to modify or to give complexion to hysteria as it appears in som ticularly in those countries. This is not more probable of this dis is of insanity, or of epilepsy, or of any other grave nervous dis conditions that predispose to hysteria and the causes that exci already been detailed; these are not peculiar to any nation, nor is any race exempt from them. It is only when from some particular social or political circumstances some particular nation or race is exposed to an unusual extent to the exciting causes of the disease that we see a distinct ethnic predominance of hysteria. This is probably the explanation of the fact that hysteria is prevalent among the Jews. This is so in Europe in contrast to this country, and this very contrast proves that the prevalence is due rather to social than to distinctly racial causes. The position of the Hebrew race in Europe at the present time is a very peculiar one. The Jews have been slowly appropriating, in fact, almost monopolizing, in some countries, as Germany, the professional and more intellectual pursuits, such as medicine, law, literature, journalism, and banking. With this gain in influence and riches there has been no corresponding gain in social *caste*, but, on the contrary, of late years the Jews have met a bitter and increasing Anti-Semitic crusade. These circumstances have developed in the European Jew a highly wrought and highly strung nervous system, and with this has occurred in the race a most significant increase of the two diseases, insanity and hysteria, which, more than all others, are the products of the struggle for modern existence.[1] This instance of the Jews in Europe probably illustrates better than could any other the real racial elements in the development of hysteria; in their case the occurrence of the disease is not due to race, but rather to circumstances. In conclusion, it is enough to say that hysteria has been observed in every quarter of the globe, and in many barbarous peoples.[2]

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[1] Op. cit., p. 73.

[2] Contribution a l'Étude de l'Hystérie chez l'homme, Paris, 1885.

Symptoms. The symptoms of hysteria are divided naturally into two classes—the paroxysmal and the inter-paroxysmal. Of these, the former are the most conspicuous, but the latter are often the most important. The former are transient, and hence are not often available for study, while the latter are more or less permanent, and hence are more available for diagnosis. The distinction in these two classes, therefore, is a most important one, especially for students of the disease in America, where, just as in England, the systematic study of hysteria has not been customary or popular.[3] In accord, however, with the plan and scope of this chapter, which is to make a strictly systematic presentation of this subject, this distinction will be observed in description, just as it is observed by nature in the disease itself. In this connection I cannot omit to emphasize the statement once more that hysteria is a morbid entity, and not a mere conglomerate of fantastic symptoms; that, in a word, it is a disease with a natural history and a definite symptomatology, and that pervading and controlling all its manifestations are definite and recognizable laws.

THE PAROXYSM. The paroxysm of hysteria has been called unfortunately hystero-epilepsy. This is a misnomer. The term seems to convey the idea that the paroxysm is a mixture of the two diseases, hysteria and epilepsy, whereas in truth there is no such commingling. The paroxysm, on the contrary, is hysterical, pure and simple. It is unfortunate, moreover, because the paroxysms of hysteria and epilepsy may occur in the same patient; but when they do so occur they are always distinct both in time and in character. This form, in which the two diseases coexist in the same patient, has been called by the French hystero-epilepsy, with separate crises. But this does not avoid the error involved in a joint term. It is claimed by some that a high grade of paroxysmal hysteria may merge into or develop epilepsy, but this statement

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[1] See a most interesting paper, "The Anti-Semitic Movement," by Sidney Whitman, in the Contemporary Review, May, 1893.

[2] Tourette gives instances of the occurrence of hysterical symptoms in the lower animals. There is nothing inherently improbable in the claim that such phenomena occur.

[3] Exception to this statement must be made in favor of the exhaustive paper on Hysteria by Dr. Charles K. Mills, in the American System of Medicine, Philadelphia, 1886.

is without adequate foundation. Once hysteria, always hyst
other hand, epileptics may exhibit both the paroxysmal and th
ysmal symptoms of hysteria, of which the epilepsy is an exciti
as other grave diseases may excite hysteria, as already shown. I
however, the diseases have separate crises. From all this it
there is no true hystero-epilepsy, but only a paroxysmal form of
would be as appropriate to speak of an hystero-tetanus or of a te

The best term, therefore, by which to designate the paroxy
hysterical convulsion, but if a more striking and distinctive ter
may be called, after Charcot, the grand hysteria.

The hysterical convulsion has been divided by Charcot, Ric
followers into four periods. For this division they have been cr
little by those who profess not to be able to see in hysteria a c
homogeneous disease. I am convinced that this criticism is not
conscientious and intelligent observation; or else that it is bia
dice against hysteria. Probably no modern instance of fine anal
in the observation and description of disease can be named that
the exhibition of the exercise of this faculty by Charcot in his o
of the hysterical fit. He has the supreme merit of having
type; and this type will doubtless endure and be recognized in a
medical literature. It has the great advantage of combining
features in one easily recognized picture, and of establishing a
which can be studied and contrasted all forms of the grand neur
atypical and diverse. It will be impossible, henceforth, for any
this convulsion with profit without reference to this type of Ch
is safe to predict that each succeeding observation by scientists
future, as in the past, confirm the general accuracy of the ico
the great French neurologist. Diversities and departures from
form doubtless occur, just as he pointed out, but they, like all
the evolution of natural forms, are best, and in fact alone, accou
reference to the type. The recognition of this fact is of great se
clinician in diagnosing some of the more obscure forms of convulsi

The paroxysm is invariably preceded by prodromes. It never
that of epilepsy, suddenly and almost without warning. These
are easily recognized by experienced observers, and even by the
or herself. Thus Richer gives the case of a woman who thou
cured, but who after a long interval of exemption recognized th
of a seizure by the appearance, several days before the onset,
mental and physical prodromes. It is not uncommon in hospital
and attendants to acquire this knowledge and to predict a convul

The first prodromes are mental, and they may appear some days
convulsion. The patient presents a change of character, or rathe
She may recognize this herself. She is no longer able to apply he
accustomed avocations; she cannot concentrate her mind; hence
read or sew or attend as usual to household duties. It is observed
that the patient is abstracted, self-concentrated, depressed, or abso
flections. She shuns the society of others, and if approached m
table, repellant, and not inclined to make confidences. The
indicate indubitably the psychical essence of hysteria; it is a dis
turbed cerebration. Later, as the climax approaches, a mo
emotional element appears: the patient's affective nature is more
easily disturbed and thrown out of equilibrium. Tears and la
excited readily and alternate without apparent cause; in fact,
emotional balance may usher in the fit. Hallucinations, delusi
and disturbed dreams are common during this prodromal perio

lady under my care had as hysterical prodromes nightmares and dreams of her scalp falling off, and these accompanied headaches, which persisted during the day. In accord with the psychic disorder are the neglect of dress and disregard of the proprieties which now appear; and a change of facial expression, which is quite characteristic in some patients. Sometimes the mental agitation is so pronounced that it becomes maniacal in character; the patient then shows incessant motor activity, paces the floor, gesticulates, utters incoherent and exclamatory phrases, and seems to be in acute mental distress. In other cases, again, the psychosis has a melancholic tinge; the patient broods, and is sluggish in both speech and movement. In all that the patient does and says in either case the disturbed emotions and the lowered volitional control are the conspicuous psychical phenomena.

Very common prodromes of the grand attack are disorders of the digestive apparatus; among these are loss of appetite, perversions of taste, and vomiting. Many patients have a peculiar spasm of the throat muscles, and perhaps of those also of the œsophagus, which produces a sense of suffocation. This was conspicuously described by the ancients. Many or all of these symptoms may also appear as inter-paroxysmal stigmata of hysteria, and will be described more in detail later.

The same may be said of most or all of the sensory and motor prodromes described in detail by Richer. These consists of various forms of anæsthesia, hyperæsthesia, paralysis, and contracture, which may appear in some patients as prodromes of the fit. They are properly, however, intervallary stigmata, and as such will be described in their appropriate place. It may be emphasized in this connection that the hysterical convulsion is often the occasion or the starting point for the display of some of the most persistent of the permanent stigmata; these may show themselves occasionally as prodromes, being excited by the peculiar psychical state that culminates in a paroxysm. After the paroxysm they may survive, and even persist for indefinite periods. We see something analogous to this in epilepsy in the occurrence of characteristic psychical states before the explosion, and in the persistence of them for some hours or days after the fit.

The hysteric, like the epileptic, fit is ushered in by an aura. This aura in reality may be classed among the prodromes. It has this distinctive mark, that whereas the ordinary prodrome may appear several days before the paroxysm, the aura immediately precedes the paroxysm, and in a sense may even be considered a part of it. The most common auræ are the *globus*, the *clavus*, and the ovarian hyperæsthesia. The *globus hystericus* consists in the sense of a ball rising in the throat; it is attended with a sense of suffocation, and usually with palpitation of the heart. The *clavus* is a circumscribed pain in the head, of very limited extent, which has been likened to a pain such as would be produced by driving a nail into the part—hence the name. It may be accompanied with ringing and beating noises in the ears and with a feeling of being beaten on the temples with mallets. The most common auræ are those emanating from the ovaries. These may be spontaneous; they may even be preceded by a degree of tenderness on pressure that suggests peritonitis. These ovarian auræ doubtless had some influence in directing the attention of the ancients so exclusively to the womb. Other areas on the surface, almost exclusively on the trunk, are sometimes the starting points for auræ. These auræ may be spontaneous, or they may be excited by pressure. Hence these areas have been called *hysterogenous* zones, and will be described briefly later among the stigmata.

The four periods of the typical or classical hysterical convulsion as described by the French school are: (1) The epileptoid; (2) the period of grand movements; (3) the period of passionate attitudes; (4) the period of delirium. These are not equally developed in all cases; in fact, fro vation, I should say that atypical or abortive attacks are perha frequent in this country. It is necessary in description, however to the type, and this will be done, with the preliminary statem author's hospital services in this city have amply confirmed the the description of Charcot, and with the further statement that attacks will be studied with care later.

The first, or epileptoid, period of the hysterical fit resembles convulsion of true epilepsy. Like it, its first phase is one of to of the muscles. The arms and legs are usually extended, the han the trunk bent, usually in a mild degree of opisthotonos, the ey fixed in conjugate deviation, and the teeth set. The breath is a the pulse accelerated. The consciousness is obtunded and even lo the degree of unconsciousness is certainly not so profound as This is proved by the fact that pressure over the ovaries, or the a electric current, usually promptly arrests the fit. In falling, the rule does not injure himself, and the tongue is not bitten. The t ceeded by the clonic phase, in which the muscles of the face an thrown into rhythmical shock-like movements. These moveme identical with those of the true epileptic fit, although it is difficult in what respect they differ. They have not the same quality of i reflex activity, and of mechanical force that marks the discha lower centres, which constitutes true epilepsy. There is in hyster psychical element, which is not altogether wanting even in the n seizure. If consciousness is lost it is at least not very far below tl and so, if the clonic movements are involuntary, they are not so so that it is impossible to summon the will again to the contro This is seen in the prompt arrest of the fit by pressure over the o the same time special and general sensibility are profoundly affe patient sees and hears nothing, in spite of the assertions of som writers that such patients are always peeping out from under half- This statement is based probably on the fact that slight clonic n may be seen in the eyelids in some patients. General sensibility is that tactile and even painful impressions are not noted. The clo ments in some cases are localized, *i. e.*, they are confined to one n one muscle group, or to one side. During the movements the re are labored and embarrassed. The clonic phase is succeeded by and last phase, resolution. The clonic movements subside grad finally disappear. The patient lies relaxed and supine. She rec head to one side; saliva flows from the mouth. Consciousne obscured, and perhaps light sleep supervenes. There is no incon urine or feces. Occasional shocks may occur during this period, common for the eyelids to present slight oscillations.

After a short interval the second period begins. The second per grand attack is that of grand movements, or, as it has been called ism." It is characterized by violent and extravagant muscular m either in the nature of disordered contortions or of regulated and a purposive feats. Movements of this character have acquired gre nence in some of the epidemics of hysteria, as, for instance, the e St. Medard, in which, as Briquet says, the movements were so a and inexplicable that it was thought necessary to attribute them divine influence. By a reversal of the same logic, they were att some of the earlier epidemics, which contributed to the brutal supe witches, to the influence of the devil. All the dances, contortions, and bizarre and grotesque feats of dexterity which we read about old middle-age crazes, or which we see occasionally at present in the devotees of emotional religion, were and are instances of this "clownism," which constitutes the second period of the hysteric convulsion. These same phenomena are seen, moreover, in some of their most important aspects, when they constitute the atypical attacks which appear sometimes in isolated cases. They may then constitute the whole of the attack and undergo such an extraordinary development as to lose almost entirely their primary affinities, and may thus present difficult problems in diagnosis. Again, this second period may almost or entirely abort; in fact, in hysteric convulsions of a mild grade it is not uncommon in my experience to see it abort, or, at least, to assume a very minor part. The most common of these movements is the so-called arc of a circle, in which the patient assumes a position of opisthotonos, which may be complete or incomplete. The patient may assume this position lying either on her back or on her side. Other movements of extreme contortion, too numerous and varied to be detailed, may be shown. In contrast to these inco-ordinate, or, as the French say, *illogical*, movements, are others of a more regulated and complex kind. Richer has described and, with his own pencil illustrated, these movements. The most common is that called "salutation," in which the patient, lying on her back, suddenly bends the body forward until the head almost touches the knees; this is repeated many times. Others consist in bending and arching the body and flexing and extending the limbs in various ways, rapidly repeated, usually while the patient reclines on her bed. This extreme motor agitation seems to have some mental association, or to be at least the expression of a mental state.[1] In some cases this mental state appears to bystanders to be quite alarming, and the cases to have a veritable demoniac element in them. There is not loss of consciousness during this second period, for the patient seems to be reacting to some kind of a mental stimulus, but there is not the same association with delusions and hallucinations as appears in the next period.

The next, or third, period has been called by Charcot the period of passionate attitudes; but as these attitudes are but expressions of mental phenomena, it would be more appropriate to name this stage accordingly, and call it the emotional period. It has been customary for French writers to say that it is characterized by delusions. The objection to this is that it conveys the idea of an insane state, in which a delusion dominates the mind. No such domination by insane delusions can be demonstrated in hysteria, and, therefore, the term delusion in the English language conveys an erroneous meaning. The same may be said of the term "hallucinations," also used by French writers. It is doubtful if the hysteric in her most perverted stages has genuine hallucinations in the sense in which the term is used in English. With us it means a false sensory concept accepted by the patient as true. It is a symptom of profound alterations, such as characterize some of the more grave states of insanity. The hysterical patient scarcely has delusions and hallucinations in the sense, therefore, in which we use these terms for the insane. What she has can more properly be termed mental images or reveries, rising into consciousness, either as a cause or a consequence—more probably the former—of her disturbed emotional state. In the height of the paroxysm, amidst the favorable conditions of lowered volition or cerebral inhibition that characterize the psychical state in hysteria, the patient simply passes into a mêlée of her emotions, the most prominent of which are, doubtless, expressive of ideas or experiences, often painful, derived from her past life. Hence the third period of grand hysteria is essentially dramatic; hence, too, it is capable of almost indefinite expansion in the hands of expert scientists, who may be tempted, perhaps, too often, to play the rôle of stage-director. This explains why these exhibitions are not common in localities or countries in which the taste for them does not exist. The tendency to them is probably just as inherent in hysterics in one country as in another, having slight diversities in such minor national traits as the expression of motion, but the greatest distinction exists among races and nations, due to causes that need not be discussed here, in their appetence or relish for the melodramatic. Perhaps the exaggeration of development which this stage has experienced in France has done much to excite prejudice against and criticism of the disease. Its true importance, however, ought not to be ignored. From the purely pyschical standpoint, it is perhaps the most significant of all the stages of hysteria.

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[1] Sauvages used the term *hysteria libidinosa* for those cases in which during convulsion the patient alternates rapidly from opisthotonos to dorsal decubitus.

Little or no interval occurs between the motor excitement of the second period and the tableaux of the third. In fact, it is well to recall that these two periods merge so naturally the one into the other that many cases do not present the typical divisions into stages. These grand movements and these passionate attitudes are simply expressions of emotions, and as such may be blended, confused, or modified in various ways, according to the circumstances and the individuality of the patient. It is obviously impossible in a short space to recount or to classify all the varied modes of dramatic expression into which hysterical patients can throw themselves. They are as countless as are the shifting phases of their emotions, reacting to every stimulus of both memory and environment. Richer, indeed, has attempted such a classification, based upon cases observed by himself, which cases had evidently been subjected to the more or less unintentional hypnotic suggestions which characterize very plainly the work of the French school. Hence it is by no means sure that his observations on the third stage of the classical attack will be confirmed by all other observers in all other countries. These manifestations will doubtless take form and complexion from the personnel of both patient and audience. This is no reflection, however, upon the scientific accuracy and value of the writings and drawings of this gifted French author. Hysteria is a disease of suggestibilities: its possibilities are countless. The varieties of passionate attitudes, as given and depicted by Richer, are as follows: the attitude of the cross, of defense, of menace, of appeal, of lubricity, of ecstasy, of dread of animals, such as rats, etc.; of listening to music, of scorn, and, finally, of lamentations. He even attempts to allot the time in seconds which each scene of this drama consumes. Thus the attitude of the cross requires twenty-three seconds; appeal, ten seconds; lubricity, fourteen seconds, etc. This is obviously an excess of refinement in description. It is sufficient for our purposes here to state merely that the third period of the hysterical convulsion is one of dramatic representation of emotional images, and that these are of countless variety, according to time and person. They are apt to preserve curiously the same physiognomy always in the same case. In other words, peculiarities of words and expressions and attitudes are repeated by the patient in successive fits. They are stereotyped, as Richer says. It is important, finally, to indicate that some particular phase of this period, as ecstasy, for instance, may acquire in some cases exceptional conspicuousness and persist as an isolated phenomenon, thus constituting an atypical form of grand hysteria. This fact will be alluded to again.

During the third period sensory stigmata of a high grade are present. The patient does not feel tactile and painful impressions, the conjunctivæ are insensate, the hearing is lost for even loud sounds, and the patient is oblivious of all her surroundings. Pressure over the ovaries and the use of electricity are said, however, always to abort the attack. After the attack the patient retains a memory of the events of this period.

The fourth period of the attack has been called the period of delirium. Here, again, some slight criticism of terms is called for. The terminal stage of the grand attack of hysteria is not marked by a delirium as intense, or even of just the same nature, as that which characterizes the febrile diseases, and which is suggested by this term in the English language. It is rather a paroxysm of emotional disturbance, continuous with that of the third period, but not marked by the active motor expressions of that period, which seem now to have exhausted themselves. The tinge of this emotion is usually one of sadness, and, just as in the former period, it feeds upon painful ideas which are gathered from the memory of the past life. Sometimes there are noisy weeping and lamentation, and instead of acting, as in the third period, the patient now declaims. Richer makes of this a vital distinction between the two periods: the former is one of acting the latter one of speech. This distinction, however, is likely to be lost in many cases; the characteristics of the two periods may blend in cases departing slightly from the type. In the fourth period consciousness is returning or has returned; hence the patient is *en rapport* with her surroundings. During this period the sensory stigmata which were seen in the third period may persist, but added to them may be motor stigmata, which were quite absent before. Among these the most common are various forms of contracture; these may persist for many days after the seizure. Other motor stigmata, sometimes originating in this period, and persisting as relics of the convulsion, are the several varieties of paralysis and tremor. The delirium, finally, may merge into an obstinate mutism, or even into more rare complications, such as trance and lethargy.

The hysterical convulsion, such as we have now described, lasts from one-quarter to one-half an hour; this is exclusive of the "delirium" of the fourth period, which may be prolonged to several hours, or even to a day. In very rare cases the convulsions may be repeated at short intervals, the patient not regaining her self-possession between the attacks; thus there may be a veritable hysterical eclamptic *status*, analogous to the epileptic status. This may continue for a day or even more, the patient having as many as fifty or even a hundred spasms. When this status occurs the fourth period is entirely omitted until the end of the series, and the phenomena of the other periods are much blended and confused.

As shown by Richer's historical study the grand attack of hysteria is not confined to any race or nation. Conclusive evidence of it is found in the older literature. It exists, however, in many varieties, and in not a few abortive and atypical forms, which will now be examined into briefly.

The varieties of the hysterical paroxysm can be shown to be, without exception, modifications of the type already described. They are abortive attacks, in which the phenomena of one period alone, sometimes curtailed, sometimes intensified, are seen; or they are combinations of two or more periods, or, finally, they merge into special or terminal stages, such as somnambulism, catalepsy, and trance, which seem, at first sight, to be epiphenomena, but which, on closer examination, are seen to have a strictly logical connection with the grand attack. Briquet, among the first, described clearly the varieties of hysterical convulsions, including catalepsy and trance, but Charcot was the first to demonstrate, as has already been shown, the laws of development of these varieties from one classical type. It can readily be appreciated how great the number of these varieties may be, and how diverse and unique may be their symptoms, when it is recalled that these four periods present many minor phases, and hence may make innumerable combinations.

whom there is no evidence of disease, even of hysteria. The can be called a patient, remembers nothing of the attack, which ied to dreaming, for in it the patient simply reacts to a dream- This is entirely distinct from, first, the automatism of epilepsy, , the pseudo-somnambulism of hysteria. These latter two, again, ng in common.[1] The somnambulism of hysteria is not a disorder ut simply a transformation of one of the phases of the third period. ramatic representation of an emotional state. In it the patient blivious to sensory impulses, but she retains a distinct memory of k after it has passed. Moreover, the attack may be preceded by hases of grand hysteria, such as prodromes, epileptoid symptoms, etc. . the patient usually presents intervallary stigmata. We see from all at the condition is more closely allied to ecstasy, just described, than so mnambulism. In other words, it is purely hysterical. Its nature is ized by the fact likewise that the pseudo-somnambulism of hysteria induced by hypnotism.

talepsy, like somnambulism, has several varieties, or, rather, the term is for a variety of mental states. It is a condition of both mental and r inertia, the patient's thoughts, apparently, as well as his limbs, tending main in the state or position in which they are placed. Hence, in some it is a natural and appropriate manifestation of the hysteric paroxysm. again, the psychic element is marked; the patient's thoughts, immobile unyielding, dominate the motor sphere. The limbs retain, for incredibly periods, a statuesque rigidity, or yield slowly, with wax-like stiffness, to influence of gentle pressure. The patient is apparently insensible, or t insensible, to external impression; the countenance is set and unintel- t; the somatic functions are sluggish. The relation of catalepsy to ria is found in the era of attitudinizing, which is named the third period. lation with this period is logical and intelligible. It may present itself phase of the grand attack, many of the precedent features of which may been lacking, and gradually assume an isolated and exaggerated rôle; may appear as a strictly isolated phenomenon from the first. In the ric it is probably not as profound a state as it is in some forms of ity, for it can often be induced by hypnotism, and it can be dispelled ressure over the ovaries or by the action of an electric current. It is, It will be possible here merely to indicate some of the most common and most important of these.

One of the most common of hysterical paroxsyms is really an abortive attack in which the prodromes are the conspicuous features, and in which few, if any, of the convulsive symptoms appear. The patient, after presenting for some hours or days some of the mental prodromes already described, such as change of moods, depression, irritability, and fickleness, has an emotional outburst of alternate tears and laughter, to be followed by a partially-developed tetanoid and eclamptic period, ending with mutism and sleep. This has been called hysteria *minor*, in contrast with the grand attack. It is the ordinary, or vulgar, hysterical fit. It may be associated with or followed by some of the physical stigmata, such as paralysis and contracture. In others of these minor attacks the period of passionate attitudes, or dramatic representation, may be partly developed, and sometimes proving, as Bernutz[1] has claimed, that in some cases there is but a short step from these phenomena to a more or less prolonged access of delirium, of somnambulism, of ecstasy, or of catalepsy. These grave terminal stages, following a brief or abortive paroxysmal seizure, are more usual in children.

Among the most remarkable of the aberrent forms of grand hysteria is that condition known as "chorea major." This term originated in Germany, and is open to the same objections as those urged against the term "hystero-epilepsy." It is borrowed from another and distinct disease, which has no connection with hysteria. The so-called chorea major is in no sense choreic. It is purely an hysterical affection, and finds its proper place in nosology by reference to Charcot's classical type of hysterical convulsions, by virtue of which, indeed, it has been rescued from the anomalous and false position in which it was formerly left. The term, indeed, is very vague and inexact, and probably has been made to include more than one variety of nervous affection, but if we observe attentively[2] we can see that most of the cases reported belong to the second and third periods of the grand attack of hysteria. They are characterized by strange exaggerations or grotesque modifications of the grand movements and the passionate attitudes of the hysterical convulsion. In children examples of isolated movements, feats of automatism, mimicry, acquired dexterity, and semi-purposive contortions occur which are doubtless abortive or atypical hysterical seizures. They may be preceded sometimes by an epileptoid phase. Thus, in a boy, whose case was reported by the writer, rotatory and gyratory movements followed an aura and epileptoid stage. One such case may cause many others in schools, by imitation; the epidemic assuming astonishing and bizarre features. Such epidemics, under religious excitement, occurred during the middle ages and later. Charcot gives to these and somewhat similar phenomena the term "clownism."

Ecstasy is a rather rare phenomenon, sometimes observed as an isolated state, which in reality is but an exaggeration of one of the phases of the third period of hysteria major. It is an exaltation of certain ideas in which the patient becomes so absorbed that all sensory impressions are unrecognized and all, or almost all, motor functions are in suspense. Occasionally, however, the ecstatic declaims and even sings. The state has been described by Michéa.[3] There are varieties of this peculiar mental state, and some cases that have been described as ecstatic were possibly not truly hysterical; but the most common form is a variety of one of the passionate attitudes of the grand attack. Here we see the patient *en rapport* with a train of mental images even to a more profound extent than in the other phases. Stigmata would probably be found in most patients during the intervals between the attacks.

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[1] Art. Hystérie. Nouv. dictionnaire de Méd. et de Chirurgie, Paris, 1874, t. xviii, p. 221.
[2] Eulenberg's Art. Ziemssen's Encyc.
[3] Nouv. Dict. de Méd. et de Chir. Prat. Art. Extase.

What has been said of ecstasy may also be said of somnambulism, catalepsy, trance, and lethargy, which sometimes complicate or terminate the hysterical paroxysm. They are modifications of the mental state of the hysterical subject. But as their relations are complex they must be investigated in some detail.

Somnambulism is another of those terms that have been used for hysterical symptoms without proper definition. Like "chorea major," it is a misnomer, and has been borrowed from a condition which has no connection whatever with hysteria. Somnambulism proper is a disorder of sleep, occurring mostly in children in whom there is no evidence of disease, even of hysteria. The patient, if he can be called a patient, remembers nothing of the attack, which is closely allied to dreaming, for in it the patient simply reacts to a dream-like state. This is entirely distinct from, first, the automatism of epilepsy, and, second, the pseudo-somnambulism of hysteria. These latter two, again, have nothing in common.[1] The somnambulism of hysteria is not a disorder of sleep, but simply a transformation of one of the phases of the third period. It is a dramatic representation of an emotional state. In it the patient may be oblivious to sensory impulses, but she retains a distinct memory of the attack after it has passed. Moreover, the attack may be preceded by other phases of grand hysteria, such as prodromes, epileptoid symptoms, etc. Finally, the patient usually presents intervallary stigmata. We see from all this, that the condition is more closely allied to ecstasy, just described, than to true somnambulism. In other words, it is purely hysterical. Its nature is emphasized by the fact likewise that the pseudo-somnambulism of hysteria can be induced by hypnotism.

Catalepsy, like somnambulism, has several varieties, or, rather, the term is used for a variety of mental states. It is a condition of both mental and motor inertia, the patient's thoughts, apparently, as well as his limbs, tending to remain in the state or position in which they are placed. Hence, in some cases it is a natural and appropriate manifestation of the hysteric paroxysm. Here, again, the psychic element is marked; the patient's thoughts, immobile and unyielding, dominate the motor sphere. The limbs retain, for incredibly long periods, a statuesque rigidity, or yield slowly, with wax-like stiffness, to the influence of gentle pressure. The patient is apparently insensible, or almost insensible, to external impression; the countenance is set and unintelligent; the somatic functions are sluggish. The relation of catalepsy to hysteria is found in the era of attitudinizing, which is named the third period. Its relation with this period is logical and intelligible. It may present itself as a phase of the grand attack, many of the precedent features of which may have been lacking, and gradually assume an isolated and exaggerated rôle; or it may appear as a strictly isolated phenomenon from the first. In the hysteric it is probably not as profound a state as it is in some forms of insanity, for it can often be induced by hypnotism, and it can be dispelled by pressure over the ovaries or by the action of an electric current. It is, however, in both conditions a psychosis favorable for the manifestation of the complex and unhealthful processes that characterize the functions of the brain in both states.

Lethargy, unlike catalepsy, is not marked by motor rigidity, unless it be occasionally a contracture of a limb. But this renders the limb stiff and unyielding, differing from the "lead-pipe" flexibility of catalepsy. In lethargy

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[1] The French, who are largely responsible for this misuse of the term somnambulism, have lately seen the necessity for the differentiation observed in the text. See "Les Somnambulisms" by Blocq. Cliniq. d. Mal. du Syst. Nerv., Guinon, 1893.

the patient lies in a semi-stupor, with closed eyes. The eyelids
a fine fibrillary tremor. This state may endure for hours. It
the grand attack, and may be an isolated and abortive form;
some cases a terminal stage.

Trance is an intensification, as it were, of lethargy. It ma
many days or weeks. It has supplied in the past many nota
patients who have lain profoundly dormant for long periods, t
ment of the curious and superstitious, and who have recovere
amidst emotional and devotional rites, to the edification of
Trance, in its profound states, presents many problems of physiol
est, which cannot be discussed here. It is best understood
thrown upon it and kindred states by a study of the grand hys
the patient has suspended cerebration, to the extent, at least, th
functions are exercised to the most limited extent compatible
sary continuance of some physiological activity. Coma, which
said to occur in hysteria, is probably only a modified form of tr
also, hysterical sleep and mutism. Attentive observation and
have demonstrated, as in the Scotch case published some years ag
in the most profound trance the patient's consciousness of the env
not entirely lost. It must not be forgotten that some cases o
probably cases of melancholia attonita.

Finally, hysterical sleep may be considered in this connection.
Halle, has reported recently a most instructive case of this slee
serve for a description. A laborer, aged twenty years, received se
on his head and arm by an accident while at his work.[3] A few
he began to have sleeping spells, which were as follows: They
intervals of from seven to thirteen days, and lasted from thirty-fou
six hours, once forty-one hours. During the spell the patient usua
quietly on his back; sometimes, however, he tossed about. The
not quiver. Contraction of the masseters was not present. The e
directed upward, and when the lids were kept open for a time t
moved slowly to and fro. Temperature, pulse, and respiration di
any considerable changes. The patient could be aroused by pres
of the scars, or on one "ovarian" region. When roused thus he
water and drank; he did not eat. During the sleep the weight o
the volume and specific gravity of the urine, and the quantity
decreased. After the sleep the patient had a violent headache;
ever, he began to eat freely, and all functions were quickly rest
prodromes consisted of change of disposition, etc. He was readily
by hypnotic suggestion, and, practically, was cured by it. This
sleep is possibly identical with lethargy, already considered. Hitzi
that the spell in his case was an abortive form of the classical
epileptic" attack.

This may end the consideration of the varieties of the grand
hysteria. As said before, these varieties, consisting of combinati
phases of the classical attack, as well as abortive and atypical s
much more numerous than there is space to describe them in. Onl
important of the class have been considered here.

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[1] Cases of trance have lost nothing in the telling. Thus, such a case is said to hav
the anatomist Vasalius, whose "subject" was aroused by the first stroke of the knife.
similar case was that of Lady Russell, who returned to consciousness at her own fune

[2] Brain: Spring and Summer, November, 1893.

[3] The traumatic origin of this case is most significant in its bearings upon the vexed q
"traumatic neuroses." Hitzig has some judicious remarks upon this question; he do
with approval the views of those who affect to ignore hysteria and hypochondria
origin.

According to the researches of Gilles de la Tourette and Cathelineau,[1] the nutrition of the hysterical patient is affected during the grand convulsive attack, but not during the intervals between the attacks. In Hitzig's case of hysterical lethargy[2] there was a loss of weight before and during the sleep of so much as seven kilogrammes. At the setting in of the prodromes there was increased excretion of urea, the patient not eating as much as usual and losing weight, the augmented excretion of urea being accounted for by the loss of weight. During the sleep the excretion of urea diminished. The urine was diminished during the sleep. For more details the original papers may be consulted. It is significant that the nutrition of the lethargic patient corresponded closely with that of patients in grand hysteria, tending to prove that lethargy is but a modification of the grand attack.

THE INTERPAROXYSMAL SYMPTOMS. The interparoxsmal symptoms of hysteria have been called with good reason the *stigmata*. They are the marks of the disease, and by this highly-suggestive name they seem to indicate how persistent and ineradicable they are in many cases. They are by far the most characteristic and most important of the symptoms of hysteria, because, first, they are the most prevalent, and, second, they are the most available and valuable for scientific purposes. Without them, indeed, few cases of hysteria can be differentiated successfully, and with them no cases can be mistaken. It is too much the custom to regard hysteria as a disease of motor explosions and of follies and affectations, but a study of the intervallary stigmata cannot fail to impress upon the mind that the paroxysmal features, while of supreme importance in their own sphere, are of but secondary importance to the permanent signs, and that instead of being a disease of simulation and conceit hysteria is one that is marked by persistent physical stigmata, which are quite beyond the control of the patient's will, and sometimes, indeed, are beyond the domain of his consciousness. So persistent are many of these signs that they are called, especially by the French, the *permanent* stigmata; but this term is open to the very obvious objection that, while these symptoms are likely to be obstinately fixed for long periods, they are not essentially permanent, because many, or all of them, may disappear in favorable cases. Hence, it is best to call them interparoxysmal or intervallary.

The interparoxysmal symptoms of hysteria may be divided into four classes: sensory, motor, visceral, and psychical.

SENSORY SYMPTOMS. The alterations of sensation in hysteria are of three kinds—anæsthesia, hyperæsthesia, and paræsthesia. Anæsthesia may be subdivided into alterations of the various modes of sensation, as, for instance, tactile anæsthesia, thermo-anæsthesia, analgesia, and loss of muscular sense.

The anæsthesias of hysteria played a conspicuous rôle for many centuries before they attracted the attention of scientific observers. As the "marks of the devil" (*stigmata diaboli*), they had been noted and described by churchmen as far back as the times of Tertullian, and they had served their purposes as the distinguishing marks of witches all through the middle ages.[3] It had been noted that witches and those possessed had areas of anæsthesia in which the prick of a needle was not felt and did not draw blood. In epidemic hysteria, excited by witchcraft crazes, the poor wretches who displayed these sensory stigmata were too often doomed. Most ingenious methods were taken by experts to detect these signs, which, far from being simulated, were disguised by the unhappy victims. In some cases, however, it is significant that the sorcerers had no consciousness, until they were detected, that they possessed these signs. How different all this appears from what would have been the case, if, as some modern writers state, these symptoms

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[1] La nutrition dans l'hystérie, Paris, 1890.

[2] Op. cit.

[3] Tourette: Op. cit.

are unreal. The anæsthesia of hysteria may occur in various
most common are hemianæsthesia, anæsthesia in plaques, and ana
limb, associated, or not, with paralysis of the limb. In some v
the anæsthesia is total.

Before studying these most important marks of hysteria it i
tion students against the too prevalent skepticism that exists
of anæsthesia in hysteria. Only a personal study of cases seen
pel this unbelief from some minds. The history of this aspect
most curious; for, in spite of the prominence of these stigmat
gious fanatics and so-called witches in past ages, the recognitio
scientists has been most tardy. In spite of the writings of Pio
and Briquet in France, and Szokalsky in Germany, all before 1
ject, as Pitres says,[1] seemed likely to be ignored again until
1872, emphasized its importance. It is sufficient to say that
anæsthesia is the most common of all the intervallary stigmata,
study of no case is complete without a careful search for these si

The hemianæsthesia of hysteria has some features that serve t
it. It is a very profound anæsthesia, and it is complete, that
from the crown of the head to the sole of the foot. Exceptio
they are exceptions. It is so profound, often, that painful impre
alter it, and it is often associated with some vasomotor change
stick does not bleed. This anæsthesia is not confined to the
volves even the subcutaneous structures, notably the nerve-tru
wounded the ulnar nerve at the elbow, so as to cause contraction
cles supplied by it, but failed to excite sensation. Occasionall
thesia is accompanied by a mottled and œdematous state of the
bleu). It is accompanied frequently by anæsthesia also of the m
brane of the eye, nose, mouth, rectum, urethra, and vagina. E
significant accompaniment is anæsthesia of the special senses.
patient does not hear, see, nor smell on the affected side, and th
is affected, usually unilaterally. Finally, this hemianæsthesia is
occasionally from one side to the other; but usually this tran
temporary, as the affection tends to resume its primary seat. T
occurs under the influence of emotion, or by suggestion, or by th
purely suggestive one), of certain æsthesiogenic agents. This p
of transfer is peculiar to the hemianæsthesia of hysteria, and dist
from hemianæsthesia of organic origin—a fact that must be bor
when studying the hemianæsthesia of chronic alcoholism and of l
ing (see *supra*). For some reason, not readily explained, the hem
of hysteria is much more frequent on the left side; in the pro
cording to Briquet,[2] of 7 to 2.

A very common form of anæsthesia in hysteria is that in which
sensation occurs in limited areas of various sizes, shapes, and locati
assume curious geometric forms. (Figs. 26 and 27.) These are
thesia may change from time to time; in fact, it is not usual to
the same upon succeeding days. The patient is ignorant, frequent
is thus marked. They are not the products of suggestion, because
may be demonstrated on patients who have never before been s
the test and who are ignorant of its object.

One of the most characteristic forms of anæsthesia in hysteria
mental form, or that in which the anæsthesia is limited to one
part of a limb. It may take the position of a gauntlet or of a st
is often the accompaniment of hysterical paralysis and contra

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[1] Des anæsthesies hystériques, Bordeaux, 1887.

[2] Op. ci

FIG. 26.

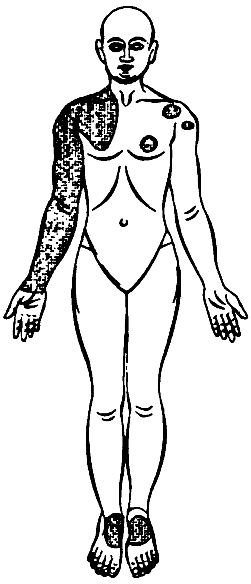


FIG. 27.

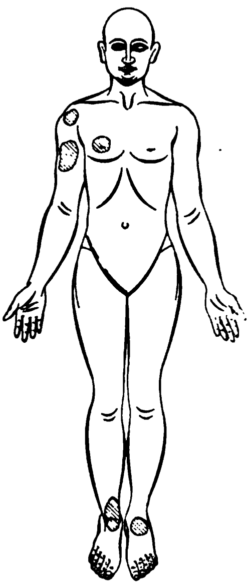
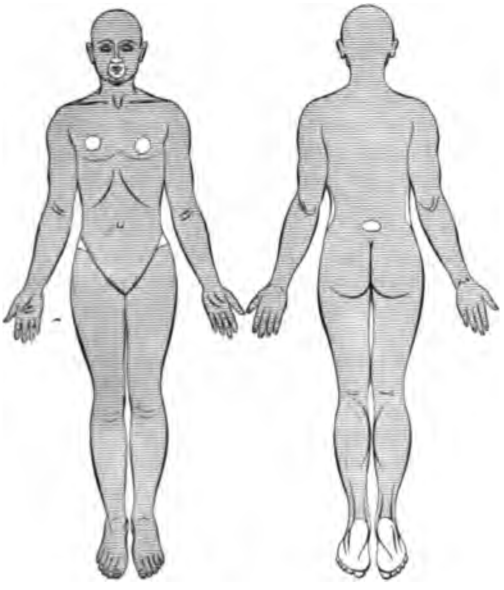


FIG. 26. Irregularly distributed ▦ *Tactile Anaesthesia and Analgesia* from a case of hysteria at the Home for Crippled Children, Philadelphia.

FIG. 27. Areas of ▨ *Thermo-Anaesthesia* in the same case.

arm or leg, and in this association is very characteristic, because no organic lesion can give just this symptom-complex. This anæsthesia is not limited

FIG. 28.



Almost complete ▤ *Anaesthesia* from author's case of hysterical astasia-abasia, Methodist Hospital, Philadelphia.

to the distribution of any one or more nerves; in this respect indicating its cortical or psychical origin. It is usually sharply delimited, the line of demarkation being at right angles to the long diameter of the limb.

Finally, very rarely, anæsthesia may be distributed universally. In some cases the anæsthesia is *almost* universal, the exceptions occurring in small islets in which sensation is normal. A diagram of such a case is shown in Fig. 28, from the author's clinic at the Methodist Episcopal Hospital. The anæsthesia is general, with the exceptions noted.

According to Pitres[1] the anæsthesia of hysteria is never isolated tactile anæsthesia; in other words, one or more of the other forms of anæsthesia is always present. The most common of these forms in hysterical anæsthesia, as given by Pitres, are: first, total anæsthesia, *i. e.*, in all modes; second, partial anæsthesia, of which the most common are analgesia, thermo-anæsthesia, combined tactile and thermo-anæsthesia, electro-anæsthesia, and loss of all modes except electro-æsthesia.

All forms of hysterical anæsthesia are likely to be worse after a fit. They may persist, however, as intervallary stigmata without the patient's knowledge. Hence, the patient's own statements about his or her ability to feel must not be accepted, if a critical examination is to be made; but he or she must be stripped and thoroughly tested. Finally, anæsthesia, while very common, is not found in all cases; hence, its absence is not decisive against a suspected case of hysteria. Briquet found anæsthesia in 240 of 400 cases examined by him with great care[2]—that is, in sixty per cent. It is probable that this percentage is too small; certainly, in my own experience anæsthesia in some of its forms is more frequent than this.

The hyperæsthesias of hysteria form an important group, especially because this group contains the hysterogenous zones. It contains, also, the almost equally important sub-group of hyperalgesias.

The hysterogenous zones may be defined briefly as localized areas of extreme sensitiveness, pressure on which has the power of provoking some of the hysterical manifestations, and especially the convulsive phenomena. The location of these zones, or areas, varies in different persons, but the most common are over the ovaries, along the spine, on the breasts, and on the trunk beneath the ribs. They have been found on the vertex. *Per contra*, pressure on these areas has the effect of arresting the convulsion even when at its height. The latter fact was observed and utilized by empirics long since. The convulsionnaires of St. Medard, for instance, were influenced thus by strong pressure upon the abdomen. The number of these zones vary; some patients have but one, others have several. In patients with hemianæsthesia they may be unilateral, and are then confined to the anæsthetic side. They are frequently the seats of spontaneous pain during the prodromal period, and may then simulate other diseases. For instance, I recently observed a woman whose severe ovarian pain simulated a local inflammation; possibly a peritonitis; but who, as events proved, was in the prodromal stage of hysteria major.[3] The purely hysterical character of such neuralgic pains is demonstrated by the fact that they disappear entirely with the cure of hysteria by some of the well-recognized means.

The hyperalgesia of hysteria may simulate, in the most puzzling way, organic disease. It simulates especially disease of the joints. This is one of

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[1] Leçons Cliniques sur l'Hystérie et l'Hypnotism, Paris, 1891.

[2] Op. cit., p. 278.

[3] This ovarian, and sometimes uterine and general vaginal, hyperæsthesia is doubtless the cause and the excuse for some useless gynecological surgery during the present epoch. A more systematic study of hysteria ought to demonstrate that it is a psycho-neurosis and not a genital affection. It ought not to be necessary to remove a woman's ovaries in order to cure her by suggestion.

the most common forms of neuro-mimesis, and was so prevalent in Brodie's time that he said that four-fifths of his cases of joint-disease were hysterical, a proportion far in excess of what can be observed at the present time, in spite of the alleged tendency now to over-describe hysteria. This nervous mimicry of organic disease of the joints is one of the most striking proofs of the fact that hysteria is a disease of perverted and fixed ideas. These are the cases that recover in an instant under the care of a charlatan or of a priest. Hysterical disease of the hip or knee-joint is not associated with deformity and shortening of bone, nor with the formation of pus, nor with the local rigidity, nor with the septic temperature that is seen in tuberculous diseases. The stiffness is caused by contracture of the muscles, which is usually much more extensive than in organic disease; and the pain is usually more diffuse and more spontaneous. There are, moreover, characteristic mental and physical stigmata. The hysterical patient dreads to move or to assist in the examination, and obviously dwells with exaggeration upon each symptom; while she is very apt to have segmental anæsthesia in the affected limb, or even hemianæsthesia, as in the case reported elsewhere. A very significant symptom is paralysis of the limb, which is never present in hip-joint disease. Finally, under full etherization, the hysterical joint is found to be freely movable in all directions. It must not be forgotten that hysterical symptoms may be *added* to those of genuine organic disease of the hip or knee, just as hysteria may be caused by any other severe disease, as already indicated.

Finally, hyperalgesia may exist as various forms of neuralgic pain. After traumata it is common to have such pains, especially about the occiput, the back of the neck, and along the spine. A common form of hysterical neuralgia is the clavus, already described as among the prodromes. A very obstinate pain is fixed sometimes in the female breast, which may even become slightly swollen and exquisitely painful to touch. It is called mastodynia. In women with the hysterical breast the mental suffering is often very great, the patient's mind dwelling constantly on her ailment, and her imagination becoming the prey to fears of malignant disease. This breast occurs not infrequently in young women, before the usual age for cancerous disease. It sometimes follows a slight trauma. Other forms and locations of neuralgia may be observed, according to persons and circumstances.

The special senses are involved frequently in hysteria. The most important of these is sight, which may be totally abolished (amaurosis) or only partially obscured (amblyopia).[1]

Hysterical amaurosis, or blindness, is a rare affection, which usually occurs suddenly, and not infrequently disappears as suddenly. Thus, Pitres relates the case of a young girl, aged sixteen years, who after a slight illness became suddenly blind. At the same time she became analgesic over the whole surface of her body. She was cured after a few days with a few applications of electricity. Hysterical blindness, however, is not always of such short duration; it has been known to persist for months, and even for years. It is sometimes unilateral, and not infrequently is confused with malingering. Its exact nature, as in the case of so many hysterical phenomena, is difficult to explain, and has been the subject of much discussion. When subjected to crucial tests, as done by Pitres, these patients with unilateral amaurosis are found sometimes to see with both eyes, and yet clinically they act and are practically like persons blind in one eye. The explanation lies not in simulation, but probably in the fact that the affection is psychical and purely subjective.

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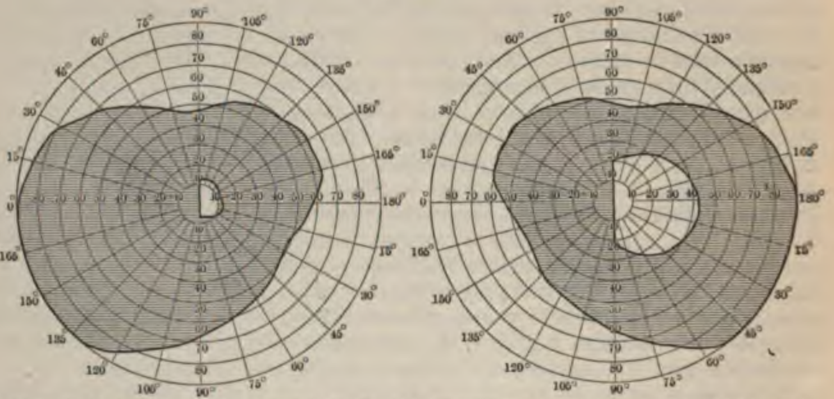
[1] Pansier: Les Manifestations Oculaires de l'Hystérie, Paris, 1892. This work contains a full bibliography.

Hysterical amblyopia, or partial blindness, may be divided into several varieties, to understand which it is necessary to understand the physiology of the visual field. When the normal eye is fixed upon a given point the extent of vision has definite limits in all directions about that fixed point. This extent of vision about a fixed point is called the visual field. Supposing this point to be the approximate centre of the field, the extent of vision upward will be about 65°; outward, toward the temporal side, about 90°; downward, about 75°; and inward, toward the nasal side, about 55°. These limits are nearly constant for all normal eyes. The visual field is affected in hysteria in three ways. There may be, according to Pitres, a central area in which nothing is seen, while all around the periphery sight is retained. This is called a central scotoma. It is rare, and many observers have never seen it. There may be, secondly, homonymous, bilateral hemianopsia, in which the half-field on the same side for each eye is blank. This is also exceedingly rare in hysteria, but not so uncommon in organic brain disease.[1] I present a diagram (Fig. 29) made by Dr. de Schweinitz from a patient in my wards at the Philadelphia Hospital. Her case is related briefly elsewhere. Hysterical homonymous hemianopsia is associated usually with hysterical hemianæsthesia and with unilateral affection of the other special senses. There is almost always anæsthesia of the conjunctivæ, a symptom which may serve to distinguish the affection from hemianæsthesia due to organic brain disease. The blind fields are always on the anæsthetic side. The half-fields in which sight is retained are usually contracted, as in the case here shown; this contraction, as will presently be demonstrated, is a peculiarly hysterical phenomenon. A rare form of hemianopsia is the binasal variety, which is illustrated in the accompanying diagrams (Fig. 30), from a case of Mitchell and de Schweinitz. Finally, there may be a concentric narrowing of the visual field. In this form of amblyopia sight is retained for the centre of the field, while it is lost in varying proportions for the periphery. In some cases the restricted field is very

FIG. 29.



Diagrams of fields of vision in a case of hysterical homonymous hemianopsia from author's clinic at the Philadelphia Hospital. Contraction of the sensitive half field. Colors correctly named at fixing-point in the left eye. Colors correctly named within temporal side in an area 10° wide in right eye.

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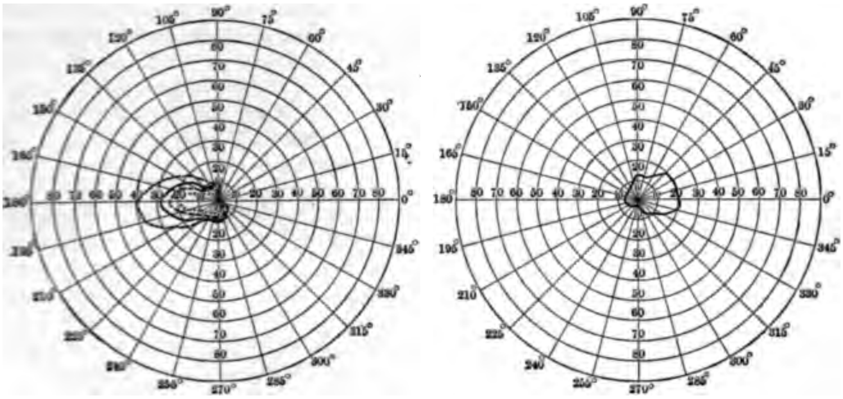
[1] Wecker et Landolt (Traité Comp. d'Ophthalomologie, article on "Amblyopie Hystérique," p. 712) refer to a number of cases of hystero-epileptic amblyopia which presented the characters of an homonymous hemianopsia. Such cases have been reported by Rosenthal, Sturge, Galezowski, Westphal, and others.

small; in some, also, it is almost round; in others it is oval, or it may be rather irregular in shape. It may be narrowed to less that 20° (Fig. 31). It is practically a *concentric* narrowing, *i. e.*, the centre of the normal field remains the centre of the restricted one. This concentric narrowing of the visual fields is one of the most common of the sensory stigmata of hysteria. It is, of course, as a rule, unknown to the patient.[1]

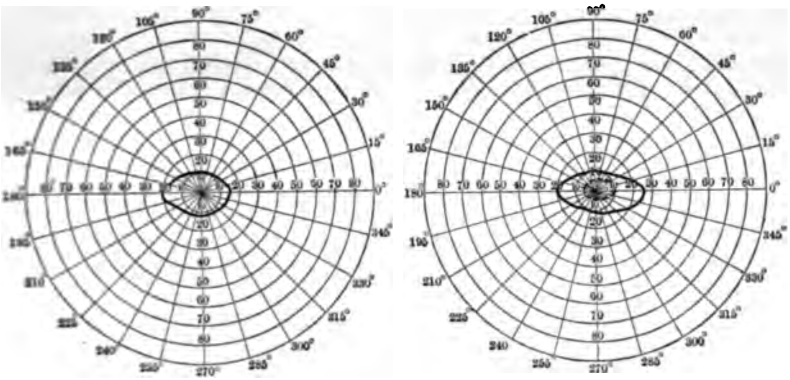
FIG. 30.



Diagrams of the fields of vision in a case of hysterical hemianæsthesia. Irregular binasal hemianopsia; partial reversal in the color fields of the left eye; color perception only at the fixing-point in the right eye. (MITCHELL and DE SCHWEINITZ.)

-·-·- — red. ------- — blue. .... — green.

FIG. 31.



Diagrams of the fields of vision in a case of universal hysterical anæsthesia, showing marked concentric contraction. (MITCHELL and DE SCHWEINITZ.)

In hysteria the perception for colors may be affected; hence there may be achromatopsia or dyschromatopsia. In the former the perception for all colors is lost. All objects are described as being of a dull-gray tint. A much more common affection is the latter state, or dyschromatopsia, in which the normal perception for colors is perverted. In the normal eye the several

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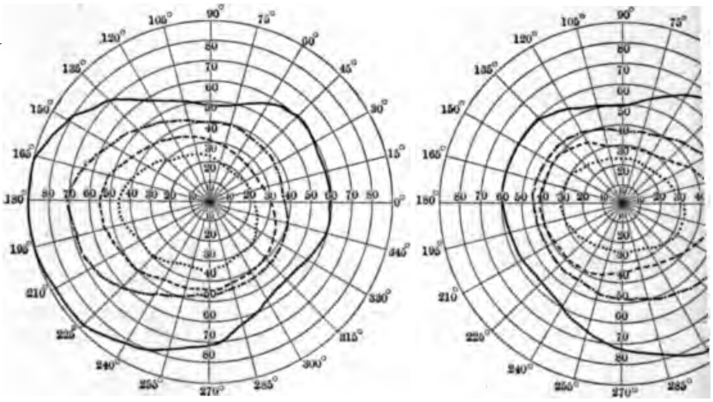
[1] It is important for examiners to bear in mind that this narrowing of the visual field is influenced readily sometimes by suggestion; and this not so much to produce it, as to abolish it. An impatient or brusque manner may defeat the object of the examiner. This may account for the varying results reported.

colors of the spectrum are not perceived in coequal and coext
thus, violet occupies the smallest or innermost field, green the :
red the next, yellow the next, and blue the largest or outermost.
of perceptions is changed in hysteria usually by the exchange of
series between red and blue. Hence, red comes to occupy the
(Fig. 32). Moreover, in the narrowing of the visual fields, alrea
the color fields are also narrowed and in the order of their nor

FIG. 32.



Diagrams of fields of vision in a case of hysteria, showing normal form field and re
red and blue lines, the red field being largest in extent. (MITCHELL and DE SCH
-·-·-·- = red. ·-·-·-·-·-·-·- = blue. .... = green.

Thus, violet disappears first, then green—or, as someone has sai
"squeezed out at the centre;" but the blue disappears before the
is the most persistent color-perception in the hysterical patient.
this the case that some writers have claimed, rather fancifully, tha
red plays a part in the deliria and hallucinations of the gra
Finally, in some cases of hysteria there may be little or no alterat
fields of vision.

A very curious visual phenomenon seen in hysteria may be
here, although it is, probably, a muscular rather than a purely sens
This is monocular diplopia or polyopia. In this the patient sees tv
images of the same object with one eye, the other being closed. It
ascribed by Perinaud[1] to a spasm of the muscle of accommodation,
confined to hysteria.[2] It was noted in an hysterical patient of tl
at the Philadelphia Hospital, and also in another patient in the s
who was suspected to have hysterical symptoms grafted on syphilis of
cord, and who was found by Dr. de Schweinitz to have a syphilitic c
The diplopia was more probably due to hysteria than to the organ
for it is difficult to see how a choroiditis could cause monocular dip

The other special senses, as said already, may be affected in va
in hysteria.

Hysterical deafness is a not uncommon accompaniment of
hemianæsthesia.[3] It may occur, however, independently, and may

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[1] De la polyopie monoculaire dans l'hystérie et les affections du systeme nerveux. A
listique, 1878.

[2] It occurs in commencing cataract, and is due apparently to a disturbance of the refr
of the lens, which would also be its immediate cause in spasm of the muscle of acco
Perinaud says it is produced sometimes by atropia, which also interferes with refractio

[3] See paper by Walton on "Deafness in Hysterical Hemianæsthesia." Brain, v. 1883,

and complete, although this is rare. It is frequently not complete; in other words, the hearing is not abolished, but only impaired. It is not accompanied with tinnitus or noises in the head. That it is a purely central or psychic affection can be demonstrated by the tuning-fork, which can be heard by aerial conduction better than by bone conduction—a proof that the deafness does not depend upon disease of the external or middle ear. In this respect hysterical deafness is analogous to hysterical blindness—it is a strictly psychical affair. In hysterical deafness the sensibility of the auditory canal, tympanum, and even of the middle-ear is abolished. The patient tolerates manipulation of the drum, and the use of the Politzer bag excites no sensation. Walton says that the degree of deafness corresponds with that of general anæsthesia. The deafness may be transferred with the anæsthesia.

Loss of smell, or anosmia, is present apparently is cases of hemianæsthesia. It seemed to be so in the case reported elsewhere in this paper from the writer's clinic at the Philadelphia Hospital. It is difficult, in hysterical cases, to make satisfactory tests of the sense of smell. In this case all the special senses on the anæsthetic side were involved.

The sense of taste may be affected in hysteria, either entirely or unilaterally, or in limited areas. The unilateral involvement of this sense in hysterical hemianæsthesia can be demonstrated satisfactorily, but the other forms, especially the loss of taste in limited areas as reported by some writers, must be very hard to be demonstrated, because both of the difficulty of limiting the action of a sapid substance to small areas on the tongue and of the extreme likelikood of influencing the patient by suggestion in the necessary manipulation. Pitres[1] states that there is no necessary connection between the loss of general sensibility of the tongue and the loss of the sense of taste. In some cases, he affirms, the taste is abolished in the anæsthetic areas only; in others the taste is lost, although the general sensibility is retained; in others, again, the taste is preserved on parts of the tongue that are insensible to heat and to pain; and, finally, in some cases, there may be areas of tactile anæsthesia and areas of gustative anæsthesia not coextensive.

MOTOR SYMPTOMS. The motor symptoms of hysteria may be divided into four groups: paralysis, contracture, tremor, and inco-ordination. They will be described in the order named.

The paralysis of hysteria has various forms. It may be a monoplegia, a paraplegia, an hemiplegia, or even, in rare cases, a total palsy. It sometimes presents itself as a very limited paralysis—*i. e.*, it may be confined to a few muscles or muscle-groups. Thus it may present the form of a facial paralysis, a paralysis of some of the arm or hand muscles, or of the muscles of the neck (causing torticollis). Finally, there may be paralysis of the muscles of the tongue, pharynx, larynx, œsophagus, and even of the anus. Most of these forms of hysterical paralysis are likely to be accompanied with contracture, so that it is difficult to describe these two symptoms apart. But for clearness this will be done, attention being called occasionally to this fact by appropriate illustration.

Hysterical paralysis, with or without contracture, has several well-recognized causes. It is especially likely to be caused or aggravated by a convulsion. Thus it may appear as a prodrome, and may persist after the fit for various periods. It may be caused by trauma—a not infrequent cause, and a most important one to be recognized. This paralysis, combined with certain other stigmata, as segmental anæsthesia, may play a conspicuous rôle in medico-legal cases due to trauma. In surgical cases forms of this paralysis may assume great importance as confusing complications. Again, hysterical paralysis may be caused by emotion, such as fright, anger, chagrin, or disappointed love. I have referred elsewhere to the case of a young woman, once under my care, who became paraplegic after an exciting episode with her favorite parson. This paralysis may be caused also by various toxic and morbid states, as by the infection of syphilis, typhoid fever, or other zymotic disease, and by such poisons as alcohol. Finally, most or all of the causes referred to under the head of the general etiology of hysteria may cause paralysis. This symptom does not always occur immediately after the primary action of the cause; thus some days may elapse, in the case of trauma especially.

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[1] Op. cit., p. 88, vol. i.

Hysterical paralysis may vary in degree from a slight loss of power (amyosthenia) to total palsy. The deep reflexes of the affected side are usually increased and the skin reflexes abolished; the tendency to contracture is often marked. Some cases, however, present a flaccid type. In mild cases the nutrition of the limb is not affected, but in severe cases of long duration slight but distinct loss of volume may be noted. True atrophy, with reactions of degeneration, is practically unknown, and when present must throw a doubt over the exactness of the diagnosis. Some French writers claim, however, that such atrophy and reactions do occur. Care must be taken not to confuse electro-anæsthesia—a common hysterical stigma—with abolition of electro-contractility.

Hysterical paralysis, as said already, may be accompanied with contracture in various degrees. It is often accompanied also with anæsthesia or hyperæsthesia. This anæsthesia is likely to be sharply defined and limited to the paralyzed part; thus in hemiplegia it is a hemianæsthesia, and in monoplegia a monoanæsthesia. This anæsthesia of the paralyzed limb is sharply delimited, the boundary being at right angles to the long diameter of the limb. In transfer of hemianæsthesia, already described, mild grades of amyosthenia also may be transferred. The paralyzed part may become œdematous and blue or mottled—a phenomenon that was noted by Sydenham. The hyperæsthesia accompanying paralysis is usually hyperalgesia. This hyperalgesia may be attended with contracture, the painful cramp-like state of the muscles causing the patient to cry out and to shed tears.

Finally, hysterical paralysis may come on suddenly, or it may develop as a mild form, gradually growing worse. In all forms of this paralysis there is likely to be some slight movement left, but there is often an indisposition in the patient to exert him or herself to make even this slight movement. The paralysis is not, as a rule, confined to the distribution of particular nerve-trunks: in other words, it is *central*, not peripheral. Occasionally hysterical paralysis is transitory and recurring—an access of paralysis comes apparently without cause, or with prodromes, endures for a period, passes away, and then, after a longer or shorter interval, recurs.[1] Such a type is peculiarly hysterical, and is produced by no other disease. Richer[2] records such a case of transitory paralysis, affecting now one limb and now the other, in which there was abolition of electro-contractility in certain groups of muscles. Briquet[3] noted that in some cases the paralysis passes from one side of the body to the other, or leaves successively one part, as the arm, the leg, the larynx, or the diaphragm, to pass into another.

The duration of hysterical paralysis may be greatly prolonged. Some cases recover promptly, but others persist so long and simulate so closely the effects of organic disease, that even the most careful observer may come to distrust the exactness of his diagnosis. I have such a case now under observation, described in this chapter which began as a pseudo-coxitis, became a paraplegia, and is now a hemiplegia plus the paraplegia, and exhibits also sensito-sensorial hemianæsthesia, and yet, after several years, gives no promise of recovery.

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[1] See "Report of a Case of Periodical Paralysis," by Dr. C. W. Burr, Univ. Med. Mag., August, 1893.

[2] "Paralysie Erratique," etc., in his Paralysis et Contractures Hystériques, p. 30.

[3] Op. cit.

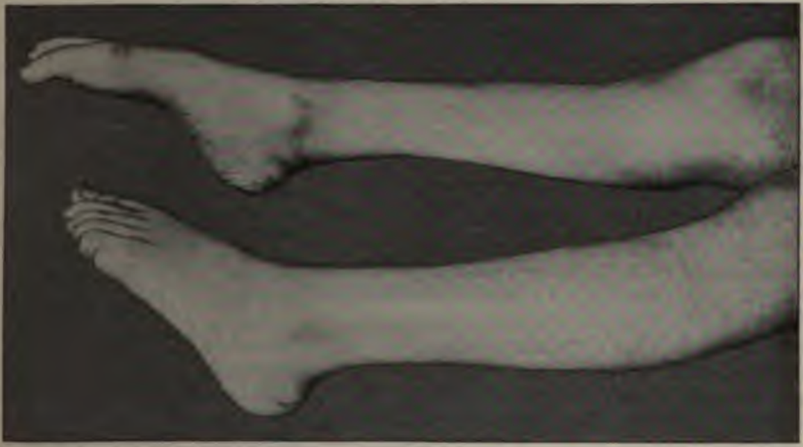
The termination of hysterical paralysis is sometimes sudden, following some shock or strong mental or moral impression. Sometimes, however, recovery is gradual under well-directed treatment.

The hemiplegia of hysteria has some special characteristics. The leg is more paralyzed than the arm. The arm usually lies flaccid by the side, but the foot assumes the position of equino-varus. There may be, and usually is, stiffness of the leg, which is extended. If the patient retains an ability to walk the leg drags as an inert mass, very different from the spastic gait of organic hemiplegia. The muscles of the face and tongue, as an almost universal rule, are not paralyzed.[1] There may be, however, a contracture of the facial muscles on either the paralyzed or the sound side. There is no true aphasia. The hemiplegia in the great majority of cases is on the left side. Usually there is hemianæsthesia, involving the special senses, of the paralyzed side. In bed-ridden cases bed-sores do not form. According to Briquet's statistics, hemiplegia occurred in 74 cases in a total of 430 hysterical patients.

The following case of hemiplegia presents a type:

D. K., aged twenty-five years, was a patient of the writer's in the Philadelphia Hospital. Her long history can be given in brief epitome. She had first presented a pseudo-coxitis of the left side. There had followed a paraplegia with contractures (Fig. 33). Then paralysis of the left arm and hand developed rather suddenly after the paraplegia had continued for several months. Thus the case became a combined para- and hemiplegia. The arm remained flaccid. At the date of these notes the case was as follows: The motor symptoms were as described. The legs were paralyzed and contrac-

FIG. 33.



Hysterical paraplegia with contractures. (Philadelphia Hospital.)

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[1] The few exceptions reported by Chantemesse and others cannot be held to invalidate this rule until they are strengthened by additional observations.

tured, with the feet in the position of equinus. The knee-jerks were retained, but not exaggerated; there was no ankle clonus. There was no paralysis of the bladder and no bed-sores. The left arm was paralyzed and flaccid, and lay extended along the body. The faradic contractility of some of the leg-muscles was slightly diminished; the galvanic response in the same muscles was also quantitatively diminished, and in some few muscles the anodal contracture about equalled the cathodal. (This is rather in accord with the claim of some French observers that the reactions of degeneration are found occasionally in hysteria.)

(*a*) *Tactile sensibility*. Hemianæthesia on the left side of the body from head to foot; anæsthesia does not touch meridian line in front, but begins one and one-half inches to the left. On the right side sensation is present on the face, arm, and trunk. In the lower limbs it is absent, except in a small area, 5 x 2 inches, on the anterior surface of the thigh. The right sole is hyperæsthetic. On the back there is the same area of anæsthesia. The anæsthesia involves the mucous membranes.

(*b*) *Pain-sense*. Analgesia corresponds to anæsthesia, except that the right arm and right side of the back are also analgesic. Analgesia includes also the bones and muscles.

(*c*) *Muscular sense*. This is almost totally lost. The patient does not know the position of her limbs. The sense of fatigue also is lost.

(*d*) *Electro-sensibility*. This is lost in the anæsthetic areas. On the left side of the neck the current produces a tonic spasm of the sterno-cleido-mastoid muscle, which is not painful.

Special senses. There is loss of taste on the left half of the tongue. Loss of smell is noted in the left nostril. Hearing is diminished on both sides, more so on left. Watch R. E., 10 cm.; L. E., 6 cm. *Sight:* contraction of the visual field, reversal of the color field in the left eye.

By the application of metals no transfer could be obtained, nor any change in the anæsthetic area of any kind.

The patient had, as characteristic mental stigmata, passiveness of mind, indisposition to exert herself, indifference to being cured, and some emotionalism. She never had had a convulsive attack.

The paraplegia of hysteria simulates closely organic paraplegia, but it has some reliable differentiating points. After trauma or violent emotion it may occur rather brusquely, but otherwise its approach is insidious. It is not accompanied with pains nor with a girdle-sense, neither does it present reactions of degeneration. It usually presents a characteristic anæsthesia. This is segmental—*i. e.*, it is sharply delimited, not involving the genitalia, and running no further than the crests of the ilia. The bladder and rectum are not paralyzed, and bed-sores and other trophic lesions do not form. The tendon-reflexes may be exaggerated, but exceptions occur. Contractures frequently occur; the legs usually are rigidly extended, the feet in the position of equino-varus. I have etherized such a patient in the Philadelphia Hospital, and found that under ether the limbs were entirely relaxed and freely movable in all directions. After recovery from the anæsthetic no complaint was made of pain in the limbs, which had thus been submitted to very energetic passive movements. In protracted cases the muscles become rather reduced from misuse, but do not truly atrophy; in fact, in some cases with contracture, rather the reverse is seen, the muscles remaining firm and well-nourished.

Paralysis of the four limbs has been seen in hysteria, but it is extremely rare. It usually occurs gradually, and, according to Richer, invades the leg before the arm, and the left side before the right. Chevalier, in a special study, was able to find only twenty-one authentic cases recorded.[1] The muscles of the face and trunk, and of respiration, are not paralyzed, but the bladder may be so. There may be aphonia and dysphagia. One side, usually the left, is more paralyzed than the other.[2]

Other special forms of paralysis are seen in the face, in the eye-muscles, in the tongue, and in the muscles of the neck. They are not to be confounded with contractures. They may coexist, in fact, with contractures, the muscles of one side being paralyzed and of the other contractured. Contracture of the face-muscles, as already shown, may be observed in hysterical hemiplegia much more commonly than paralysis. The tongue also may deviate because of contracture. I have seen the tongue in a case of hysterical hemiplegia deviate to the *sound* side by reason of contracture of its muscles.[3] Part of the face only may be involved in palsy.

Contracture, as already said, is very likely to coexist with paralysis in hysteria; still, this is not a constant rule. Neither is the reverse true, that the contractured limb or muscle is always paralyzed. The contracture of hysteria has a few distinguishing traits. It may be caused by most of the agents which cause paralysis, and its onset may be sudden or gradual; a sudden onset is the more common. It may follow and complicate a paralysis, or it may occur independently of one. It is a most obstinate and resisting contracture, being very difficult to be overcome, even with great force. Moreover, the antagonistic muscles are involved; in other words, the limb is held in a vise-like immobility. The contracture is sometimes so persistent that it does not relax even in sleep; it does relax, however, under ether or chloroform. The muscles retain their nutrition, although in long-continued cases the limb may waste. Sensation is often abolished in the affected limb. The contracture may appear, disappear, and reappear: it may return to the same limb; or it may be erratic, like some forms of paralysis, and reappear in another limb. The duration of this symptom is very variable; sometimes it is most protracted; sometimes it yields suddenly to some unexpected cause. In some cases the contracture is painful; in such cases the general health suffers, the patient loses flesh, and passes into bad *morale*. I have known such a case to present a marked remittent type, a contracture in the arm coming on in the midst of an abortive convulsive seizure, and enduring for some hours. It could be excited by pressure on the musculo-spinal nerve, and was so painful that the patient cried out with it.

The case briefly was as follows:

W. T., aged twenty-three years, female, dressmaker, was admitted to the Methodist Episcopal Hospital under my care. There was marked heredity. The parents were both neurotic, and one sister had had convulsive hysteria. Six years before admission the patient had an hysterical convulsion, with unconsciousness. Subsequent attacks occurred. They were preceded by an aura of pain in the ball of the left foot, spreading up the leg and thigh. Following the first seizure the patient was confined to bed for one year and a half. Ever after the attack she had aphonia, the voice being scarcely audible, and she had also pains in the back and sides. One year before admission she fell and struck her back, after which trauma the seizures were worse and the aphonia increased. Pains increased; there were hyperæsthetic zones on the dorsal and cervical spine. She had felt a numbness in the neck for a long while after falling. The patient could always foretell a fit by the pain in the ball of her foot; she voided much urine of light color and low specific gravity before the attack. In the attack opisthotonos was marked, and the period of grand movements, the clonic stage, was aborted. She never bit her tongue. There was much embarrassment of breathing. There was slight intervallary tremor and occasional vomiting. Nutrition was poor, but weight had not decreased for some years. A single whiff of ether was said to stop the convulsion. On admission the patient was found to have anæsthesia, as depicted on the diagram (Fig. 34). She was not aware that she had any loss of sensation. There was ovarian hyperalgesia. A few days after admission she was excited by an accident case that was admitted into the ward; shortly afterward a spasmodic painful contracture of the fingers, hand, and forearm occurred, as shown in the photograph (Fig. 35). It could be relaxed gradually by firm, continued extension. It was accompanied with great pain not only in the arm, but in the chest, neck, and back. In a few minutes the left thigh was drawn up on to the abdomen, with the leg and toes extremely flexed. On forcible extension of the leg the patient lapsed into unconsciousness, with eyes closed, lids quivering, pupils widely dilated, mouth open, and head retracted. The other arm and leg then became tonically contractured. The patient wept toward the close of the attack. Pressure on the supraorbital nerve ended the attack. In some attacks there was great dyspnœa following the convulsive phenomena. It was found that all these phenomena could be induced by making firm pressure on the musculo-spinal nerve. The painful contrac-

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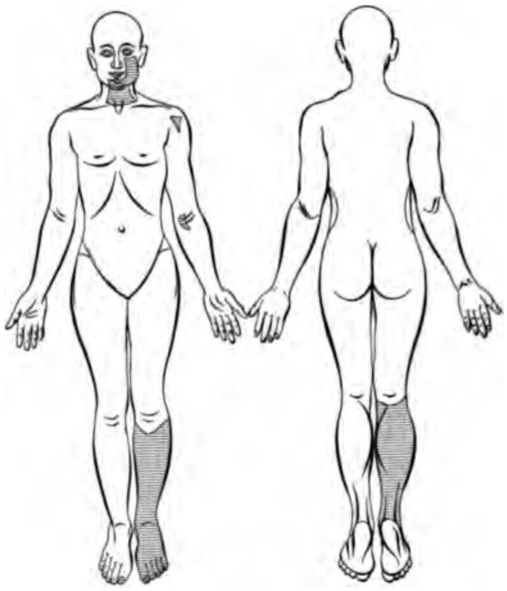
[1] Two instances, evidently of this form of hysterical paralysis, are reported in the St. Bartholomew's Hospital Reports for 1892, under the misleading title of "General Spastic Rigidity." The authors of the report do not recognize the hysterical nature of the cases.

[2] Moebius has described a form of paralysis which he calls *akinesia algera* (pain-paralysis), and which depends apparently upon an inhibitory imperative conception. The patient dreads to move for fear of pain. The affection is rare and is allied probably to the intention psychoses, such as claustrophobia, agoraphobia, etc. It is a symptom of degeneracy rather than of hysteria. Genuine hysterical symptoms were not observed in Moebius' patients, one of whom became insane.

While not ambitious to coin new names, I suggest that *kinesiphobia* better expresses the mental state in these patients than does the compound name given to the affection by Moebius. It is a fear of movement analogous to the fear of contamination (mysophobia) and the other morbid fears of this group that seems to characterize his patients.

[3] The tongue is capable of complex co-ordinated movements, hence it is not likely that the muscles of one side alone are contractured in hysterical cases. These hysterical contractures of the tongue are co-ordinated movements in which muscular fibres on both sides may be involved.

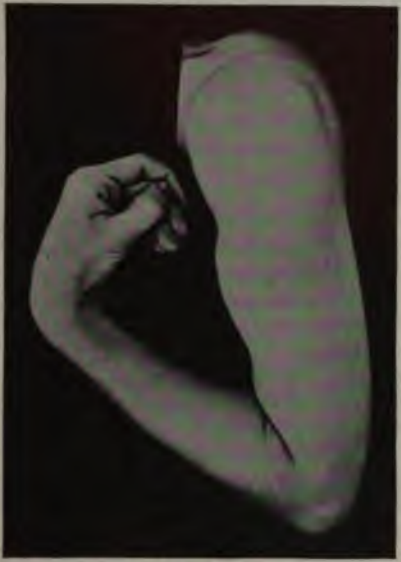
FIG. 34.



Segmental and insular ▤ *Anaesthesia* in a case of hysteria. (Methodist Episcopal Hospital, Philadelphia.)

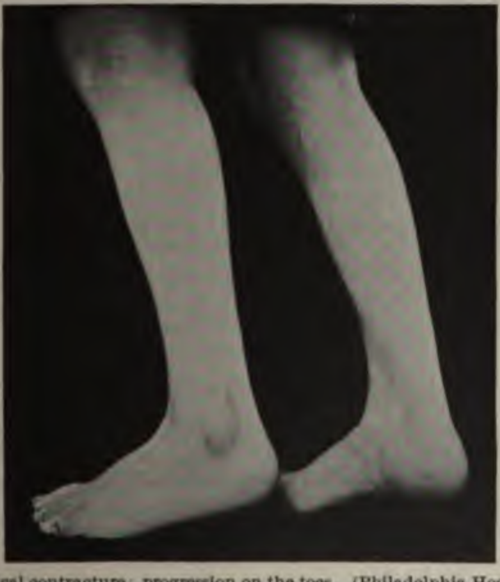
ture of the arm was the most persistent symptom. This patient was cured by isolation, hydrotherapy, massage, and the ministrations of a wise and firm nurse. The aphonia was the first symptom to yield. She was discharged cured, with much increased weight, in about one month.

FIG. 35.



Hysterical contracture. (Methodist Episcopal Hospital, Philadelphia.)

FIG. 36.



Hysterical contracture; progression on the toes. (Philadelphia Hospital.)

Hysterical contracture may be confined to some few muscles of a limb. In such cases the limb may not be disabled, but only limited in movement to the extent of the contracture. Some of these cases assume strange types, and may be very puzzling for a while in the diagnosis. One of the rarest of these types that has come under my notice was the following:

S. A., aged eighteen years, widow, Russian Jewess, was in the Philadelphia Hospital under my care for several months. She was dwarfish in stature and of rather low intelligence. No history was attainable. She was in good general health and attracted attention merely by the peculiarity of her gait. She walked entirely on her toes, *i. e.*, on the balls of her feet and toes, the heels being elevated almost one inch from the ground (Fig. 36). On close inspection it was observed that this mode of progression was caused by firm contracture of the calf muscles. These were so firmly contractured that the utmost allowable force could not overcome them. The tendines-Achillis were taut as in cases of club-foot, to which class of affections it was at first thought that the case belonged. It was soon seen, however, that the case was not an example of ordinary double pes equinus. The muscles were well nourished, and reacted normally to the electric currents. The skin was not cold, although slightly mottled. There was no true deformity of the foot. The contractures were not painful except when attempts to overcome them were made. The pain then was felt mostly in the regions of the popliteal spaces. The two legs were exactly alike, which would be rather a rare occurrence in double club-foot. A diagnosis of hysterical contracture was made. It was confirmed by the discovery of anæsthesia in extensive areas, involving especially the left arm and left chest. There was also aphonia. The patient continued to walk as described for many months. Close observation by internes and nurses failed to detect any remission of the affection. It suddenly disappeared, however, on the reception of some pleasant news, which occasioned the patient's removal from the hospital. She walked out cured.

Other forms of contracture are in the muscles of the face, tongue, eyes, and neck. Some of these, as already observed, may coexist with paralysis of other muscles. Facial hemispasm is not uncommon in hysterical hemiplegia, and must not be confused with paralysis. It may be associated with peculiar contractures of the tongue, drawing it to one or the other side, the appearance and seat of these contractures producing deformities that are not identical with the hemiparesis of the lower face and of the tongue as seen on the same side in organic hemiplegia. Thus the contracture may protrude the tongue *away* from the paralyzed side, instead of its being protruded *toward* that side as in organic disease. The contracture of the face may be associated with contracture of some of the eye muscles, called blepharospasm; this may give a superficial appearance of ptosis, when in fact, instead of paralysis of the upper lid, there is a slight contracture of the orbicularis muscle. Torticollis, due to contracture of some of the muscles of the neck, is seen occasionally in hysteria.[1]

Tremor is one of the most important of the motor stigmata of hysteria.[2] It is caused especially by trauma and by toxic agents, as alcohol, lead, and mercury. In traumatic hysteria this symptom is always bound to play an important part, being conspicuous sometimes in medico-legal cases, and in men as frequently, if not more frequently, as in women. It may be a very persistent symptom, and may simulate closely the effects of organic disease. In toxic cases, as from alcohol, lead and mercury, it is likely to be confounded with the organic effects of the poison; yet, as shown elsewhere (page 95), the tremor observed in these cases is sometimes purely hysterical. I have twice seen persistent tremor caused by the shock of a supposed poisoning, once by mercury and once by saltpetre. In neither case was there any real injury done by the poison. I have also seen it caused by a severe fall, by the terror from an earthquake, and by ether anæsthesia during a surgical operation. Two of those cases lasted for years, one being cured entirely; the others lasted for variable periods of months. This tremor may be divided into classes, as Dutil has done, according to the number of oscillations per second. Thus (1) oscillations rapid or vibratory (8 to 12 per second), (2) medium rhythm (5½ to 7½ per second), (3) oscillations slow (4 to 5½ per second). In the last two groups some tremors occur only on voluntary motion, but the most common is what is called the "type Rendu," in which the rhythm is from about 7 to 9 per second, and in which the tremor continues during repose, but is much increased in amplitude, but not in rhythm, during voluntary movement. This type was presented by all of my own cases, as well as by the cases of Luys and of Westphal (which latter are of interest because they were not attributed to their true cause), and also by cases reported by Charcot and others. The following is an illustrative case:

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[1] See Richer's work for details of these various forms.

[2] I have described elsewhere at length the symptoms and course of hysterical tremor. "Hysterical Tremor and Hysterical Anorexia, etc." Am. Jour. of Med. Sci., September, 1893.

S. C., aged twenty-four years, white, single, has been an inmate of the Home for Crippled Children under my care for eleven years. During all of that time she has been confined to her bed. She is a case of muscular dystrophy. She is almost totally paralyzed in her legs, but retains some movement of her feet and toes. Gradual contractures of her legs had been established during several years until the heels were against the buttocks. These contractures were very painful, and caused the patient much unrest. In order to straighten the legs, both for the patient's comfort and for the better nursing and care of her person, she was etherized. It was proposed by

FIG. 37.

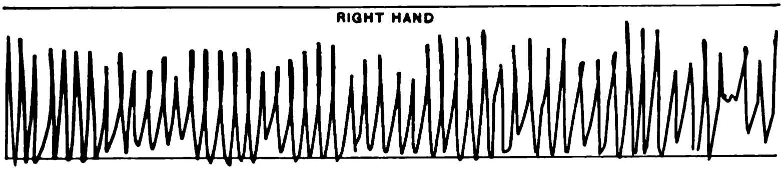
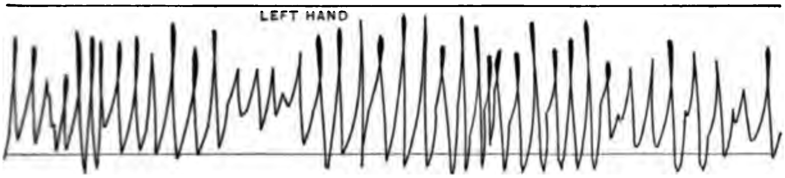


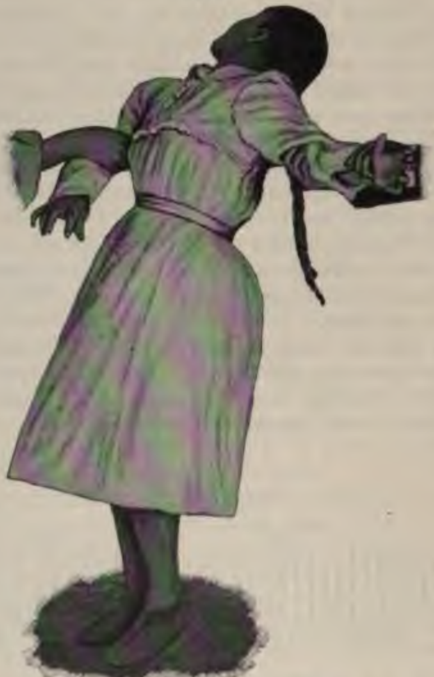
FIG. 38.



Tracings of hysterical tremor following surgical anæsthesia. (Home for Crippled Children.)

Dr. Willard, the surgeon to the Home, to perform tenotomies and to put the legs in plaster dressings. To our surprise it was found that under ether the contractures were completely relaxed, and that the legs were freely movable in all directions. No tenotomies were required. The plaster dressings were applied, and the patient returned to the ward. For many days after the etherization much pain in the legs was complained of. The patient was depressed, and was much impressed with the idea that an operation had been performed. In less than one week a very marked tremor appeared in the hands, arms, and toes. It was constant during repose, but much increased by voluntary motion. It prevented the patient's constant work, sewing and embroidering. It could be increased by suggestion, and was eventually, after several weeks, cured by it. A tracing of this tremor is shown in Figs. 37 and 38. This patient has had other stigmata, as aphonia of several weeks' duration, and segmental anæsthesia of the legs. The contractures were suspiciously like those of hysteria. She has worn the casts now for twelve weeks. The tremor is cured, and the patient contentedly works at her sewing.

FIG. 39.



Hysterical astasia-abasia. (Methodist Hospital.)

Finally, the fourth motor affection of hysteria is inco-ordination, or hysterical ataxia, or, as it has come to be called, after Blocq, *astasia-abasia*. This curious and rare affection was referred to by Brodie and later by Jaccoud. It was described by Charcot and Richer in 1883 and by Weir Mitchell in 1885. Briquet refers to a form of hysterical ataxia dependent upon anæsthesia of the legs and feet. But the term astasia-abasia is confined almost exclusively now to a species of inco-ordination, which is not dependent upon anæsthesia or paralysis, and is not associated with even loss of the finer co-ordinated movements. As the name implies, it is a loss of power of standing (astasia) and of walking (abasia.) When the patient sits or reclines there is full muscular power in the legs, and the ability to execute co-ordinate movements, as crossing the legs, or even walking on "all fours" is preserved. There are various grades of the affection, from total inability to stand, and of course to walk, up to a slight swaying and ataxic gait. In cases of medium grade the patient may progress if supported by an attendant on each side. The gait, as I have seen it, is characterized by a series of inco-ordinate movements of the legs, associated with alternate bending forward and arching backward of the body and even of the head. But little forward progress is made, and the limbs seem to be so relaxed that without support they would double up beneath the patient. Occasionally there is an intercurrent stiffening of the legs, the patient tending to rise on the toes. This occurs with an associated backward, or opisthotonic, movement of the trunk. The knee-jerks usually are normal. There may or may not be hyterical anæsthesia, but it is not the cause, when present, of the inco-ordination.

Astasia-abasia, according to Blocq's[1] study, occurs most frequently in children and young persons. It is especially likely to be caused by trauma, and by moral or emotional disturbance.[2] In one case it followed a hard labor, in another an attack of typhoid fever.

The following case illustrates this anomalous affection:

M. R., aged, twenty-eight years, German, was a patient in my wards at the Methodist Episcopal Hospital. She had a history of convulsive attacks and of several spells of ataxia. Five weeks before admission she had been badly frightened. Then followed a series of hysterical prodromes, such as fulness of the head, auræ in the stomach, pulsations in the chest and throat, and headache. Then followed an abortive seizure. Then the astasia-abasia developed suddenly. This was the most marked symptom for some weeks. It is illustrated in the photograph (Fig. 39). The patient required support on either side. The movements of the legs were wildly inco-ordinate, tending now to fly outward, now to be drawn up, and again to be arrested by a tonic contracture, which drew the patient up on her toes, with her body backward as in opisthotonos. But little forward progression was made unless the patient was urged or partially carried along. When sitting or lying she had full control of her legs, and full power in them. The knee-jerks were exaggerated. This patient had other stigmata, the most marked of which was an almost total anæsthesia, as shown in Fig. 28.

Contrary to the views formerly held, muscular atrophy has been claimed recently to occur in hysteria. It affects the limbs which are the seats of other stigmata, such as paralysis, contracture, and anæsthesia. It may even show the reactions of degeneration. I have never seen such a degree of muscular degeneration in hysteria, except possibly in the case mentioned elsewhere in this paper, and can only say that the observations of it are still far from numerous. Löwenfeld[3] classifies atrophies in hysteria as follows:

1. Simple atrophy, (*a*) in connection with paralysis and contracture, developing quickly, without fibrillation or De R; (*b*) unconnected with paralysis or contracture, but occupying the site of an hysterical anæsthesia.
2. Degenerative atrophy, with fibrillation and De R.

VISCERAL SYMPTOMS. Among the visceral and internal disorders of hysteria may be considered vomiting, cardiac, vascular, and vasomotor affections, rapid respiration, pyrexia, cough, aphonia, yawning, phantom tumors, and anuria.

Hysterical vomiting, or *anorexia nervosa*, as I have said elsewhere,[4] is dominated by a mental state that is often difficult to unravel. These cases sometimes exhibit the effects of profound mental impressions, as supposed poisoning or imagined disease of the gullet or stomach. The two symptoms, vomiting and anorexia, are not, however, strictly synonymous. Laseque[5] has shown that a prolonged and almost persistent refusal of food may coexist with the preservation of quite a remarkable vitality, but not associated with vomiting. Sir William Gull described a series of similar cases, in which refusal, not vomiting, of food was the symptom. Where vomiting is added to anorexia an element of danger is found which does not appear to have been present in Laseque's and Gull's cases. In the case reported by me[1] the vomiting had been caused by a supposed poisoning by saltpetre. It had continued for many months, had reduced the patient to a condition of extreme emaciation, and had even endangered her life. The vomiting of hysteria has some special features. It is accomplished without effort, and apparently without nausea. It is not always so much an act of vomiting as it is one of regurgitation; in fact, it seems that in some cases the food does not always enter the stomach, but is rejected from the œsophagus. Hence this symptom is called sometimes *œsophagismus*.[2] Sometimes, as in my case, these acts of regurgitation follow each other in regular succession, especially if the patient is observed. As the gullet and stomach frequently are empty, these acts do nothing more than detach from the depths of the throat viscid and tenacious matter, as recorded by Naudeau,[3] of a case more than a century since. This vomiting, with the attendant emaciation, may cause the case to simulate organic disease. The cause of the malady, the character of the vomiting, the age of the patient, who is usually young (although exceptions to this occur), the absence of cachexia, and the presence of other hysterical stigmata, may serve to distinguish the affection. Its course, as said already, is sometimes very prolonged, but its cure may be quite sudden and due to some unexpected cause; or gradual and produced by tactful management and the wise use of suggestion.

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[1] "Sur une Affection characterisée par de l'astasie et de l'abasie," par Paul Blocq.

[2] Féré, "Pathologie des Emotions," attributes the affection always to disturbed emotions. Later writers, given by *Féré*, p. 247, are Cahen, Séglas, and Maigre.

[3] *Pathologie und Therapie der Neurasthenie und Hysterie*, München, 1894.

[4] *American Journal of the Medical Sciences*, September, 1893, op. cit.

[5] *De l'Anorexie Hystérique*, Arch. gén. de Méd. Ap., 1873, 385.

Cardiac, vascular, and vasomotor symptoms are not uncommon in hysteria. Rapid pulse is observed frequently. It is not associated with cardiac or vascular murmurs, unless the patient happens to be anæmic, nor with dyspnœa. It may continue in spite of long rest in bed, and may be very obstinate to drugs, especially to digitalis. In paroxysmal hysteria the heart is variously affected; in the beginning of a seizure its action is usually quickened, but in the atypical crises, such as hysterical sleep and trance, its action is so depressed both in power and pulsation that it can be perceived only with difficulty. Vasomotor changes, such as flushings, blushings, œdema, and erythema are seen. In hysterical women a tendency to flush in circumscribed areas about the face and neck can be seen occasionally. The bloodless state of the paralyzed limb in hysteria and its inability to bleed to a pin-stick were objects of observation in past centuries; sometimes the opposite, a flushed or erythematous state, is seen in the part. Œdema and a blue mottling of the paralyzed limb also are seen.

Rapid respiration is observed occasionally in hysteria, and when present is highly characteristic, because it is not observed in exactly the same form in any other condition. The rate of respiration may be as high as 75, 80, or even 90 to the minute. It is not accompanied necessarily with a rapid heartbeat, in fact the contrast between the hurried breathing and the tranquil pulse is sometimes quite marked. Neither is it accompanied with dyspnœa, nor with any evidence of failure in the proper aëration of the blood. This is, perhaps, its most distinguishing mark. The color of the face and lips remains normal—there is no cyanosis. This respiration is decidedly of the upper costal type. Auscultation and percussion give negative results. I once saw this symptom in a lady during prolonged convalescence from a

[1] Loc. cit.

[2] The affection described rather sensationally as *merycism*, or chewing of the cud, is probably a pure neurosis, and similar to, if not identical with, some forms of hysterical vomiting.

[3] Sur une Maladie Nerveuse, Journ. de Méd., Chir., Pharm., etc., Juillet, 1789, p. 197.

severe surgical operation. In her case the number of respirations was about 75 per minute. The symptom persisted for several weeks. It ceased during sleep. It was cured by improving the general health, and by change of scene.

Pyrexia in the course of hysteria has been observed. This elevation of temperature is considered by some to be a true fever, by others it has been called a pseudo-pyrexia. The amount of urea excreted has not usually been estimated. The fever may be continued, intermittent, or remittent. It is possible that some reported cases have been instances of mild malaria or abortive typhoid. This hysterical pyrexia is observed sometimes after childbirth. I have seen it in one patient in two successive lyings-in, each time caused by an emotional disturbance. It is usually caused by disturbed emotions. Chauveau described a pseudo-meningitic type of hysteria with fever. Debove succeeded by means of suggestion in raising the temperature. Rosenthal thinks that there may be febrile symptoms in hysteria, but never true fever, *i. e.*, an elevation of the temperature of the body. These pseudo-pyrexial symptoms are flushing, rapid pulse, thirst, anorexia, etc. It is evident that some confusion exists as to what constitutes true fever; but there can be no doubt from the number of cases now recorded that an elevation of temperature is observed sometimes in hysteria.[1]

Persistent cough is sometimes one of the symptoms of hysteria. It is a peculiarly exasperating cough to those who are obliged to hear it, but it does not seem to particularly harass the patient. It is dry and brazen in character, and is not accompanied with expectoration.

Aphonia is a not uncommon permanent stigma of hysteria. In some cases the voice is entirely gone; in others an almost inaudible whisper is distinguished. Aphonia usually occurs suddenly; it may depart as suddenly, although it does not always depart thus. Sometimes the reappearance of the voice is a surprise even to the patient. In one of my patients who had had aphonia for six years a momentary sound of her natural voice occurred one day while she was laughing, and quite astonished her. Rapid restoration of the voice followed. Mitchell refers to the case of a lady with aphonia who talked aloud in her sleep; she was awakened by the unusual sound, but could not speak after awaking.

Yawning of a peculiar type is seen as a rare symptom in hysteria.[2] It may occur in paroxysms of long duration, during which the patient may be perfectly conscious, but unable to speak because of the alternate contractures of the muscles of the jaw in the repeated acts of gaping. The individual gape is much exaggerated and prolonged. There is an extraordinary intensity and duration of each gape. It differs from the normal act also in not being accompanied with the deep and noisy inspiration so familiarly known. These bouts of yawning may be followed by other hysterical symptoms.

Phantom tumor of the abdomen occurs occasionally in hysteria. In women it may simulate pregnancy, and may be accompanied even with enlarged breasts. It is seen also in men. It may occur with surprising rapidity in some cases, and may disappear gradually. It depends apparently upon an accumulation of air in the intestine.

Anuria was observed by the old writers on hysteria. It is usually taken for granted that it is a common symptom, which it is not, according to my observation. I had seen it in hysterical paraplegia. In one case in a young woman the catheter had been used. But this is not a wise thing to do. An excessive flow of limpid urine, terminating a fit, has been noted for ages.

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[1] For a full discussion of the subject of hysterical pyrexia, with citation of numerous authorities, see "Critical Digest of Hysteria and Neurasthenia," by J. Mitchell Clark, Brain, Spring number, 1894.

[2] "Contribution à l'étude des bâillements hystériques," par M. M. G. de la Tourette, Georges Guinon, et Huet. Clinique des Maladies du Syst. Nerv. Charcot, Paris, 1892.

Cases, like the one recorded by Finch, of anuria lasting for many weeks, are frauds, not true hysterics.[1]

PSYCHICAL SYMPTOMS. It is a difficult task to describe in short space the mental stigmata of hysteria. When it is recalled that hysteria is essentially a psychosis, and that all the paroxysmal, as well as all the interparoxysmal symptoms, are but representations, in some sort, of psychical states, this difficulty may be appreciated. In the hysterical child or young person the affective faculties especially are impressionable, because at this period of life these faculties dominate the mental life. Hence the depressing emotions, as disappointment, shame, and chagrin, have especial prominence in juvenile hysteria. Moods change, and motives are not always discernible. Sometimes these emotions are conspicuously unrestrained; the volition is at fault; a changeable and capricious temper often gives way to atrocious anger. Then there may be marked depression, sufficient to excite the anxiety of friends; and this depression may alternate with exaltation, even maniacal in tone. These affective disturbances, existing as hysterical stigmata, especially as prodromes of or even as substitutes for a grand convulsive attack, are the true instances of "hysterical insanity," which unfortunately has been confused with other mental states, especially with monomania and degeneracy. With Tourette, we think that the one mental trait that especially characterizes hysteria is the facility for receiving peculiar forms of impression, and that this mental state may be called *suggestibility*. The suggestions may come from without or from within, but in every case they make a profound impression, which is reflected by the patient's mind out along some motor or sensory nerve-track, and thus comes to make some one or more of the numerous stigmata which have been described. Thus violent emotion, trauma, and the various other causes already described may make in the vicissitudes of daily life these suggestions. A prolific source of these suggestions is no doubt within the patient, and this gives forth its store of ill-contrived ideas at the time especially of a convulsive paroxysm. Hence the frequency with which a grand attack is followed by some of the most obstinate permanent stigmata. The delirium of the fourth period is especially prolific of these. It influences remarkably the mental state between the attacks. Great importance, moreover, is to be attached to the nocturnal dreams and nightmares of hysterical patients. It is from suggestions received or prompted by these terrors of the night that a whole train of hysterical symptoms sometimes dates. Under this head come the disorders of sleep, such as the motor shocks and palsies, sensory disturbances, hallucinations of sight and hearing, and even dream-like delusions, that are not infrequent, in some degree, in hysteria, and which often store the mind with the evil promptings of disease. These are seen in an exaggerated degree sometimes in traumatic hysteria, the patient living over again night after night the alarming incident. Important medico-legal questions may arise with reference to the hallucinations and nightmares of hysteria. False accusations have been made by patients while in a true delusional state due to these causes. Attempts at suicide have been prompted thus, and the fact that they are usually abortive in hysteria is possibly because they are but the reflex acts from an hallucinatory and dream-like state. The explanation, usually given, that they are merely simulated, is totally inadequate to explain their exact motive, and the fact that occasionally they are successful.

According to the most accurate recent observers the mental state in hysteria may be said to be an alteration in consciousness, to which the term

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[1] The writer has used the word "hysteric" as we use the word "epileptic," *i. e.*, as a substantive to designate the patient suffering with the disease.

"hypnoid" applies appropriately. The hysterical patient lives in a sort of double consciousness; his mind forms two groups of phenomena, the one constituting the ordinary personality, the other forming an abnormal one, differing from the first and ignored by it. In this abnormal or duplex personality, the strange association of ideas, dating back to the initiation of the morbid process in some painful emotion or shock, perhaps even as far back as in childhood, has its field of action. From this background these ideas spread out and influence in a myriad of ways the bodily functions, often totally without the consciousness of the patient, much less with his consent or connivance. In this "hypnoid" state we see the relations of hysteria to the processes known as hypnotism; we recognize, in fact, that hypnotism is but an artificial production of a mental state clearly analogous to hysteria; hence we recognize the necessity for being cautious about admitting that deep *hypnosis* can be induced in perfectly healthy brains, or about encouraging its production, lest we induce irremediable hysterical perversions. This peculiar mental state leads to a contraction of the field of consciousness by reason of which perceptions cannot be organized; in other words, the synthetic faculty of the brain is weakened. Hence there result the various stigmata, such as extensive anæsthesia, paralysis, contracture, and loss of memory. In this weakened or hypnoid state a marked feature is the suggestibility to which Tourette refers as the essential characteristic of hysteria. From this purely psychological standpoint, as from no other, can be understood the phenomena of somnambulism, trance, and catalepsy which we see occasionally in the hysterical patient.[1]

It is needless to say that some of the traits of character so often ascribed to hysteria by former writers, such as simulation, sexual perversions, scruples, and morbid impulses, are the symptoms of degeneracy and not of hysteria. As Löwenfeld says truly, hysterical persons often fulfil their duties well as daughters, wives, and mothers, and are not always seeking to attract attention to themselves and their complaints.

Diagnosis. If, as is commonly claimed, hysteria simulates all diseases, it certainly differs from all in this very respect, that it has such a multiformity of symptoms. It is in the recognition of this multiformity that success lies in the diagnosis of the disease. I have endeavored to present a simple arrangement and concise description of these symptoms, and to show that they are not changeable and indeterminate, but that they are fixed in their characteristics and often of long duration. If a proper search were made for these stigmata we should hear less of the difficulties of diagnosis, and see less of a tendency to call hysteria by misleading names. Hysteria, instead of closely simulating all diseases, really simulates none exactly. The stigmata of the disease and their peculiar groupings ought to be unmistakable. No other method of successful diagnosis can be given but a reference to a study of them.

Nevertheless, there are some prevalent errors, as well as some moot points, that must be discussed briefly.

The most common vulgar error is to identify hysteria with the machinations of frauds and malingerers. The young woman who, decked in a bridal garment, fasts and is fed by stealth, the heroine of the story of the Cock Lane ghost, and the vixen who gives birth to frogs, are not hysterics—they are impostors. There is no more reason to call them, than to call any other jugglers, examples of this profoundly interesting psychosis, the very first essential of which is a series of mental impressions of undoubted originality and genuineness. Again, hysteria like any other disease may be feigned; but the counterfeit bears a superficial stamp; the malingerer has either no knowledge of, or no ability to ape, the permanent stigmata. An emotional crisis, a perverted temper, a dramatic outburst, or a crafty display, do not constitute the disease of which I am here writing.

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[1] For an elaboration of some of the ideas expressed in the text the reader is referred to Janet, "État Mental des Hystériques;" to some of Charcot's recent writings, and to the treatise of Gilles de la Tourette.

A more serious error, and also a not uncommon one, is to confuse hysteria and paranoia; in fact, the word "hysteria" has been used indiscriminately for all odds and ends of peculiar and perverted mental phenomena. However strange the case, it is enough to call it hysteria, and all is explained. Hence it is not uncommon to see the stigmata of degeneracy confused with those of hysteria; thus we may read descriptions of so-called hysterics, whose symptoms are described as having been those of the insanity of doubt, such as uncertainty and scruples; or mysophobia, agoraphobia, claustrophobia, pyromania, and kleptomania.[1] The instability of character, the weakness of will-power, the bizarre aspect of these cases of degeneracy is quite sufficient, in the opinion of some, to merit for them the term "hysteria." Still more heinous is the ascription of sexual perversion to hysteria, instead of to paranoia and degeneracy, to which properly it belongs. This error is no doubt a relic of that ancient pathology that attributed hysteria to disorders of the sexual function. In this connection it must not be forgotten, however, that hysteria may coexist with various forms of insanity, just as it may coexist with epilepsy or with organic disease of the nervous system. This conjunction, added to the confusion of the stigmata of degeneracy and hysteria, already referred to, has led to the belief in that somewhat hypothetical entity known as "hysterical insanity," a form of mental disease that no two authors describe alike. The truest forms of insanity in hysteria are the exaggerations of the prodromal symptoms of depression or exaltation into veritable melancholy or maniacal episodes, and the extraordinary development of delusional and hallucinatory states in the deliria of the grand attack. Beyond these it is difficult to understand what is meant by true "hysterical insanity" unless the term be applied to the cases of monomania or other psychosis, to which the stigmata of hysteria are added as a sort of epi-phenomena. But to call such a case of insanity "hysterical" would be no more proper than to call a case of tabes hysterical, because there happened to coexist with it some of the hysterical stigmata.

It is not unusual to ascribe some of the hysterical symptoms to that state called neurasthenia. In fact, neurasthenia, as usually described, is made to borrow largely from both hysteria and hypochondria. It were better if medical writers were always careful to distinguish it clearly from these two classical neuroses. This is not the place thus to distinguish it, but simply to state that the hysterical stigmata, when made to do duty for neurasthenia, are filched from their proper place. Neurasthenia means a state of malnutrition of the nervous system in which it is "without strength." To this meaning its application should be limited, but the tendency at present is too often to extend it almost without limit until it is made to include a confused medley of the symptoms of hysteria, hypochondria, dyspepsia, and chronic ill-health from various causes. This tendency is seen especially with reference to traumatic cases, all the hysterical stigmata of which are included in the more vague and more comprehensive term. Thus Löwenfeld (*op. cit.*) says that "our present knowledge indicates the resemblance of hysteria and neurasthenia in essential points." He seems to think it of small importance that some authors (apparently German) use the terms indiscriminately and interchangeably. All this is not to the advantage of consistency and clearness in scientific nomenclature. Hysteria, as has been shown, is a clearly distinguishable disease, and when its symptoms arise, from whatever cause, it should be called by its proper name. To distinguish it from other states of the nervous system, it is but necessary to study its symptoms. (See also page 77.)

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[1] As done by Magnan (quoted by Tourette).

The diagnosis of the so-called "traumatic neuroses" from hysteria has given, and will continue to give, rise to the most animated and unprofitable controversy. Hysteria, as has been shown in this chapter, numbers among its best recognized causes trauma. Hence it would appear easy, when the hysterical stigmata arise after injury, to ascribe them to their proper cause and to call them by their proper name. This, however, is not done willingly in all cases, with the result that we see most of the hysterical symptoms, such as localized pains, typical anæsthesias and paralyses, contractures and tremors, contractions of the visual fields, and paroxysmal phenomena, boldly made to do duty in a double rôle. It is needless to say that such a confusion in nosology is not defended here.

The hysterical convulsion sometimes simulates that of epilepsy. This is so especially when the grand attack is in part aborted, and the first period alone is prominent. The difficulty is greatly increased, of course, if the physician does not have the opportunity to witness for himself the seizure. The presence of some of the permanent intervallary symptoms is, of course, a valuable guide, but not to be relied upon exclusively, because in some cases hysteria and epilepsy may coexist, the patient having at one time an hysterical and at another an epileptic convulsion. It is rare, however, for the true hysterical convulsion to present the phenomena of only the first period; some of the features of the other periods, such as grand movements, passionate acts, gestures or attitudes, or a final emotional or even delirious stage usually presenting themselves. The prodromal stage, also, gives very important proofs of the hysterical fit; nothing like it occurs in epilepsy. Suggestion, or even full hypnotization, ought always to be tried in doubtful cases. Hypnotism, in fact, may supply a crucial test. If by its means it is possible to provoke a convulsion, this fact alone is almost proof positive that the fit is hysterical. Thus, too, the convulsion can be studied more at leisure. I have made satisfactory use of this test on more than one occasion in doubtful cases. The rules formerly laid down that loss of consciousness and the receipt of injuries were always proofs of epilepsy are of course not reliable. Loss of consciousness does not occur in all epileptic fits, and does occur in many of the major attacks of hysteria; while the receipt of injury is comparatively rare in epilepsy and not unheard of in hysteria. I would make exception, however, of biting the tongue, which is always very suggestive of an epileptic convulsion.

The diagnosis of the various stigmata, such as paralysis, tremor, astasia-abasia, hemianæsthesia, etc., from the organic affections that they resemble, has been indicated more or less clearly in the description of these symptoms. Hence they will not be taken up again seriatim. A general rule, however, should always be observed, *i. e.*, to look carefully for all stigmata, because it is by the peculiar grouping of these that hysteria often is characterized. Thus an hysterical hemiplegia, without facial palsy, is apt to be accompanied with hemianæsthesia, involving the special senses and the conjunctiva of the eye on the affected side; hysterical contracture of an arm is almost always accompanied with the peculiar segmental anæsthesia; an hysterical amaurosis or amblyopia with various cutaneous anæsthesias; astasia-abasia with anæsthesia; while the hysterical fit usually leaves as its vestiges some of the same symptoms.

Pathology. Hysteria cannot be said, in the ordinary sense, to have a gross pathology. What finer histological changes underlie the manifestations

of the hysterical diathesis it is not in our power yet to say. Tha
changes in the nerve-cells occur and are maintained for long per
be doubted when we consider the turmoil and excitement of
attack, or the complex and sustained phenomena of such symptom
hemiplegia, and contracture. That the nerve-cell in the cortex i
at fault is proved by the absoluteness of hysterical anæsthesia. It
however, to speculate upon these histological changes in hysteria
be in the cases of all psychic phenomena. The pathology of
among the unsolved problems of the infant science of psychology.

The nutritive changes that occur secondarily to the seizures
have been described among the symptoms of the disease.

Treatment. The treatment for hysteria ought to be of two
first is to be directed to the mental state, the second to the bodily
The first is vastly the more important, and, in fact, the success o
in the second class often depends in a measure upon the fact that t
strong mental impressions. In the first class, therefore, are includ
influences of a strong and appropriate personality in the phys
attendants, all moral impressions, all appeals to the emotions and
tion, all suggestions, and the skilful use of the association of ideas.
these influences can be grouped and discussed under the general ter
tism. This term has come to mean a rather elaborate ritual, whic
posed to symbolize some mystical influence. This influence, howeve
respects mystical, and it requires very few symbols or rituals. Th
of it is *suggestion*, and this has been used from time immemorial
skilled to treat the neurotic and hysterical. It is used, in some me
all successful physicians who aim to gain an ascendant influence
minds of their patients. It is not my intention here to discuss the
hypnotism in detail, but simply to indicate its bearings. Its es
already said, is suggestion, and this may be used in many cases wit
ceremony that is usually described. In other words, it can be used
influencing the patient's mind, gaining his or her confidence, and
by every means the firmness, tact, and knowledge of human nature
just as essential to the practitioner as they are impossible to be descr
is a mistake to suppose that all hysterical patients do not wish to
many will welcome and second a wise physician's efforts; they esca
their disease as from a bondage. Upon some minds the elaborate c
and profound impression of a genuine hypnotic *séance* do not act fa
Still, there are others in which it is advisable and permissible to gai
possible advantages of hypnotic suggestions by all the accessories
described. The hysterical mind often is very susceptible to the infl
hypnotism—that is to say, it is not difficult to bring the patient u
sway; when so influenced suggestions of the abolition of this or tha
tom at a certain time, or of the total restoration to health, are made.
cases this method is very successful. The temptation to experime
very susceptible patients, and to produce or abolish symptoms at will,
great, and ought to be resisted, because it may end in the more
hysterical perversion of the patient instead of his or her recovery.
tails of hypnotism the reader is referred to special papers and treatis

Hydrotherapy is of the first importance in the treatment of hyst
have seen more striking results from its use than from any one agent
from suggestion and moral influence. In fact, it is possible that som
efficacy of the hot and cold bath is derived from the powerful im
that this makes upon the patient's mind. Its roborant effect upon t
too, is very striking, and, as hysterical patients are not unusually in
tone of health than normal, it acts thus with direct benefit from the

side. A shrinking hysterical patient will naturally dread a heroic use of the bath, and on this account it ought to be administered with a firm hand by a nurse or attendant in whom the patient has confidence. Hence it can be used often to greater advantage in hospitals and in conjunction with isolation. The alternate hot and cold baths will produce a quick result in some patients, but if for any reason they are contra-indicated at first, a milder bath can be used, and the alternate effects can be secured gradually. A plunge or shower bath also can be given if practicable. I have had but little experience with sea-bathing in hysteria, but if given in the midst of proper attendance and environment I think that, combined with the change of scene that it usually entails, it would be highly advantageous in most cases.

After the bath dry friction should be used briskly to bring about reaction. It is very advantageous, in fact, to use systematic rubbing, or massage, in conjunction with the bathing. The rubber ought to have some special qualifications to do good work with an hysterical patient. An unpleasant personality, irritating qualities, inappropriate talk, and cold hands are graver faults than ignorance of the exact number of strokes and passes that custom dictates. In other words, the rubber ought to be in accord with the physician and attendants, and have intelligence enough to understand some of the mental characteristics of hysterical patients. Some of the best results obtained by me have been by having the rubbing done by a good attendant nurse, whose presence, touch, and conversation all *suggested* to the patient that she was expected to get well. Massage, in other words, may be made an avenue of approach to the patient's mind by which more wholesome and stimulating ideas may be introduced. This is of infinitely more importance than its purely physical effect as a mechanical aid to the circulation.

In order to establish and practise successfully the treatment here laid down it is often absolutely necessary to procure isolation. In many cases nothing can be done in the patient's home. The sympathy of friends, the demoralization of the household, the vicious association of ideas, must all be combated. They can only be met and their effects neutralized by removing the patient from their influence. The change of scene, the powerful impression made upon the patient's mind, the new and wholesome train of ideas suggested to the patient's thoughts by the removal to strange surroundings, and by the presence and control of strange and skilled attendants, all have a sovereign curing power, the credit for which is sometimes given exclusively to the physician.

Electricity, especially static electricity, has been highly lauded, especially by French practitioners, as a remedy in hysteria. Its effect is probably entirely psychical. It ought to be used cautiously at first, because it might excite the patient, and even fix more stubbornly some of the most obstinate symptoms. On the other hand, electricity acts sometimes marvellously on some particular symptoms. I once saw an hysterical paraplegia of long duration cured promptly with a small faradic battery. But such a case is an exception.

It is not necessary to discuss drugs in the treatment of hysteria; their office is a strictly secondary one. They may be required for the relief of special symptoms, and are to be given then according to the recognized principles of applied therapeutics. In general terms it may be said that all treatment should be sustaining and tonic; hence the drugs that meet this indication are to be preferred. I believe that sedative and depressing drugs ought never to be used in hysteria; they cannot control the symptoms, except temporarily, and they may even aggravate them permanently. The whole list of bromides, chloral, opiates, and antipyrin is to be condemned. Drugs are used sometimes by good practitioners for their moral effects, and, provided

the drugs are harmless, no harm can come. It is undoubtedly i
regulate the digestion and nutritive functions in hysteria—in a
the general health in the best possible condition.

Finally, among special therapeutic agents may be mention
therapy and the use of magnets, much advocated in France. B
cation of metals and magnets to the skin important effects may b
especially in anæsthesia. This symptom may be abolished or
from one side to another. It is enough simply to say that these
produced by suggestion and by influencing the imagination.

Surgery has but little, if any, field in hysteria. The excis
ovaries for pure hysteria is a barbarism founded upon the false
of the disease that was taught by the ancients and in the middle
a crime both against nature and against science. It is an operatio
has well said, that is not justified even by its successes. The doubt
remain in any given case whether a cure was not attainable by
and less unscientific means.

CHAPTER IV.

GENERAL MORBID STATES OF THE NERVOUS SYSTEM.

(CONTINUED.)

BY PHILIP COOMBS KNAPP, M.D.

NERVOUS AFFECTIONS FOLLOWING RAILWAY AND ALLIED INJURIES.

TRAUMA is classed as one of the causes of the majority of diseases of the nervous system. It may be either a direct or an exciting cause; but in many cases its influence must be regarded as slight. It does not materially modify the type of the disease in such cases, so that the affections themselves need no special etiological classification, and they, therefore, need no consideration here. There are, however, a number of obscure affections of the nervous system in which injury is a frequent and important cause, and the injury often gives a peculiar stamp to the affection, so that it is proper to group these affections under one heading.

It is probable that the traumatic nervous affections to be considered here have increased in frequency of late years. There has, of course, been an apparent increase by reason of our greater knowledge of their symptoms and our greater ability to recognize them; but it is also fair to suppose that there has been an actual increase on account of the enormous increase in railway travel, the introduction of the trolley and cable system of street railways, the increased use of powerful electrical currents and of many other modern inventions, all of which afford greater opportunity for accidents. In addition to these, the increased vulnerability of the nervous system is also to be considered.

The study of these affections has been rendered obscure from certain obvious causes. In many cases the victim of the injury has, or fancies he has, a claim on account of his injury against some corporation or individual for "damages," or against some accident insurance company for support. It is, therefore, for his interest to exaggerate his trouble, just as it is for the interest of the company to make light of it; and the physician, too often called merely as a partisan, is apt to take a biased view.

In consequence, however, of the medico-legal considerations which may arise, such cases have a peculiar importance. A single railway company doing business in Boston pays each year, on an average, more than $150,000 for damages for accidents. Out of one hundred consecutive cases of injury of all sorts claiming damages from this corporation as a result of their injury, fifty-six had symptoms referable to the nervous system. It will be seen, therefore, that a careful study of the various nervous affections is essential in order, on the one hand, that the corporations may not be defrauded by swindlers, and, on the other, that the victims of injury may not be deprived of their just claims by unjust charges of simulation or exaggeration. In injuries of other sorts there is comparatively little likelihood of such risks for either side.

Fractures, dislocations, lacerated wounds, and similar injuries can be readily recognized, and the dangers to life and health which they entail can be readily estimated. In nervous affections, where the knowledge of the average physician in this country is still so limited, there is greater room for error.

Before considering the various forms of traumatic affections involving the nervous system, it will be necessary to review briefly the various theories which have been held in regard to them.

The works of Erichsen,[1] which appeared in 1866 and 1875, may be taken as the starting point, not only because they are among the first works written upon the subject, but because they had for years a most important influence. Written by a surgeon, twenty years ago, when spinal anæmia, spinal hyperæmia, and spinal meningitis had an important place in nosology, before hysteria and neurasthenia were fully recognized or nervous pathology had made much progress, it is little wonder that Erichsen's theories as to the nature of the cases are to-day obsolete. Erichsen regarded the symptoms as due to concussion of the spine, and considered that the shock of the injury caused molecular changes in the spinal cord, which ultimately led to chronic spinal and cerebral meningitis, or that the shock caused anæmia or hyperæmia of the cord, especially in its posterior columns. Erichsen, however faulty his pathology may seem to-day, recognized clearly the existence of cerebral symptoms, of symptoms purely hysterical, and of symptoms clearly referable to injury to the muscles and ligaments of the vertebral column. In consequence of the unfortunate choice of the term spinal concussion to represent these various conditions, many writers have scouted his whole work and have imagined that, because they have proved that the spinal cord was not concussed, therefore the affection described could not exist—as sound a conclusion as it would be to deny the existence of hysteria in persons without a uterus.

In 1880, Hodges,[2] of Boston, emphasized the frequency of strain of the vertebral muscles, and of purely neurasthenic symptoms in many of the cases, and gave a much more favorable prognosis than Erichsen had done. In the following year Page[3] emphasized Hodges' conclusions that the majority of cases were simply muscular strain and neurasthenia, and that the spinal cord was in no way affected, except in cases of true violence causing fracture or dislocation of the vertebræ, with secondary laceration of the cord or spinal hemorrhage. Page furthermore tabulated two hundred and thirty-four cases which he had seen as surgeon of the Northwestern Railway in England, and claimed that the majority of them made a complete recovery within a year or two after their claims were settled.

In 1884, Putnam[4] and Walton[5] pointed out the existence of hemianæsthesia in certain cases that had come under their observation, and noted its resemblance to the hemianæsthesia observed in hysteria. Soon after Charcot began the study of hysteria of traumatic origin, and to his researches and the researches of his pupils[6] we owe our present knowledge of the subject. The greater frequency of hysteria in France and the interest naturally aroused by the work of Charcot have led the French to exaggerate the importance of hysteria and to regard most of the obscure forms of traumatic nervous disease as merely manifestations of hysteria.

Some years before this, however, Westphal[7] had advanced the hypothesis that, in some cases, the symptoms were due to small disseminated foci of inflammation in the brain or cord, and that they closely resembled the symptoms of disseminated sclerosis. In 1884 his assistants, Thomsen and Oppenheim,[1] made an elaborate study of sensory disturbances in the various forms of nervous disease. They disputed the French claim that hemianæsthesia was indicative of hysteria, and Oppenheim inclined to Westphal's hypothesis that definite structural change was the probable lesion in a majority of cases.

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[1] On Railway and Other Injuries of the Nervous System, 1866. On Concussion of the Spine, 1875.

[2] Boston Med. and Surg. Journ., April, 1880.

[3] Injuries of the Spine and Spinal Cord.

[4] Boston Med. and Surg. Journ., September, 1883; Amer. Journ. of Neurol., November, 1884.

[5] Archives of Med., 1883; Boston Med. and Surg. Journ., October, 1883.

[6] Charcot: Leçons sur les maladies du système nerveux, T. iii.; Leçons de mardi, T. i., ii. Also the works of Pitres, Gilles de la Tourette Janet, and Guinon.

[7] Charité. Annalen, 1878.

In 1889 Oppenheim published a monograph[2] embodying a further study of these conditions, and he and Strümpell[3] described them at length under the name of the traumatic neuroses. Abandoning in large part his former views, Oppenheim regarded them as functional in character, that is, as dependent upon no definite structural lesions. Although recognizing their close alliance to hysteria and neurasthenia, both Oppenheim and Strümpell have endeavored to establish a separate type under the heading of traumatic neurosis. Strümpell speaks of a "local traumatic neurosis," where the symptoms are limited to one portion of the body. Under this head he classes the monoplegias, contractures, joint neuroses, etc.; but most of these local neuroses must be grouped under the more general heading of hysteria. The general traumatic neurosis is, in Oppenheim's opinion, characterized by pain, mental anxiety and irritability, hypochondriasis, sleeplessness, tremor, paresis or paralysis, anæsthesia, contracted visual field, exaggerated tendon reflexes, and cardiac irritability.

Oppenheim's views have met with much opposition from the French writers, who claim that the conditions which he describes are hysterical. In Germany, too, since 1890, a considerable number of neurologists, headed by Schultze,[4] Hoffmann,[5] and Mendel, have opposed the establishment of a special form of disease known as traumatic neurosis, maintaining that these cases are hysteria or neurasthenia. They have also maintained that simulation was very common in these cases. This opinion, although long upheld by surgeons and general practitioners, had, up to that time, lacked support by neurologists, and even now comparatively few neurologists believe that successful simulation is at all common.

Since then many German observers have studied the individual symptoms in these cases (Wichmann[6] and others), especially the visual field (Wilbrand, Saenger,[7] and König[8]). The question of simulation has been thoroughly discussed, and the cases reported by Schultze, Hoffmann, and Seeligmuller[9] have been subjected to a destructive criticism. In this country Dana,[10] Seguin,[11] and Dercum[12] have studied the conditions and have endeavored to classify them; Walton[13] has made a further study of the symptoms, inclining to the belief in the frequency of simulation, and giving a favorable prognosis in the majority of cases; Clevenger[14] has attempted to group the symptoms under one heading, under the name of Erichsen's disease; and Dana[15] has recently published an admirable and exhaustive article upon the whole subject. The experimental and pathological researches will be spoken of later.

It seems better to differentiate the various traumatic nervous affections as much as possible rather than to group them under one general heading of traumatic neuroses. Cases where there has been some definite gross lesion of

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Archiv für Psych., xv.

Berliner Klinik, H. 3.

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Der Werth der Symptome der sogen. traum. Neurose.

Ueber Sehstörungen bei functionellen Nervenleiden.

Berlin. klin. Woch. No. 31, 1891.

N. Y. Med. Rec., December, 1884.

Therapeut. Gaz., May–October, 1889, and Am. Journ. Med. Sci., September, 1891.

Spinal Concussion.

Hamilton's System of Legal Medicine, ii, 297.

[2] Die traumatischen Neurosen. Second edition, 1892.

[4] Samml. kl. Vorträge, No. 14, 1890.

[9] Deutsche med. Woch., July–October, 1890.

[11] Annual Univ. Med. Sci., 1890, 1891.

[14] Journ. Nerv. and Ment. Dis., July, 1890.

the nervous system, the result of accident, such as crushing or laceration of the brain, cord, or peripheral nerves, hemorrhage, and the like, or cases presenting the symptoms of some well-recognized disease, differing from the ordinary type only in etiology, such as epilepsy, progressive muscular atrophy, transverse myelitis, and the forms of mental disease, will not be considered here.

Before dealing with affections involving the nervous system it will be necessary to consider one condition which often complicates many of these affections, namely, traumatic lumbago, after which the true nervous affections can be better appreciated, since the effect of the complicating symptoms of lumbago can be properly estimated.

Among the most important of the affections to be discussed in this chapter are two which are treated of elsewhere. Their importance and the fact that the traumatic origin gives them a somewhat different stamp warrants their further detailed consideration here. These affections are traumatic neurasthenia and traumatic hysteria.

Neurasthenia is one of the commonest of nervous affections, and it is also one of the commonest of traumatic diseases. It is the first manifestation of disintegration of the nervous system from any cause, and, as Dejerine[1] has shown, it may arise in the healthy organism and form the foundation upon which more profound degenerations may develop, either in the individual or in his descendants. Its origin, as will be shown later, is more often psychical than physical. With more profound disintegrating causes, or with causes acting on a weaker organism, there may develop an affection which betokens more pronounced disintegration of the nervous system, and which presents more marked symptoms of deficit, absolute loss of function instead of impairment, namely, hysteria. These two affections represent, not true diseases, but morbid states of the nervous system; they are allied, and the symptoms of the one shade into symptoms of the other, so that it is difficult to fix a definite boundary between them. As will be seen later, the majority of the cases of traumatic hysteria present distinct symptoms of neurasthenia, and many neurasthenic cases are also somewhat hysterical.

For reasons that will be mentioned later, it has not seemed best to accept Oppenheim's classification of traumatic neuroses as a separate affection; but, on account of the prominence given to it in literature, a brief description must be devoted to it.

In the section on pathology I shall state my reasons for rejecting the old distinction between functional and organic disease. There are probably fine cortical changes in cases of neurasthenia and hysteria. In the severer and more chronic forms of these affections it is probable that the changes become more marked. In another class of cases, where the physical injury is more pronounced, there are probably even greater changes in the central nervous system. I have, therefore, thought it justifiable to describe this class under the term of traumatic sclerosis, a term selected simply for convenience. In such cases I believe that there may be more marked changes in the brain and cord, either a diffuse or disseminated sclerosis. As the symptoms in these cases have considerable resemblance to the symptoms of hysteria and neurasthenia, it is not impossible that the changes in these two affections are similar although not so pronounced. In a limited number of cases the morbid process is located chiefly in the spinal cord, giving rise to a special form of disease which demands a separate description. This affection will be described as traumatic spinal sclerosis.

Etiology. The affections to be described may arise from injuries of various sorts. The injury, however, usually has this characteristic feature, namely, that it gives rise to a general jarring or concussion of the whole body, and that it is often associated with profound psychical disturbances.

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[1] L'hérédité dans les maladies du système nerveux.

Local violence is much less likely to give rise to general disturbance, unless there are coexisting psychical factors. In most cases it will produce only a localized injury to the nervous system at the seat of the blow, giving rise to the symptoms of a focal lesion. A severe blow on the head, however, may give rise to general cerebral disturbances, due, probably, to the general concussion of the contents of the skull. Blows upon the back, even of moderate violence, seem also peculiarly apt to produce symptoms similar to those due to general concussion.

It is obvious that the special accidents which may give rise to general concussion may be of all varieties—a fall on a sidewalk, from a ladder, down a flight of stairs, or from a carriage, any violent and severe blow, especially on the head or back, the buffetting of waves, electrical shocks, and, above all, railway accidents of various sorts. When the individual is exposed to great mechanical violence, as in railway accidents, the concussive force is naturally much greater than that from a simple fall, and the chances of injury are naturally increased. Even in the less severe railway accidents, the sudden stopping of a train may throw the individual forward with considerable force, and he may then be thrown backward with almost equal force when he strikes the seat or partition in front.

The amount of injury to the nervous system cannot, however, be measured by the intensity of the physical shock. In many cases another factor comes into play, sufficient of itself to produce serious results. This factor is the psychical shock. In simple falls upon the sidewalk or in accidents where the victim is at once rendered unconscious by a blow on the head, this factor is not material, but where there are terrifying or distressing circumstances attending the accident the emotional disturbance is much greater than the physical. Even to an onlooker a great railway accident, with the cries of the injured, the mangled bodies, and all the distressing circumstances, will produce a most profound psychical disturbance. When, in addition, there is the terror from the personal danger incurred it is not remarkable that, even without physical injury, the victim may be profoundly affected. Beside the general emotional disturbance the terror of the accident may, as Charcot has indicated, give rise to a mental condition resembling that of the hysterical state. The victim is dazed with terror; he is conscious of only a limited number of the phenomena of the external world; there is a clouding of consciousness; a limitation of the field of consciousness. This condition has been thought by Charcot to be closely akin to the somnambulic stage of hypnotism, and, just as in the hypnotic state a slight blow on the arm may give rise to an hysterical paralysis by suggestion, so, in this state, by a process of auto-suggestion, either as the result of a slight blow or merely from the idea of injury, hysterical paralyses and other hysterical symptoms may arise.

Oppenheim and Strümpell are disposed to lay chief stress upon these psychical factors as the most potent causes. While they are of great importance the influence of the physical shock is also great, and of late there has been a tendency to depreciate it. The psychical factor in the simple fall on a sidewalk is usually not great, for it is an accident which most men have repeatedly experienced without harm; the injurious effects are more probably due to the physical shock. When the victim is at once stunned by a blow there is no time for the psychical influences to take effect. In a railway accident, however, these psychical influences are of paramount importance. Physical injury is more likely to give rise to coarser structural changes—traumatic lumbago, and the traumatic scleroses; but when the injury is due chiefly to the psychical disturbances we are more li conditions of hysteria or neurasthenia. In many cases, howev factors, physical and psychical, coexist, so it is not easy to say w greater influence.

A word must be said in regard to predisposing factors. It is traumatic nervous affections in children, especially affections wh distinct psychical element, such as hysteria or neurasthenia. Th however, and several cases have come under my observation in chil fifteen years. Men, too, are much oftener affected than women, b are so much more exposed to accidents. Out of ninety cases o four were women, but out of forty-six cases of hysteria and n eighteen were women, showing, as might be expected, that the factors are more potent in women, and that accidents occurring are more prone to give rise to psychical affections.

The hereditary nervous taint is of considerable importance as a ing cause. A comparatively slight injury in the neuropathic ma to serious consequences, but the neuropathic taint is much more predisposing cause of hysteria or neurasthenia than as a cause of th

If there be pre-existing disease, especially structural disease of t system, trauma may greatly aggravate the symptoms. More th have seen patients in the initial stages of tabes or paretic dementia, still able to attend to the duties of life, rendered incapable of furth by a slight accident or by a moderate psychical shock, without sical injury. The injury seems capable of hastening the morbid p that the patient may soon after become ataxic or show so much m disturbance as to require restraint. These cases, furthermore, are i since, without careful inquiry into the previous condition, the physic erroneously ascribe the entire trouble to the injury. In tubercu especially in syphilis, the injury may act as an exciting cause for th tion of the disease in the brain or spinal cord.

When once the injury has been sustained there are other fact come into play which have a profound influence upon the progr disease. In most cases of illness the patient feels a natural anx whether he will recover, and as to how soon he can resume work an for himself and those dependent on him. This anxiety is usually nervous diseases, especially when they are chronic, for thepatient an exaggerated idea of their seriousness, and is not familiar with the outcome. In many cases of nervous disease the disorder itself giv anxiety and depression. In cases where a claim for damages is invol factors arise which are even more injurious. After a railway acciden ners for the accident lawyers sometimes reach the scene before the train and the surgeons, and, before the victim's wounds are dressed, th him that he has sustained incurable injury, for which he is entitled damages, and that they can procure them. From that time on the harassed with preparations for a trial. Instead of the absolute rest an from anxiety essential for recovery, he is exposed not only to the na inevitable anxiety and excitement resulting from his accident and hi but to all the worry and confusion of a suit at law—in itself sufficien great distress to a person in feeble health. Under the absurd syste perts" which exists in this country he is made to undergo repeated tions, often by men who show manifest bias and distress him by treati a swindler. In addition the lawyers and physicians, sometimes dir more often unconsciously, by repeated examinations and questioning symptoms, and in such nervous states, as is well known, the patie susceptible to suggestion. This state of things continues for a ye

fore the case is reached. Then comes the trial, with all its vexations and de-
ys, with renewed examinations and the anxiety of appearing in court as a
ness. After this some question of appeal may prolong matters for a year
r more. During all this time it only too often happens that nothing material
s done in the way of treatment. The attending physician is more apt to
pare the patient for the trial than to cure his ailment. The patient, thus
glected, cannot afford to get well. By doing so he will prejudice his case,
d, if he do not incur the odium of a swindler, he will at least receive a
ch smaller award. The anxiety about the claim and excitement of the
aminations and trial, the neglect of proper treatment, the evil results of
ggestion, and the consciousness that recovery would prejudice his interests
—all these, kept up for several years, naturally have a most deleterious influ-
ce, and they may easily convert a mild, curable trouble into a severe,
curable disease.

With the extensive use of electrical currents of high potential a new source
f accidents has been brought into daily life, and, as these accidents are be-
ming more frequent, the influence of electrical shocks in causing nervous
fections becomes important. The symptoms resulting from these shocks are
milar to those resulting from a shock from lightning. In fatal cases the
ief changes have been congestion in the nervous system with occasional
chymoses, and alterations in the blood, which is dark and fluid. There is,
rhaps, some chemical change in the blood, and Peterson[1] thinks that the
rrent divides and disarranges the molecular structure of the body. In
me cases Dana[2] maintains that there may be a disorganizing effect upon the
rvous tissues, causing paralysis from coarse lesions. Linemen, and others
ho work in dangerous positions, may be subjected to severe falls, from the
ct that even a moderate current may cause a sudden start; in other cases
he strong current causes severe burns without any other symptoms. With
rsons unused to electrical shocks the effect is chiefly pyschical. Electricity
s a new and mysterious agent. In the form of lightning it is most awe-inspir-
g, and it has, from the earliest times, given rise to many superstitions. The
merous fatal accidents from the currents now employed have given rise to
ch discussion in the press. Hence an electrical shock has a most powerful
fect upon the imagination even of educated people. As might be expected,
e nervous affections resulting from electrical accidents are chiefly of a neu-
sthenic or hysterical character. In cases where the symptoms could fairly
ascribed to the electrical shock and not to any attendant fall or burn,
arly two-thirds of those that I have seen have been cases of hysteria—a
ch greater proportion than is the case with accidents from other causes.

Pathology. It is unnecessary to cite cases showing the numerous direct
fects of trauma upon the nervous system. Among them we may note
morrhage, contusion, laceration, and softening, involving the brain and
rd, or the peripheral nerves. It is also generally admitted that trauma
y act as a direct or as an exciting cause of more chronic processes, such as
w growths and degenerations. The pathology in these conditions is well
cognized and the symptoms are usually well defined.

The pathology of the more obscure affections is still very vague. Although
has been much discussed, and although many pages have been written upon
it rests unfortunately upon a very slender basis of fact. The extremely
ll number of autopsies, considering the frequency of the affections, has,
times past, led certain writers to doubt the reality, or at least the severity,
the cases in question. The absence of autopsies seemed to afford proof
t all cases got well. The weakness of this argument, however, is exposed

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[1] N. Y. Med. Rec., Nov., 1889.

[2] Ibid.

when we consider that, in spite of the frequency and severity of neurasthenia not of traumatic origin, there are very few autopsies in this disease on record; yet many neurasthenic patients do not get well, and some of them must die from some cause or other, if not from neurasthenia itself.

Erichsen believed that where an accident had given rise to definite physical injury, especially fracture of a bone, the symptoms of concussion did not appear, that the violence expended itself upon the bone and did not exert injurious effects upon the nervous system. This opinion cannot be maintained, for, in a number of cases which have come under my own observation, nervous affections of various types have developed as the result of injuries which also caused fractures, either of a large or small bone.

A certain amount of work has been undertaken on the experimental side to show the nature of the changes which may result from concussion accidents.

Mendel,[1] believing that hyperæmia was an important feature of the early changes in general paralysis, sought to excite an intense chronic hyperæmia of the brain in dogs. For this purpose he fastened the animals on a revolving table, with their heads toward the periphery. Rapid revolutions, 125 to 130 a minute, continued for half an hour, produced punctate hemorrhages. Slower revolutions (110) for six minutes a day, produced, after some weeks, symptoms of general paralysis, and, on killing the animals, he found adhesions between the skull, the meninges, and the brain, an increase in the nuclei and cells of the glia, an increase in the number of vessels, and changes in the ganglion cells. Fürstner[2] repeated Mendel's experiments, with fewer revolutions (60 to 80), for a shorter time (one to two minutes), and continued for months. He found double primary degeneration of the lateral columns and of a particular part of the posterior columns, changes in the optic nerves, and changes in the brain similar to those found by Mendel.

Watson[3] carried on a rather elaborate series of experiments, the aim of which was to show that concussion accidents were not likely to produce injury of the spinal cord. He dropped dogs from a height of twenty-five feet, hoppling them so that in most cases the blow was delivered on the nates. From his experiments he concluded that concussive accidents never produce pathological changes in the spinal cord, except where great force has been applied to the spinal column, and those cases are generally, if not always, complicated with the fracture of a body of a vertebra, dislocation of the same, rupture or stretching of vertebral ligaments, or severe lesions in other parts of the body, which terminate quickly in death. The symptoms indicative of these morbid conditions are immediately developed, rarely become intensified by reason of morbid changes occurring in the spinal cord, exceptional cases being limited to fractures and dislocations, or those in which a slow hemorrhage occurs, causing pressure on the cord. Watson's conclusions, however, are weakened by the fact that he regards such changes in the cord as hyperæmia, punctate hemorrhages, and a granular appearance of sections, with indistinctness of fibres and a difficulty in making out the axis cylinders, as insignificant, and that in hardly a case did he keep his dogs alive long enough to determine whether any of the more chronic processes might develop. Schmaus[4] conducted a series of experiments on rabbits by placing a bit of wood over the spine and giving it repeated blows with a hammer. The vertebral canal was uninjured, but spinal symptoms were produced, and various changes were found in the cord—fine granular degeneration of the ganglion cells, swelling and enlargement of the axis cylinders, foci of softening, and fascicular degeneration. His conclusions are as follows:

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[1] Neurol. Centralbl., May, 1884.

[2] Arch. f. Psych. xix., 438.

[3] Experimental Study of Diseases Arising from Severe Concussions.

[4] Virchow's Archiv, cxxii., 326.

beginning and positive anatomical lesions.

3. Gliosis—termination in tumor formation.

These experiments, although interesting and to some extent suggestive, are by no means conclusive. Neither the rotation employed by Mendel and Fürstner, nor the concussion experiments of Watson or Schmaus, reproduce very closely the conditions that most commonly bring about morbid manifestations in man. Furthermore, the psychical factor, which undoubtedly plays a great part in many of the cases that occur in man, has no part in such experiments.

Human pathology affords some information with regard to the possible effects of trauma upon the nervous system. At the autopsies of several persons killed at a great railway accident at Charenton, Vibert[1] found very abundant punctate hemorrhages in the upper part of the body, and he suggests that they arose from lesions of the nervous centres. Willigk[2] found in one case dilatation of the finest vessels, with infiltration into the perivascular spaces, and degeneration of the coats of the vessels. Changes in the lateral columns have been found after death, in patients who had suffered from "concussion," by Dumesnil and Petel,[3] and also by Edes,[4] who has called attention to the occurrence of symptoms of spastic paraplegia in certain cases. Gowers[5] also has found, in a case where paraplegia developed some days after the injury, subacute myelitis in the thoracic region, chiefly in the white columns and greatest in the pyramidal tract.

Sperling and Kronthal[6] have reported a case showing melancholia, headache, mental impairment, muscular weakness, and circulatory disturbances, in which, after death, they found pronounced arterio-sclerosis, with spots of slight degeneration in the white matter of the cord, hemorrhage into the cord, and degeneration of ganglion cells in one portion of the cord. Both cerebral and spinal bloodvessels were sclerosed. Bernhardt and Kronthal[7] report another case, showing symptoms of hystero-neurasthenia, where the patient committed suicide by hanging. The cord, which alone was examined, showed disseminated foci which stained more deeply. Here the glia was increased, and some axis cylinders were lost; the vessel-walls were much thickened. Both these cases showed arterio-sclerosis and spots of slight degeneration in the white substance of the cord. Friedmann[8] found in the brain extreme hyperæmia, dilatation of the smaller vessels, with infiltration of their sheaths and proliferation of endothelial cells, and hyaline degeneration of the vessel-walls

The interpretation of these autopsies is still vague. Oppenheim[1] h degeneration in Kronthal's cases was merely the result of the art and he thinks that neither these cases nor Friedmann's can fairl establish the pathology of the traumatic neuroses. Friedman maintains that the source of the trouble in most of the cases i The concussion gives rise to a tendency to frequent fluxions with vasomotor regulating apparatus is weakened, and the nutrition o walls and their resisting power is at last impaired. Clevenger[2] ha without any definite pathological evidence, to refer the symptom these cases to injury of the sympathetic system and the consequen changes.

The notion of functional diseases as distinct from organic—th ease due to no structural change in the diseased organ—is fa obsolete. We are establishing the pathological changes of epile neuralgia, paralysis agitans, and other affections once classed as "Spinal concussion," paralysis due to molecular disturbances i without structural changes, has no place in modern pathology. concussion" is equally obsolete. In any fatal case of brain inj properly examined structural changes can be found. In neuras teria, and certain allied affections the notion of "functional di holds sway in many minds.

In hysteria the symptoms are chiefly of cerebral origin, and M Bastian,[4] among others, have attempted to apply the ordinary rul ization to hysterical anæsthesia and paralysis. For many yea have been made to refer both affections to alterations in the bloo the affected parts, and especially to vasomotor disturbances. In of traumatic neurasthenia and hysteria there are profound dist the circulatory system—a rapid pulse, palpitation, impaired circu diac distress, flushings, etc.—which, in connection with the scanty p evidence, would favor the theory of a vasomotor affection. This vasomotor disturbance, although not improbable, has little positiv as yet in its favor, so that we must regard it as still undetermined.

Although still in the very untrustworthy domain of speculative I cannot dismiss this subject without reference to certain experim seem to be of great significance as pointing out the way for futur In an elaborate study of the changes due to functional activity in Hodge[5] has found that in normal fatigue the nuclei decrease in s an irregular, jagged outline, stain darker, and lose their open, ret pearance; the cell-protoplasm shrinks in size, and shows vacuoliza spinal ganglia, or enlargement of the peri-cellular lymph-space in brum and cerebellum, and has a lessened power to reduce osmic the nuclei of the cell-capsule, where there is a capsule, decrease in size fatigue has a close kinship with neurasthenia, and it is not improba the latter affection, we may find changes in the nerve-cells of a simi of a more pronounced type.

Reasoning from analogy, therefore, it seems probable that in th functional affections we have at first cellular changes resembling scribed by Hodge. These may give rise to degenerative changes those described by Schmaus, or to secondary vascular disturbances, the severer and more chronic cases to more pronounced and mo sclerotic processes. Such a pathology is as yet largely speculat gaining consciousness after some interval. The unconsciousness is due to rect injury of the brain, probably contusion. With more serious injury the turn to consciousness is naturally delayed, and serious symptoms will be anifest when the patient comes to himself. In such cases it often happens at the patient has no memory of the accident or of events immediately eceding it. Such a condition is, of course, more often found in cases where here has been definite injury to the skull or its contents, such as fracture or rebral hemorrhage.

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[1] Op. cit., 2d ed., p. 176.

[2] Op. cit., p. 277.

[3] Centralbl. f. Nervenheilk., 1889, No. 12.

[4] Hysterical or Functional Paralysis.

[5] Journal of Morpho

In other cases the symptoms are those of ordinary surgical shock—pallor, intness, vertigo, a weak and rapid pulse, coldness of the extremities, nausea, omiting, etc. The faintness may go on to actual syncope, and the memory f events that happen during this period is much impaired. This condition ay last for several hours.

In other cases the symptoms of shock are slight or absent, but the patient thrown into a peculiar nervous state. He is apparently dazed, he performs rious actions correctly yet in a sort of automatic way, and he has no subquent consciousness of his acts. He may also forget events that happen mediately before the accident. This condition is closely akin to the somnambulic state of hypnotism, and its importance for the development of ggestion has already been mentioned.

In other cases still, the patient is not aware of the injury received; he may perience some slight pain, and feel a trifle dazed or faint for a moment; he en revives and goes on with his duties; in a railway accident he will relieve e injured, carry them from the wreck, and do many similar acts. After a rying interval symptoms develop. The symptoms of neurasthenia and steria are apt to come on suddenly: after the patient gets home and is free om excitement he may break down in a fit of hysterical weeping, or pass a eepless night, and the next day begin to complain. In other cases, especially th the scleroses, the symptoms are of more gradual onset. Local injuries y cause temporary disturbance, and, not until the patient recovers from m, do serious symptoms manifest themselves. There is seldom an interval more than two or three days free from any morbid symptoms, but it may a number of weeks before they become sufficiently pronounced to disturb patient materially or to demand the interference of a physician.

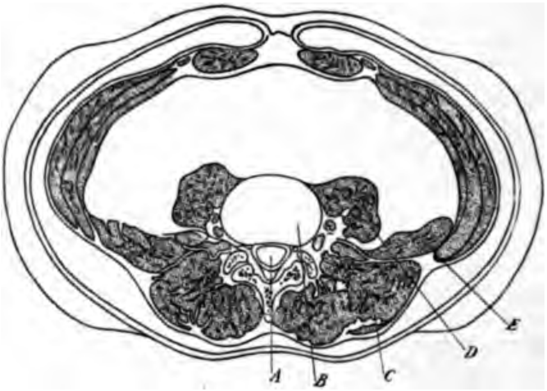
n rare cases pronounced mental symptoms appear soon after the injury. commonly there is a condition of extreme nervous excitement, insomnia,

TRAUMATIC LUMBAGO. (Strain of the Muscles of the Back.)

Although not properly an affection of the nervous system, traumatic lumbago may be described with the traumatic nervous affections, because it is a very common complication of such affections, and because it formerly gave rise to erroneous theories as to the nature of some of these affections. The pain and stiffness in the back which it causes have often been referred to diseases of the spinal cord.

The accompanying diagram (Fig. 40) shows clearly the size of the muscular mass which is attached to the vertebral column in the lumbar region, but it does not show the complexity of the tissues which make up this mass, which includes tendons, ligaments, fasciæ, and small bloodvessels and nerves, as well as muscular substance.

FIG. 40.



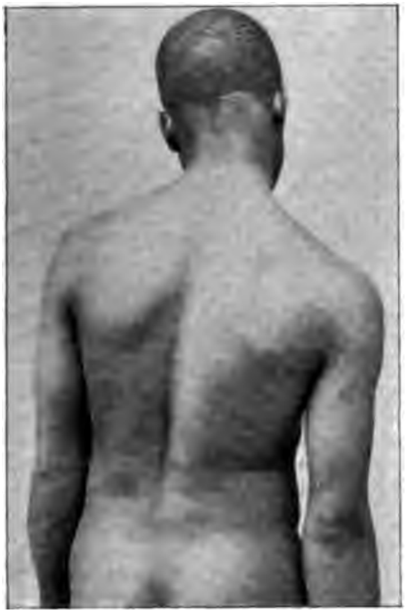
Horizontal section through the body at the umbilicus. (After BRAUNE.) The double line marks the boundary of the section. *A.* Cauda equina. *B.* Cartilage between the third and fourth lumbar vertebræ. *C.* Erector spinæ. *D.* Psoas. *E.* Quadratus lumborum.

Traumatic lumbago is due to physical causes, and it may arise either from a direct blow upon the back or from violence which leads to a twisting or wrenching of the trunk. It is, therefore, especially common in railway accidents and collisions, where the victim is tossed like a shuttlecock back and forth between the seats. Where the physical injury has been severe the victim usually shows the ordinary signs of surgical shock, or he may even lose consciousness. Rarely he may think himself at first uninjured, the symptoms developing after a few hours.

The striking feature of traumatic lumbago is pain in the back. The pain is usually moderate in severity, and seldom becomes sharp or lancinating while the patient is at rest. Any attempt at movement, however, increases the pain. The muscles are consequently kept by reflex action in a state of tonic spasm. The patient moves slowly and deliberately, in order to avoid any jar, the back is held stiff, and when he stoops there is little or no bending of the spine; he turns the head, but he avoids any twisting of the trunk; he often walks with his hand upon the back, protecting and supporting the painful part. Other muscles are apparently affected. The importance of the vertebral muscles in fixing the spine and affording a firm basis for other movements is seldom fully appreciated, but it is very great, and, as the vertebral muscles cannot be moved without pain, the limbs cannot act with their full strength, because they require the co-ordinated simultaneous action of the vertebral muscles to afford support for their extreme contraction. The legs, and, to a lesser degree, the arms, are therefore weak. Furthermore, as the vertebral muscles can no longer act properly, the action of the abdominal muscles is impaired, and the expulsive movements are less well performed, so that defecation and micturition are no longer performed naturally. There is a sense of greater effort, and it seems as if a greater strain were required. It is harder, too, to perform the last expulsive movements, so that there may be a little dribbling of urine. There is never, however, any involuntary discharge of urine or feces or any true incontinence.

The attitude and bearing of the patient are characteristic, from his effort to avoid any jar, bend, or twist of the spine. (Fig. 41.) If he be made to bend forward, or to one side, the rigidity becomes more manifest, and expressions of pain are at once elicited. In the severer cases the pain may be so great as to cause redness or pallor of the face, an outcry, an outburst of sweat on the forehead, faintness, or a quickened or weaker pulse. If the spine be rotated or jarred by any method, as by the physician clasping his hands over the patient's head and pulling down suddenly, the pain is even greater. The erector spinæ muscles may be felt tense and firm to the touch. There is no special sensitiveness of the skin, and the tenderness is usually more marked over the muscles by the side of the spine than over the spine itself. If the tenderness be at all marked, firm pressure on the tender spot will cause an increase in the rapidity of the pulse, and other manifestations of pain. (Mannkopf.)[1] Sensibility is unimpaired. The knee-jerk is somewhat exag-

FIG. 41.



Attitude of a patient with traumatic lumbago. (DERCUM.)

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[1] Centralbl. f. Nervenheilk., 1889, No. 12.

gerated, the movement is quick, but the excursion is short, as if hel The jar causes pain, and thus inhibits the movement. There ma evidences of exaggeration of the reflexes, such as front-tap contr the reflexes are not exaggerated beyond normal limits, and are n than we often see as a result of painful conditions.

In addition to these symptoms there may be various general sym to the pain and confinement, such as loss of appetite, constipation like. Owing to the difficulty of getting into a comfortable positi the pain on any slight movement, sleep is very apt to be disturbed general health may suffer in consequence.

Traumatic lumbago is not often an independent affection. It more frequently seen as a complication in various forms of traumati diseases, where it may obscure the clinical picture and add to the di diagnosis. In most instances, however, its presence can easily be d

The diagnosis is not difficult. Careful examination will reveal an affection, such as fracture, dislocation, or spinal caries. Neuralgia is by a more acute, shooting pain, often increased on motion, but co certain nerve distributions. Examination may reveal Valleix's p the muscular rigidity and tenderness are less pronounced.

In some cases of hysteria and neurasthenia there is very great pa back, usually described in much more emphatic terms than the pain matic lumbago. The patient says that a knife is sticking into the b the spine seems to be opening and shutting, and uses other extrava pressions. Motion and jarring aggravate this pain, too, so that t spasm of the erector spinæ muscles may be present. The pain is rather higher up than the pain of traumatic lumbago, or, at an shoots up the spine to become connected with the "pain at the ba brain" so often complained of. The spinous processes themselves extremely tender to the touch, but the muscles by the side of the s less sensitive. A slight touch usually causes more pain than deep The two conditions may coexist, the "spinal irritation" of neurasthen associated with lumbago.

The pathology of lumbago is quite unknown. It is assumed that tearing of certain muscular or ligamentous fibres, which is certai probable. In some cases there may have been a slight dislocatio vertebræ, or, more frequently, probably, an arthritis of some of th vertebral articulations.

In injuries involving the neck and the upper part of the spine we n similar symptoms in that region. In such cases the arms, as migh pected, show greater weakness than the legs, and there are often sy suggesting some injury or disturbance of the brachial plexus.

TRAUMATIC NEURASTHENIA.

One of the most frequent effects of severe injury upon the nervous is the production of a state of nervous weakness or nervous exha neurasthenia. In about one-third of all the cases of traumatic nerv ease coming under my own observation I have been led to consider t of nervous weakness as the chief trouble; but it often exists in co with other affections. In any case of serious disease, no matter nature or its origin, we are apt to see a condition of neurasthenia manifestation of general asthenia, although in many of these cases vous weakness is not especially prominent. So, too, in the majority matic cases a condition of neurasthenia is apt to supervene or to

Consequently it becomes a difficult matter to define its importance and to differentiate cases of pure neurasthenia from neurasthenia associated with other forms of nervous disease.

The causes of neurasthenia are various. Non-traumatic neurasthenia may arise from both physical and psychical causes; it may develop as a result of acute infectious diseases, gastric disorders, or uterine affections, or it may be due to grief, anxiety, or mental strain; often, indeed, the physical and psychical factors may be combined. The same is true of traumatic neurasthenia. In the stress that has lately been laid on the psychical factor in the etiology of traumatic nervous affections the physical factor has been somewhat slighted. Neurasthenia may follow an ordinary fall on a slippery sidewalk, or down two or three steps, where there has been no special psychical disturbance; but the most severe and pronounced cases usually arise where the psychical factors are greater. Some of the severest cases are to be seen in the survivors of great railway accidents, who may have suffered very little physical injury. The symptoms are often much influenced by the psychical factors. Neurasthenia may also develop secondarily as the result of the physical suffering from other injuries, especially when associated with the causes of anxiety and worry already referred to.

Von Hösslin[1] thinks that only persons with a predisposition to neuroses suffer from traumatic neurasthenia, but, in many cases, no evidence of predisposition can be found. The victims had previously good nervous organizations, with no history of inherited or acquired nervous taint. This is what we might expect, for neurasthenia is the most readily acquired form of nervous weakness, and it can hardly be considered a type of degeneration. The essential elements of traumatic neurasthenia are the same as those of other forms of neurasthenia. The weakened nervous system usually shows no definite loss of function, but it is incapable of persistent and prolonged effort. While the patient may perform many acts apparently as well as ever, they are done at a greater cost than in health, the subsequent exhaustion is more profound, and a week's suffering may pay for an hour's effort. With this the power of persistent mental and physical application is diminished. The neurasthenic cannot perform the ordinary daily routine of life, or do any work requiring persistent and constant application. The powers which at first seem normal are soon exhausted.

The second element in traumatic neurasthenia is that of irritability, often manifested as irritable weakness; the nervous system responds to weaker stimuli, and stimuli which are scarcely noticed in health give rise to unpleasant or distressing effects. This combination of an unduly irritable nervous system, with incapacity for protracted effort, is characteristic of all forms of neurasthenia.

The mental symptoms vary greatly. In cases due to physical causes they are less pronounced, but in cases due to psychical causes they may become one of the most marked and distressing features of the disease. The commonest and most persistent symptoms are nervousness and irritability. The patient starts at a sound; the slamming of a door distresses him; every slight, unusual noise makes him start; he is fretful and fault-finding; his children, of whom he was once devotedly fond, he dreads to have near him lest their noise disturb him; if he is able to be about he can no longer endure the petty annoyances of life; he is captious, querulous, and irritable; he can no longer get on comfortably with his fellows; in short, he presents the familiar picture of the complaining, irritable, nervous invalid. In cases due to railway accidents the victim often has a peculiar dread of railways; the whistle of the locomotive and the noise of the train may cause alarm, and he d into a train to take a journey.

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[1] Müller's Handbuch der Neurasthenie, p. 62.

In many cases mental depression is a very marked feature. sion is most frequently personal, referring to the patient's cha covery; he is often hypochondriacal. The introspection, com neurasthenic patients, leads him to dwell on his symptoms, to exa slightest ills, and to look with gloom and apprehension on the fut this may be coupled the depression that comes from fear of finan rassment or the dread of absolute penury. Some patients, howe markably brave in combating their ill-feelings; they force their the background, make light of their symptoms as much as they ca serve a calm, hopeful demeanor through it all, understanding the their symptoms, yet requiring close questioning before they will tel real sufferings. The depression may vary greatly in intensity, so experiencing sudden waves of intense depression which come suddenly and last for a considerable time.

In addition to these two striking symptoms of irritability and other mental symptoms are also seen. The spirit of doubt and h not uncommon. Every task, even writing a letter, seems Hercule is only after much effort that the patient can bring himself to un When it is done there may be questionings and doubtings as to performance. Certain insistent and dominant ideas may assert t such as are seen in the milder forms of the insanity of doubt. The anxious and apprehensive; he dreads solitude, the dark, a crowd, trains, but the dread is often more from association or from the f pending calamity to himself; the intention psychoses, such as ag are rare.

The mental powers are somewhat impaired. The power of mental application, as has been said, is diminished. Attention a hension are weakened. In the great majority of cases the patient is of doing anything very long; he rarely reads for more than a few n a time, and does not care to listen to reading much more than th patients read over one hour in the twenty-four, and that not cons In a part of the cases this is due to the fact that reading tires the e creases the headache. The judgment is seldom accurate; the pa neither think nor work so well, but the memory is not much impaire from pain, the patient may demand absolute rest, and therefore he r apathetic. True apathy, however, is of graver significance, and, wl ciated with pronounced loss of memory and actual mental impai points to more marked changes in the brain.

Emotional disturbances are common. One of the victims of a accident, whom I once saw, after working at the wreck attending the came home quietly, told his story calmly, and then burst into tears— manifestation of a profound psychical shock. Many neurasthenics to shed tears when any unpleasant or distressing subject is brough attention, and some show rapid transitions from tears to laughter.

One of the gravest and most distressing symptoms is insomnia. I thenia from physical injury it is less severe, but in neurasthenia from shock it may form one of the chief subjects of complaint. The patie to go to bed at all, and, although sleepy, he cannot get to sleep. the insomnia is due to the pain which comes from any unguarded m at other times it is due to extreme restlessness or to the psychical shock sleep comes it is attended with bad dreams, often referring to the but always of a distressing character.

SENSORY SYMPTOMS. One of the chief symptoms of traumati ence of neurasthenia intensifies all the symptoms. The pain is more intense, and often shoots all over the body; neuralgia is a common complication; the patient may describe the feelings in most graphic ways, as if an electric battery were moving up and down, as if the spine were pulled apart, or as if a red-hot iron were thrust into it. In other cases the pain is duller and of a more dragging character. In a few cases, especially if the injury be higher up on the spine, the pain is above the lumbar region.

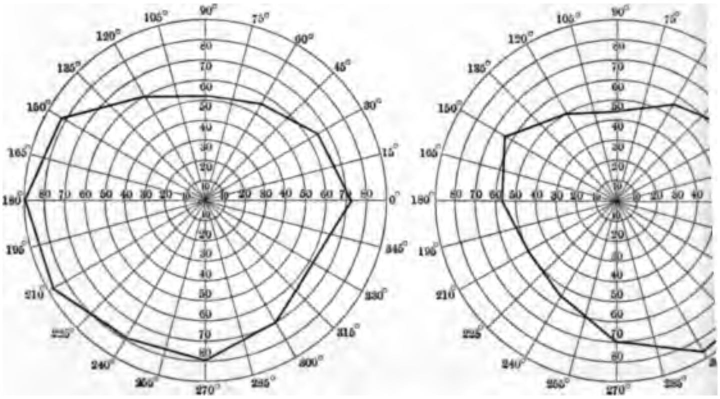
In addition to the pain there is pronounced hyperæsthesia. The vertebræ and the adjacent parts become exceedingly sensitive, not only to deep pressure, but also to a light touch. Sometimes the sensitiveness may exist only or deep pressure, sometimes only for touch. Touching or pressing the tender art is usually attended by an increase in the pulse-rate. Headache is common, and is increased by excitement or mental application. It is rather more common in the back of the head, the "pain at the base of the brain," nd often is of great severity. The pain in the back and head may incapacitate the patient for days, demanding absolute rest, quiet, and darkness.

Pains in other parts of the body are not uncommon. Many of them are a neuralgic character. If there have been a blow on any portion of the ody, this region is apt to be the starting point of pain.

Hyperæsthesia is another common sensory disturbance. The hyperæsthesia the back has already been mentioned, but it may extend over the whole dy or over definite portions. The nervous system, responding to weak imuli, may suffer pain from conditions that would hardly be noticed in alth. Coarse clothing, rumpled bedclothes, hard seats, and the like, may ve rise to actual pain. The hyperæsthesia may extend to the special senses; rong scents, noises, and bright lights increase the headache or the sense of dis- mfort. Paræsthesia of different sorts, tingling, burning, flushing, crawling, nd the like, are common. Anæsthesia does not occur. If we find any form anæsthesia we may conclude that there is some more serious disturbance the nervous system.

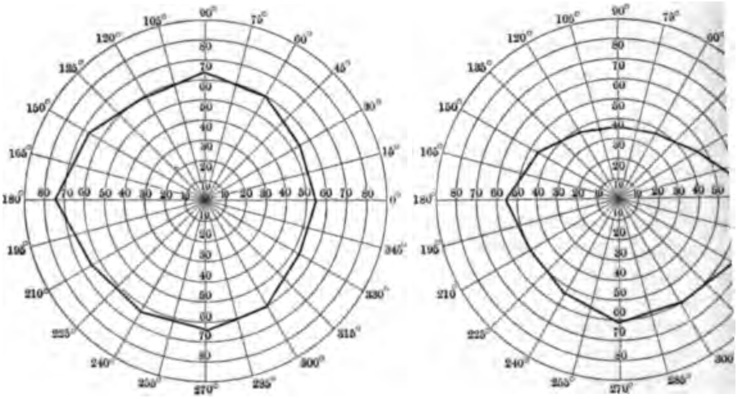
Of symptoms referable to the special senses, the visual disturbances are of e greatest importance. It may be said of these, as of other neurasthenic mptoms, that symptoms of deficit are not often met with. Asthenopia the chief trouble. In most cases central vision is unimpaired unless there e errors of refraction, but the visual power is soon weakened, and it may dly fall off if the tests be too long continued. The condition of the thenia, and Fig. 43, from a patient who presented distinct r symptoms without other hysterical stigmata, shows a distinct, thou great, contraction of the field.

FIG. 42.



Normal visual field in traumatic neurasthenia. (Personal observation.)

FIG. 43.



Contracted visual field in traumatic neurasthenia. (Personal observation.)

Various German observers (Förster,[1] König, Wilbrand and Säng lately called attention to a condition often observed in the visual neurasthenics, which indicates the fatigue which is so easily induced conditions. If the field be tested from the periphery to the centre, outward from the centre to the periphery, the second field will be fou the smaller. (Shifting type, Verschiebungstypus.) This is due to that the peripheral portions of the field speedily become fatigued. method of detecting this fatigue of the peripheral portions of the been described by Wilbrand and Sänger.[2] (Fig. 44.) The testing moved along the horizontal meridian of the perimeter from the temp to the nasal. At 0 the object first appears on the temporal side, ar disappears on the nasal side. It is at once moved back over the s ridian to the temporal side, where it will be found to disappear at some point nearer the centre, as 2. It is then moved back in the same way to the nasal side, when it disappears at 3, and so on. Another method, described by Förster, has been called by Schiele[1] the diametral method. The object is moved in on all the meridians of the temporal field, across to the nasal field, and the points of appearance and disappearance are noted. This may give a field similar to that inclosed by the unbroken line in Fig. 46. After a period of rest the process is repeated on the meridians of the nasal field, and a field similar to that inclosed by the dotted lines may be observed. The limitation of the field on the side toward which the object is moved is due, of course, to the speedy exhaustion of the retina. Tests made in these various ways thus afford a very trustworthy indication of the easily induced fatigue of the retina. It is needless to say that such tests require the use of the perimeter.

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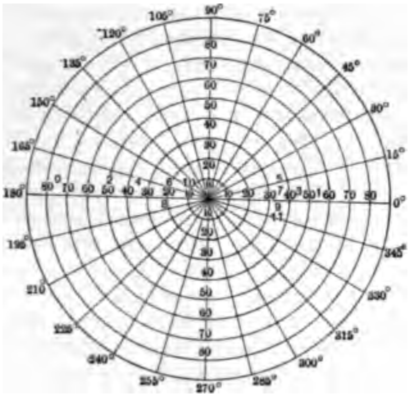
[1] Wilbrand und Saenger, op. cit., p. 15.

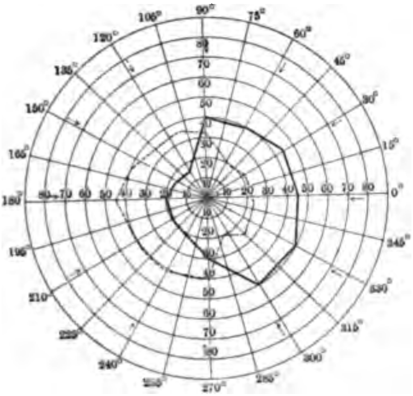
[2] Op. cit., p. 10.

FIG. 44.



Ready test for contraction of the visual field from fatigue. (WILBRAND.)

FIG. 45.



Contraction of the visual field from fatigue. Diametral method. (WILBRAND.)

In addition to these sensory symptoms referable to the eyes there are also certain indications of motor weakness. On closure of the lids it be noted that the closure is incomplete, and that the lids constan The pupils are often large, and react to very slight stimuli; at t light is thrown on them, they contract at once and then dilate a though the light is still thrown on them, contracting and dilating This may happen independently of light or accommodation. W has been slight hypermetropia or astigmatism, compensated by the accommodation, the patient will complain of a failure of vision, weakness of the muscles of accommodation, which has been first by the injury. Insufficiency of some of the external muscles of the cially the internal recti, may also be noted, but there is never any

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[1] Arch. f. Augenheilk., xvi.

Aural symptoms are less pronounced. The chief complaint is o hyperæsthesia. Even the slightest noise in some cases seems to agg headache and the various pains, while loud, sudden noises, such as ming of a door, prove very distressing. Some patients, too, are susceptible to sounds of a certain character. One man under m tion, who was much disturbed by noise, could still listen to music of character, but he could not bear to hear singing by a soprano voice, himself any high notes, although he could sing and enjoy singing in register. Baginsky[1] has found that perception of the whispered vo the tuning-fork was diminished in some cases, and that the sound o was not so well perceived by bone-conduction. This he ascribes to cause, although he does not absolutely exclude labyrinthine affectic turbances of smell and taste have also been noted, chiefly in the hyperæsthesia, the patient being much disturbed by certain odors o

MOTOR SYMPTOMS. The motor disturbances of neurasthenia re many respects the sensory. Symptoms of absolute deficit, such as pa not occur, but symptoms of weakness are very common. In traum rasthenia muscular weakness is the rule. In some non-traumatic cas the symptoms are chiefly cerebral (cerebrasthenia), Löwenfeld claims muscular strength may be unimpaired; but that must be rare, and matic cases wholly exceptional. The degree of muscular weakness i greatly, from inability to walk across the room to merely an impair to walk long distances. The gait may present nothing unusual, unl often the case, there be also a traumatic lumbago, when the char features of that condition are added. In some cases the condition tenderness ("spinal irritation" of old writers) may lead the patient similar precautions against sudden movements or jars. The gait may guarded, feeble, and somewhat uncertain from weakness, but there is ataxia. When asked to stand with the eyes closed and the feet close it may be noted, first, that it is difficult to make him close the ey lutely, and, second, that there may be considerable swaying, althoug so pronounced as in tabes.

In many cases there is manifestly diminished muscular strength, a by the dynamometer, but in other cases the weakness is manifested ra the lack of endurance. The dynamometer needle can be forced u average point, but it cannot be kept there long; the patient can pu heavy a dumb-bell as ever, but he cannot hold it up as long; if th of muscular work be taken on a registering apparatus it may rise n high, but it will fall off much more rapidly. Oppenheim[2] has emph condition which must have often been noted before, and which is not mon, where, although the patient apparently makes a profound eff calls many muscles into play, the effect of the movement is but sli often suggests the attempt to make a brave show of effort without accomplishing anything, and hence it is erroneously regarded as indicative of simulation, but Oppenheim rightly regards it as due to a loss of memory of the requisite division of motor impulses in order to execute a given movement.

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[1] Löwenfeld, op. cit., p. 151.

[2] Op. cit., p. 148.

The quivering of the eyelids has already been mentioned. In some cases we may also note a fine tremor of the lips, and, more especially, of the hands. This is never very marked in ordinary neurasthenia, and it does not impair the finer movements of the hands very materially. It may show itself somewhat in the handwriting, and it is then more marked at the end of a letter than at the beginning.

REFLEXES. As might naturally be expected from the nature of neurasthenia, the reflex activity, as a rule, is everywhere exaggerated. This, however, is less noticeable in the skin reflexes than in the deep reflexes; in fact, the former are, as a rule, not much exaggerated. The exaggeration of the deep reflexes is seen most markedly in the knee-jerk. Not only is the kick more vigorous when tested in the usual way, but the knee-jerk may be elicited in various ways not usual in health. Thus a movement may be elicited by striking the tendon of the quadriceps above the patella, or by striking the broad upper part of the tibia. If, while the leg is extended and relaxed, the finger be placed over the upper edge of the patella and the patella be drawn downward toward the foot, and if then the finger be struck a sharp blow with a percussion hammer in the direction of the traction, a twitch of the quadriceps may be obtained (patellar twitch). Gowers' front-tap contraction is often present, but a true patellar or ankle clonus is not found; on testing for them the muscles may respond by several contractions, but not by persistent, rhythmical clonus. The reflexes never differ on the two sides. In some cases, especially when there is coexisting traumatic lumbago, the knee-jerk has the characteristics already described; it is short and quick, and the excursion seems suppressed; the attempt to elicit it apparently causes pain, probably from the slight jar.

TROPHIC DISTURBANCES AND ELECTRICAL CHANGES. The trophic disturbances in traumatic neurasthenia are of slight importance. The chief change to be noted is the change in weight, which is usually dependent upon the condition of the appetite and digestion. Other changes seldom occur. The hair may fall out or turn gray, the skin may become dry, or there may be profuse sweating, but such symptoms are rare.

Changes in the electrical excitability of muscles and nerves have been but little studied. Rumpf[1] has claimed that in the later stages of some of the traumatic affections there is a quantitative reduction in the galvanic excitability of motor nerves, kathodic closure contraction requiring a current of four to ten milliampères, instead of one or two. This has not been confirmed by other observers, and in the healthy subject I have frequently found that a stronger current than two milliampères is required to produce contractions. Rumpf also claimed that after faradization of a nerve trunk the contraction was followed by large fascicular and fibrillary contractions lasting several seconds, another point which most subsequent observers have failed to confirm. If this be true, however, it is of some value as indicating the increased irritability of the nerve trunks.[2] Mann has claimed recently that in various forms of "functional" brain diseases, including neurasthenia and the so-called traumatic neuroses, there was a diminution of the electrical resistance of the head. By placing a large electrode (5 to 10 centimeters) on the back of the neck and a similar one on the forehead, and using a few cells, he found that normally the resistance was from 4000 to 6000 S. E., but that in the traumatic neuroses it fell to 1500 and 2500 S. E. Basile[1] has also found similar diminution of resistance in the spinal region in cases of so-called spinal neurasthenia. If this reduction in resistance be confirmed it may perhaps be explained by supposing that the vasomotor disturbances, which are not uncommon, have given rise to an increased cutaneous blood-supply, and thus to a lessened cutaneous resistance. Qualitative changes and degenerative reactions do not occur.

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[1] Deutsche med. Wochen., 1890, No. 9.

[2] Berlin. klin. Wochenschr., 1893.

RESPIRATORY AND CIRCULATORY DISTURBANCES. The respiratory disturbances in traumatic neurasthenia are of slight consequence. A rapid respiration and slight dyspnœa on exertion are very common, but, as a rule, they are to be referred to cardiac irregularities. A weakness of respiration associated with general weakness is of course common. This may manifest itself in the voice, which is often low and feeble, conversation requiring much effort, and occasionally being attended with sighing or gasping for breath. In one of my own cases, where neurasthenic symptoms were prominent, there was a curious gasp or sob in the breath as the patient spoke, a catch, as if the effort caused great pain, yet really not due to pain. Such spasmodic respiratory disturbances however, are rare.

The circulatory disturbances are of great importance, and, as has been noted in the section on pathology, they probably have some bearing on the nature of the affection. One of the striking features of traumatic neurasthenia is the rapidity of the pulse. In twenty-five cases it ranged from 72 to 140, the average rate being 95, and in only three cases did it fall below 80. In many cases the pulse is weak, irregular, or intermittent. Any slight exertion or excitement will send it up to 150 or more. This rapidity of pulse is not the temporary quickening often seen in nervous patients from dread of an examination or fear of detection of simulation; such a quickening is merely a transitory affair, and very soon subsides; but the increased rapidity in traumatic neurasthenia is permanent and is to be found on repeated examinations. In a few cases, after this rapidity has continued for a year or more, it has been observed that hypertrophy and dilatation of the heart set in and that insufficiency of the valves developed with the usual symptoms (Oppenheim).

Various subjective symptoms may be referred to the heart. Palpitation, shortness of breath, pain in the præcordium, and the feelings of distress that so often accompany nervous disturbances of the heart, are common. At times this distress may increase to severe paroxysms of cardiac anxiety, but this is, in my experience, rare. Oppenheim has noted arterio-sclerosis in a number of cases, and this has been found by Kronthal in the two autopsies already cited.

Symptoms of defective or irregular circulation are also frequent. The extremities are cold, and it may be harder to keep the patient comfortably warm. At times the skin may be so congested as to be quite cyanotic, and Kriege[2] has noted symptoms of Raynaud's disease. In addition to this, patients often complain of flashes of heat, flushings, congested feelings, rush of blood to the head, etc. Slight irritation may lead to persistent redness of the skin. Occasionally there may be profuse perspiration, so that the extremities are cold and clammy.

DIGESTIVE DISTURBANCES. Digestive disturbances are common, but they seldom dominate the picture. The commonest symptom, as in other forms of neurasthenia, is more or less loss of appetite. This is often due in part to an absolute disinclination for food, and in part to a dread of the distress which the ingestion of food may cause. In a few cases there is inability to retain any solid food, and the patient is obliged to live on small amounts of some prepared liquid food, such as beef extracts or partly digested milk. In other cases the stomach can retain food fairly well, but there is considerable distress after eating, a sensation of a weight in the stomach, gastric pains, eructations, flatulence, or occasionally pyrosis. Partly from muscular weakness, partly from inactivity, constipation is not infrequent; in a few cases there may be nervous diarrhœa. As a result of the diminished amount of food and the imperfect digestion the bodily nutrition not infrequently suffers.

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[1] Neurol. Central., 1884, p. 118.

[2] Arch. of Psych. xxii, 241.

URINARY SYMPTOMS. There is rarely any disturbance either of the urine or of micturition in uncomplicated neurasthenia. Many patients complain either of an imperative need of making water as soon as the desire comes, or of slowness or weakness in emptying the bladder. In most cases this is due to a complicating lumbago. General weakness may lead to some enfeeblement of the stream and perhaps to a slightly imperfect expulsion; there may be a slight dribbling after the act, but there is never any real retention or incontinence.

The urine itself shows few changes. In rare cases the amount may be increased or the need of passing it may be more frequent. Hyperacidity, phosphaturia and oxaluria have been noted by some writers, but their significance is not great. Glycosuria is rare, but it has been observed by Eisenlohr and Oppenheim.[1] Albuminuria is also a rare symptom which has occasionally been noted. Transitory albuminuria may occur in health, as is well known, but a permanent albuminuria, unless it be due to some foreign cause, such as coexisting nephritis, would probably exist only in those cases where there is passive congestion of the kidney from the enfeebled heart—at least such was the probable explanation in the only case in my own experience where albuminuria occurred.

SEXUAL SYMPTOMS. In the majority of the cases of traumatic nervous disease where there is any claim for damages much stress is usually laid upon the impairment of the sexual functions. For obvious reasons, the sexual power cannot well be tested by the examining physician, and we can decide definitely as to permanent impotence only where there are definite symptoms of disease of the spinal cord destroying or inhibiting the sexual centre. The sexual power, however, is influenced greatly by the physical condition and by many psychical causes, so that it might well be expected, when such etiological factors are present as have been mentioned above, and when the general health is suffering from any form of disease, that the sexual power would be lost or else much impaired. This loss or impairment, however, is not necessarily permanent, and unless there be a cord lesion it will usually disappear with the restoration to health.

In traumatic neurasthenia the disturbance of the sexual function is also manifested, as might be expected, by symptoms of irritable weakness and of exhaustion. In the milder cases one may see too frequent pollutions or premature ejaculation; but, as a rule, there is a distinct failure of power; desire is usually much impaired, and, as a rule, desire and power both disappear entirely.

In women disturbances of menstruation are not uncommon. The physical concussion in any traumatic case may lead to local disturbances, especially uterine displacements, or in pregnant women it may cause abortion. Such disturbances in themselves may give much trouble and may aggravate the condition. In other cases, owing to the general hyperæsthesia, there may be increased pain at the menstrual period and, not infrequently, great irregularity in the occurence of the function. There may be a cessation of the

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[1] Op cit. p. 174.

menses for several months, or profuse flowing at the periods.
borne in mind that an injury may start up troublesome symptoms
existing uterine fibroid that previously has been unnoticed. In
menstruation is unaffected, and conception, pregnancy and partu
go on normally.

TRAUMATIC HYSTERIA.

As Strümpell[1] has shown, hysteria must be reckoned as a mor
stage in the process of disintegration of the nervous system th
thenia, although not necessarily so grave a condition. It is chara
more profound symptoms: where neurasthenia shows only a weakn
may show an absolute deficit, as in paralysis; where neurasthenia m
increasing motor excitability, hysteria may show distinct motor di
in convulsions. There is, however, no distinct line of division b
cases. In the majority of neurasthenics we may note the emotional
once thought characteristic of hysteria, and in any collection of ca
that the boundary between the two morbid states cannot be distinc
Neurasthenic symptoms are often to be found in cases that prese
symptoms of hysteria. This is not to be wondered at, for neith
thenia nor hysteria can be regarded as definite diseases, but rather
states of the nervous system, due chiefly to cerebral disturba
generally considered to be chiefly mental affections.

The essential characteristics of neurasthenia have already been
The definition of hysteria is much less easy. It is something m
disease of representation or ideation, or of reduplication of person
limitation of the field of consciousness. Perhaps the best definiti
recently elaborated by Janet:[2] "Hysteria is a mental disease bel
the considerable group of diseases of degeneracy; it has only v
physical symptoms, consisting especially in a general diminution of
it is characterized especially by moral symptoms, the chief symptom
enfeeblement of the faculty of psychological synthesis, a limitation o
of consciousness. A certain number of elementary phenomena,
and images, cease to be perceived and seem suppressed from pers
ception; these constitute the stigmata. From this there follows a te
the permanent and complete division of personality, to the for
several groups of phenomena independent of one another. These
of psychological facts alternate with one another or coexist, wh
rise to attacks, somnambulism, or unconscious acts. In short, this
synthesis favors the formation of certain parasitic ideas which are c
developed independently of the control of personal consciousness,
manifested by the most varied disturbances, apparently purely physi
is to say, they are manifested by hysterical attacks."

It will be seen that Charcot's theory, mentioned above—that t
hysteria is developed by auto-suggestion, the patient being in a som
state, a theory afterward made more general by Moebius,[3] who
hysteria was a disease of representation, is applicable to only a pa
phenomena. Janet, Jolly,[4] Oppenheim,[5] and others have shown th
the fixed ideas which may well give rise to certain symptoms, there
conditions which demand some other explanation, such as the s
which persist when the subject no longer thinks of them, the atta
nambulism, etc.

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[1] Spec. Pathologie u. Therapie; seventh edition.

[2] État mental des hysté

[3] Centralb. für Nervenheilkunde, Feb., 1888.

[4] Berlin. kl. Wochenschr., 1892, No. 34.

[5] Charité Annalen. 188

Hysteria is distinctly less common in America than it is in France. The pronounced stigmata, hemianæsthesia, contraction of the visual field, etc., are not very common in the clinics for nervous diseases in this country with which I have been familiar, except among the Russian and Polish Jews, and the typical attacks of grand hysteria are distinctly rare. Traumatic hysteria is relatively more common. About twenty per cent. of all cases of traumatic nervous disease of which I have record have been classed as hysterical, but this is a very liberal percentage, as many doubtful cases have been included.

Heredity seems, in the cases that have come under my own observation, to be of less importance in the etiology of traumatic hysteria than in the idiopathic forms, but Bataille,[1] Charcot and others have laid much stress upon an hereditary taint. Dana[2] has found traumatic hysteria to be less common in women than in men. Such has not been my own experience. Thirteen out of twenty-five cases of traumatic hysteria under my own observation occurred among women, yet, as I have said before, less than one-third of all cases of traumatic nervous disease that I have seen have been women.

In the majority of cases of traumatic hysteria other symptoms have been noted, such as have already been described as neurasthenic—headache, backache, insomnia, depression, a rapid pulse, dyspepsia, nervousness, irritability and incapacity for protracted effort. To these are added the special stigmata of hysteria. In other words, we rarely meet with hysteria of a pure type, it is usually combined with neurasthenic symptoms—hystero-neurasthenia.

The mental symptoms of traumatic hysteria differ considerably from those of ordinary hysteria. The old ideas that the hysterical patient is a deliberate, conscious liar, simulator and *poseur* can no longer be admitted. The apparently voluntary theatrical demonstrations, exaggerations and misstatements have been shown to be merely the results of unconscious suggestion, often arising from hallucinations, and as little blameworthy as the delusions of the paranoiac. The characteristic feature of the hysterical mind is its exaggerated suggestibility; to the suggestions that arise from other people or from the patient's own dreams, to hallucinations and morbid ideas in general, may be traced many of the phenomena presented by the hysterical, especially the ideas and actions which, in old times, were attributed to demoniacal possession, and in our day are still too often regarded as affectations or falsehoods.

In traumatic hysteria, however, the suggestibility, the modifications of personality, the exaggeration of the ego, the posing, and the divisions of personality, catalepsy, trance, double consciousness, and the like, seldom predominate. As has already been said, the great majority of cases are associated with neurasthenia, and, as Charcot[3] has shown, the mental symptoms of neurasthenia are the first to show themselves, especially in the male, and they may remain dominant throughout. Charcot speaks of this step as a "period of neurasthenic preparation."[4] We note the irritability, the depression, the aversion to society, the introspection, the hesitancy, the inability for protracted mental effort, and the occasional emotional instability, already described. The insomnia and the bad dreams also exist. Later on, but comparatively rarely, however, the hallucinations of hysteria may become manifest.

From the clinical point of view, the hysterical stigmata are of the first importance in the diagnosis of hysteria and in differentiating it from neurasthenia and other conditions, so that they must be first described.

SENSORY STIGMATA. *Anæsthesia.* Anæsthesia in some form is one of the commonest stigmata of hysteria. Tactile sensibility is most impaired. The tactile sensibility is most easily tested by to skin lightly with the finger-tip or a camel's-hair brush, the pat being blindfolded or closed by the thumb and finger of the other hand. The various æsthesiometers, which try to show at wh the patient can distinguish two points from one, have comparat value. The degree of anæsthesia may vary greatly. In a fa hystero-neurasthenia with hemianæsthesia the patient could disti lightest touch on the anæsthetic side; but it was not felt so disti the other side. There are all varieties between this and anæsthe lute that the patient can feel no sort of sensory stimulus, not even lus of the strongest faradic current conveyed through a single nee electrode.

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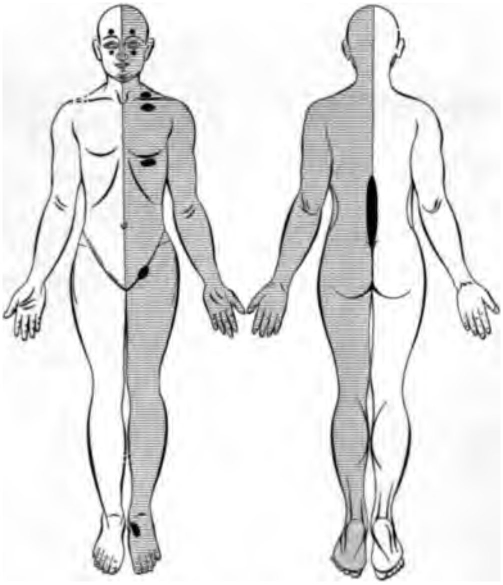
[1] Traumatisme et Névropathie.

[2] System of Legal Medicine, ii., 335.

[3] Leçons du Mardi, 1888–1889.

[4] Gilles de la Tourette. Traité de l'hystérie, p. 548.

FIG. 46.



Hysterical ▤ *Hemianaesthesia*, with hyperalgesic zones. (Personal observati

The distribution of the anæsthesia may vary very greatly. Gene thesia as a permanent stigma is very rare, Briquet[1] finding it c times in two hundred and forty cases. The commonest form, in m ence, is hemianæsthesia, which may be complete or partial (Figs. 46 As a rule, hemianæsthesia is sharply limited at the meridian line, boundary may be a little to one side. Briquet regards the diss islets of anæsthesia, of which a remarkable specimen is shown in Fi the commonest form; but I have rarely encountered it, either in t or non-traumatic cases. A fourth form is the geometrical anæsthe 49), which may involve various segments of the body, covering the stocking, a glove, a sleeve, or a helmet. This distribution of anæsth been shown by Charcot and Horsley[2] to be characteristic of cortic thesia, and in hysteria we never find anæsthesia limited to the distrib any special nerve or sets of nerves.

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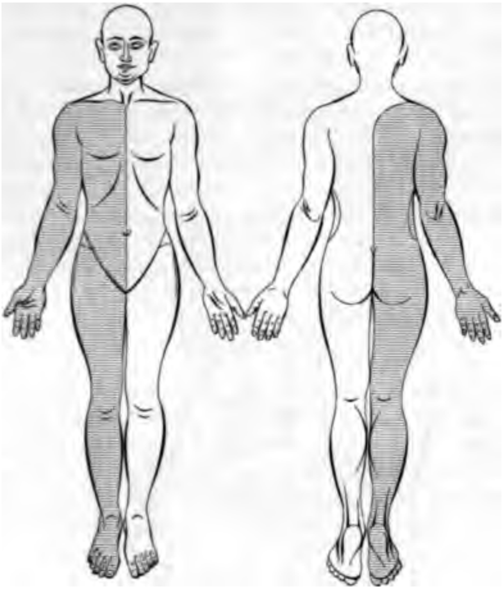
[1] Gilles de la Tourette. Op. cit., p. 126.

[2] Trans. Am. Cong. Phys. Sur

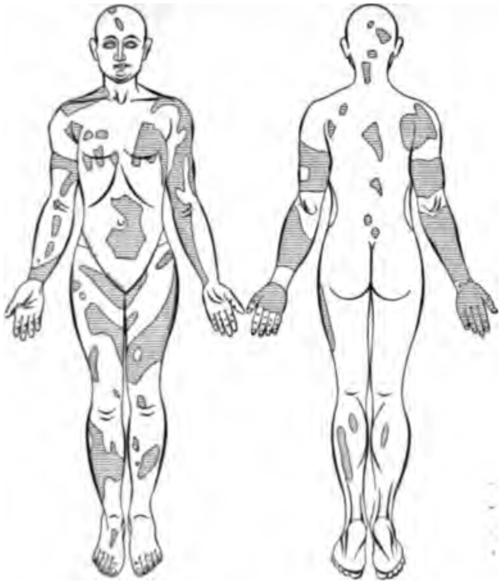
Head[1] has recently attempted to divide hysteria into two forms, a psychical form, where the anæsthesia follows natural lines, as in the geometrical anæs-

FIG. 47.



Partial ▤ *Hemianaesthesia*. (Personal observation.)

FIG. 48.



Islets of ▤ *Anaesthesia*. (PITRES.)

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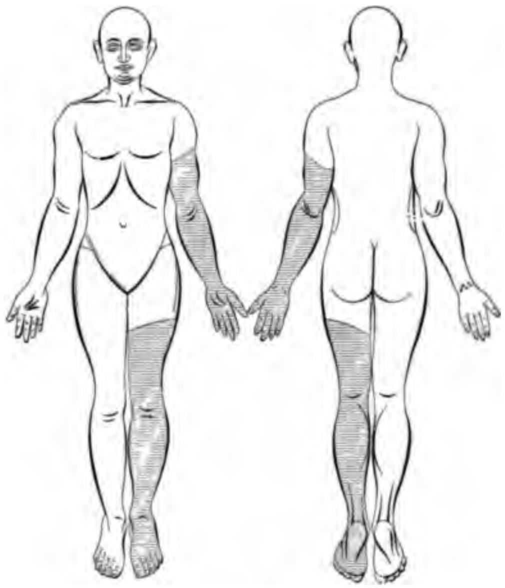
[1] Brain, xvi, 1.

thesia already described, and the cerebro-spinal form, where tl temperature senses especially are lost in the early stages in areas ing to the spinal segmentation. In the psychical form the skin retained, and the visual field contracted. In the cerebro-spin: reverse occurs. This latter form Head considers to have a wors and to be more likely to be confused with disseminated sclerosis.

FIG. 49.



Geometrical ▤ *Anaesthesia*. (Personal observation.)

The anæsthesia may be of varying extent. In some cases the de are involved, and the anæsthesia extends to the mucous membranes a loss of sensibility to all forms of stimulation, pain, temperature, the sensibility of the muscles and joints is impaired. In other case anæsthesia only to certain forms of stimuli—partial or dissociated a The commonest form of partial anæsthesia that I have noted has loss of sensibility to pain (analgesia), the sensibility to touch r normal. There may also be a loss of sensibility to temperature anæsthesia), or loss of sensibility to electricity—electro-anæsthesia, merely a form of analgesia. In fact, analgesia may be estima exactly by placing a wire brush, or, better, Erb's sensory elec different parts of the skin, applying the faradic current, and n coil distance at which the first sensation of pain appears. Analg however, be most readily determined by pricking with a pin. anæsthesia may be tested by tubes filled with warm and cold wa metal cylinders warmed and cooled. Goldscheider[1] has given very methods for testing the temperature sense, which, however, dema time and patience. His tests, however, may be of use in doubtful c

These various forms of sensory disturbance are apt to be pretty but they may suddenly change, and they may be modified in vari

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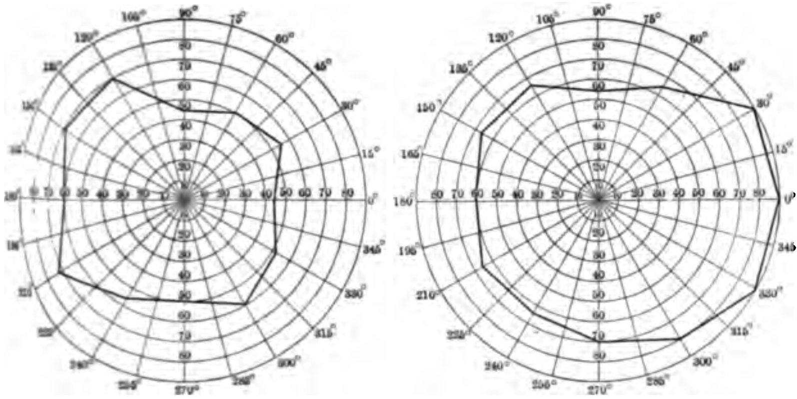
[1] Archiv f. Psych., xviii.

In cases of hemianæsthesia, for instance, the anæsthesia may be transferred to the other side of the body by the application of a magnet or of certain metals. It is probable, however, that this transfer is due solely to suggestion, for recent researches have cast grave doubts on the possibility of any influence being exerted by these agents where suggestion was absolutely excluded.

In many of the forms of hysterical anæsthesia the special senses may also be involved. In hemianæsthesia it is not unusual to have a loss of taste on the corresponding half of the tongue, of smell in the corresponding nostril, of hearing in the corresponding ear, and of vision in the corresponding eye. In hysterical deafness, which may be of varying intensity, it has been found that, as might have been expected, the trouble was of central origin, as indicated by Rinne's test. In this test the tuning-fork, which has been made to vibrate, is placed upon the mastoid. When the patient no longer hears the sound it is moved in front of the ear. In deafness of central origin the sound is heard again when conducted through the air; but in deafness from disease of the middle or external ear it is heard longer by bone-conduction.

The most important disturbance of the special senses is that of vision. After excluding the cases of impairment of vision, where there are errors of refraction, there are some cases where there is complete amaurosis, usually in the eye on the anæsthetic side. In the majority of cases, however, there is not complete amaurosis, but a concentric limitation of the visual field, often most marked on the anæsthetic side (Fig. 50). In the cases that have come under my own observation this limitation of the field has not been very great, but in some cases it is extreme, as shown in Fig. 51. With the contraction of the field may often be noted a change in the relation of the color fields. Normally, the area in which blue can be detected is the greater; red next, and green the smallest. These fields may also be contracted, and the field for red, for instance, may be greater than that for blue. The perception of certain colors may be impaired (dyschromatopsia), or the perception of color may be wholly lost (achromatopsia).

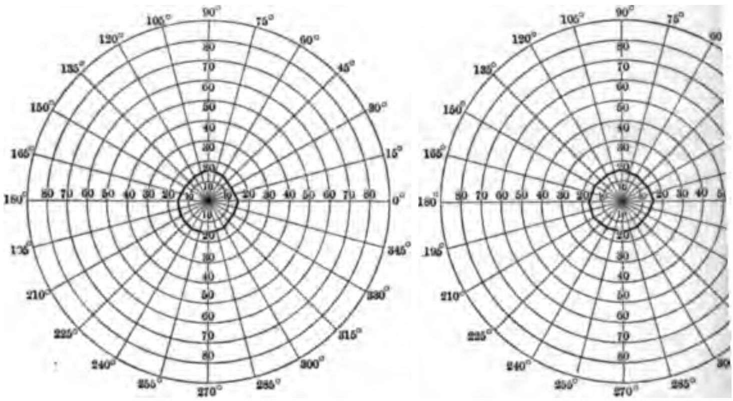
FIG. 50.



Irregular contraction of the visual field in one eye in traumatic hysteria. (Personal observation.)

It is a matter of great importance, in view of the frequent claims that the patient is a malingerer, to bear in mind that these sensory disturbances of hysteria, the anæsthesia and the amaurosis, are purely psychical in character, and that, in spite of the actual inability of the patient to feel o can be demonstrated that she actually does feel and see. Pitres[1] quite conclusively that hysterical amaurosis exists only on visioı affected eye. If, for example, the left eye be blind and a screen vertically between the two eyes parallel to the axis of vision, tl with the right eye closed, sees nothing; and, with the left eye cl letters only on the right of the screen; but, with both eyes opeı read the letters across the page, including those to the left of ı which could be seen only by the left eye. Janet[2] has also show interesting experiments, confirmed in this country by Prince,[3] and l observation, that in cutaneous anæsthesia the patient actually fee If the anæsthetic part be touched or pricked while the patient is b

FIG. 51.



Marked contraction of both visual fields in hysteria. (GILLES DE LA TOURETT

she does not feel it; but if, later, the patient be hypnotized, or if her self" be questioned by means of automatic writing, she will repeat ac where, and how many times, she was touched. One of my own pati a marked diminution of color-sense in one eye saw colors plainly w eye when a prism was used in such a way as to give crossed imag right eye being affected, she saw the colors of the left-hand image c although actually seen with the right eye. In the same case, with ı anæsthesia and loss of muscular-sense in one hand, the patient co wink and grasp my hand with her hand when her eyes were closed cases are of much importance in the study of the nature of hysteria, l are also of great importance as proving the great liability to error who, ignorant of hysterical peculiarities, are prone to find simulation case.

It is important to determine whether the definite sharply-defined ana either in the form of hemianæsthesia or geometrical anæsthesia, is pa monic of hysteria. It is beyond a doubt that in the great majority where we find such a distribution, especially if associated with cor visual fields, the case is hysteria; but the researches of Thomsen and heim[4] have yet to be contradicted, and they have found that hemian may occur in other affections. Contracted visual fields are not alway hysteria. They are seen in the early stages of optic atrophy and in nervous affections.

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[1] Leçons clin. sur l'hystérie, i., 193.

[2] L'Automatisme psychologi

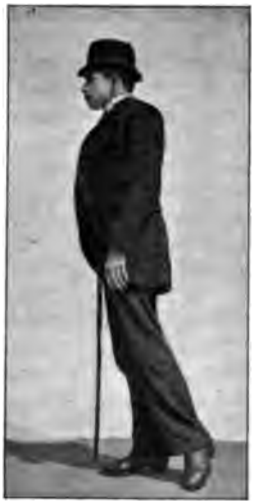
[3] Boston Medical and Surgical Journal.

[4] Arch. f. Psych., xv.

Hyperæsthesia. Hyperæsthesia, or, to speak more correctly, hyperalgesia, since there is usually increased sensitiveness to pain rather than an increased sensitiveness to touch, is not uncommon. In some cases it may take the place of anæsthesia, and may be distributed in the same way, but it is more commonly limited to certain regions. If the injury has implicated any special region it may exist in that region, and it may exaggerate or prolong the tenderness due to the local disturbances caused by the physical injury. It is thus often seen in the hysterical joint affections, which will be described later. In other cases the hyperæsthesia develops spontaneously, and is manifested with some constancy in certain regions, forming the so-called hyperæsthetic zones. Of the relation of these zones to hysterical attacks I will speak later. The most constant seat of the zones is in the ovarian region (Charcot's "ovarie"), but they are not uncommon in the epigastrium, over the vertebræ, near the breast, and occasionally elsewhere. Fig. 46 shows their distribution in one case. Another important form of hyperæsthesia is the well-known clavus or localized intense pain in hysterical headache. The tenderness over the vertebræ is allied to the "irritable spine" of neurasthenia, and it may also be associated with traumatic lumbago. The reality of the hyperæsthesia may often be demonstrated by the increase in the frequency of the pulse when pressure is applied. In some instances a light touch seems to produce more pain than deep pressure. Pitres[1] has described a rare symptom, haphalgesia, where the contact of certain metals caused great pain.

MOTOR SYMPTOMS. *Paralysis.* In traumatic hysteria absolute paralysis is less common than anæsthesia. Muscular weakness, however, similar to that described in neurasthenia, and local paresis, is not uncommon. The distribution of the paralysis may vary very greatly. In traumatic cases hemiplegia and monoplegia are the commonest forms. It is characteristic of hysterical hemiplegia that the leg is dragged stiffly up to the sound leg, instead of being circumducted, as in hemiplegia, from cerebral hemorrhage (Fig. 52). The face is not involved, but a spasm of the muscles on the other side of the face may suggest facial paralysis. In the same way there may apparently be in rare cases a paralysis of the sixth nerve, or external ophthalmoplegia; but Gilles de la Tourette,[2] on careful analysis, has shown that in most of these cases the trouble is not a real paralysis, but a spasm. It is of importance to note here that in these apparent oculo-motor paralyses the voluntary movements only are affected, the eye moving normally in unconscious movements.

FIG. 52.



Gait of a patient with traumatic hysterical hemiplegia. (Personal observation.)

It is a valuable diagnostic feature in hysterical paralysis when the paralysis is also associated with anæsthesia. The paralysis may be flaccid or spastic. In rare cases, as has been noted by various French observers, there may be muscular atrophy with the paralysis, not merely the atrophy of disuse, but also atrophy with electrical changes.

Hysterical paralysis is never limited to the distribution of a single nerve; but, like all cerebral paralyses, it affects definite movements or groups of

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[1] Op. cit., i., 65.

[2] Op. cit., p. 415.

movements. It may attack a limb in segmental fashion, like am
it may affect only certain movements. Bremer[1] has reported a c
terical astasia-abasia attributed to a very slight accident. The
such cases can move the limbs perfectly well while in bed, with go
and good co-ordination, but he is wholly unable to stand or walk
cases, while unable to walk, patients can hop on one foot, or w
tragic stride, or in some peculiar way.

Contractions. Hysterical contractions are often associated witl
but, except in the hysterical joints, they are not common in traun
French writers describe, as not uncommon, the "diathesis of coı
where a comparatively slight irritation (a bandage, a blow on the
nerve, the application of the faradic current, the magnet, or me
produce a pronounced contraction of the limb. Such a condition
is distinctly rare in hysteria as seen in this country. When c
occurs it is apt to be well-marked, unlike the very slight rigidity
movement sometimes seen in the early stages of hemiplegia.

Tremor. It is only in comparatively recent years that tremor
recognized as an important symptom in hysteria, but Charcot[2] has a
it is common in men, and more especially in hysteria of traumat
The tremor may be persistent or paroxysmal, general or localized
pronounced, and of variable rhythm. Dutil[3] has, in fact classi
according to the rapidity of the rhythm, but Charcot classifies the
ing to the character of the tremor. Tremors which are exagge
voluntary movements may be slow (three to six a second) or rapid
nine or more a second). The former often resembles the tremor o
or of paralysis agitans; the latter is like the tremor of exophthalm
or alcoholism. Another class of tremors may or may not exist durii
but they are true intention tremors, movement increasing the amj
the vibrations rather than their rapidity; this resembles the trem
seminated sclerosis and of mercurial poisoning. In the cases of l
tremor that have come under my own observation this seems to be
monest form. It is sometimes seen associated with paralysis, in t
lyzed limb.

Spasm. In addition to the contractures and tremors not uncoı
hysteria, we may also note various forms of spasm in the traumati
well as the non-traumatic forms—the various forms of tic, the aı
choreic movements, which have been described as electric, rhythı
saltatory chorea, saltatory reflex spasm, paramyoclonus multiplex,
like. These conditions, however, are distinctly rare in traumatic
and do not differ from similar conditions in non-traumatic hysteria,
described in detail in Chapter III.

THE HYSTERICAL ATTACK. The striking feature of the hysteric
is the spasmodic seizure, which may assume the form of the sever
ical convulsion with its classical stages of epileptiform convulsi
tortions and grand movements, plastic poses and passionate attit
delirium. Motor spasm, however, is not an essential feature of th
In the broadest sense the hysterical attack may include the paro
laughing and crying, the spasmodic coughs and other respiratory
ances, attacks of vertigo, cardiac anxiety, and the like. The ordin
vulsive attack seldom manifests the various periods of the grand att
is often preceded by some irritability, and it may manifest itself af
emotional disturbance. The attack is often preceded by unpleasaı

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[1] Journal of Nervous and Mental Disease, January, 1893.

[2] Progrès méd., 1889, 1890.

[3] Nouv. Icon. de la Salpêtrière, 1890, 1

tions, such as palpitation, headache, vertigo, gastric distress, or the familiar globus; and it may be of varying intensity, from a slight weakness or faint feeling with tremor, up to a pronounced convulsion with exaggerated and disturbed movements. In the majority of cases there is probably some slight clouding of consciousness, which may go on to absolute loss of consciousness. The classical attack is extremely rare in this country in any form of hysteria, and the attack in any form plays a very subordinate part in traumatic hysteria. Charcot and other French observers have noted the severe attacks, with pronounced convulsions, the *arc du cercle*, and similar manifestations in a number of cases. Pitres[1] found the attack less common in male hysteria, occurring in only seven out of thirty-one cases, while of sixty-nine women fifty-six had attacks. In the cases of traumatic hysteria seen in this country, however, I have never found the classical attack with its four periods, nor even the severe convulsion, and in less than ten per cent. of the cases of hysteria was there anything that could be regarded as even a mild attack.

The relation of the attacks to the so-called zones is extremely interesting. In many cases it has been found that pressure over certain regions is sufficient to give rise to an attack, and these regions are called hysterogenous zones. These zones are apt to correspond with the hyperæsthetic areas already described, and their most common seat is in the ovarian region, over the spine, under the breasts, and in the epigastrium. Fig. 46, although showing merely hyperæsthetic areas and not hysterogenous zones, shows the favorite location of these zones. Charcot long ago demonstrated that pressure over the ovarian region might also cut short an hysterical convulsion. Further research has shown that there may be various "hystero-frenic zones" in other parts of the body, but they are less often seen than hysterogenous zones. In traumatic hysteria in this country such zones are rare, and pressure on the hyperæsthetic areas has in no case under my own observation produced any symptoms except those indicative of pain, such as an increase in the pulse, faintness, sweating, flushing, etc.

HYSTERICAL JOINT AFFECTIONS. The hysterical joint affections, first described by Brodie,[2] are usually of traumatic origin. They seem rather less common than other forms of traumatic hysteria, and are much more common in women than in men. Out of seventy cases reported by Charcot,[3] the knee was affected in over one-half, and the hip in over one-fourth. The striking symptom of an hysterical joint affection is pain, which may be severe when the joint is at rest, and always becomes severe on any attempt to move the joint, and also on any pressure about the joint. The pain may be confined to the joint, or it may extend to parts above and below. In addition to the pain there is often pronounced hyperalgesia, sometimes being very marked in the skin covering the joint, and sometimes extending to the soft parts surrounding it. This tenderness is usually much greater than the tenderness of the joint itself; the ends of the bones may be pressed together with much less pain than is caused by handling the soft parts. In some cases the hyperalgesic region may be bounded by an anæsthetic area. With the pain and tenderness comes disability. The patient cannot move the joint on account of pain, and with the pain there is often weakness. The tenderness on motion gives rise to a reflex spasm, so that there is often a pronounced contraction of the muscles about the joint; in the hip, for instance, we may observe an apparent contraction, with adduction and rotation inwards. In rare cases there may be some muscular atrophy. In a few cases the joint may be swollen.

It is probable that in many cases of hysterical joint affection there is at first some trifling lesion in or about the joint which gives rise to pain. As any attempt at moving the joint gives rise to pain, the ideas of motion and pain become so firmly associated that finally the idea of motion may be sufficient to give rise to the idea of pain, even after the physical source of pain has disappeared. Prince[1] has shown that, by introducing the idea of some unusual motion in the joint, the new motion, which has never been associated with any idea of pain, may be performed without pain, and that thus the morbid association may gradually be broken up.

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[1] Op. cit., i., 207.

[2] Lectures Illustrative of Certain Local Affections.

[3] Leçons sur les maladies du système nerveux, iii., 370.

Hysterical joint affections are apt to be obstinate, and they may last for years. Cases that recover may suffer from relapses. The diagnosis must depend on the presence of other hysterical symptoms, the absence of swelling, the character of the pain and tenderness, and the influence of psychical factors. Charcot has claimed that anæsthesia by ether or chloroform will at once cause the rigidity and contraction to disappear, and that this is a valuable diagnostic criterion, but it must be remembered that anæsthesia will also cause the contraction and rigidity to disappear in the early stages of actual arthritis, although they persist in the later stages, so that the distinction is by no means absolute.

Reflexes. The examination of the reflexes in traumatic hysteria usually gives valuable confirmatory evidence of the conditions existing. Where there is anæsthesia the skin reflexes are either diminished or wholly absent in the anæsthetic region, even in cases which in other respects correspond to the "psychical form" of hysteria described by Head. The tendon reflexes are, as a rule, exaggerated, as they are in neurasthenia. In the majority of cases of hysterical paralysis, and occasionally in simple anæsthesia, the reflexes are greater in the affected part, but in a few instances of hysterical hemiplegia they have been found more marked on the unaffected side. The loss of the pharyngeal reflex is often noted, and it is important in diagnosis.

The other symptoms of traumatic hysteria are of less importance. Trophic disturbances are not very common. Muscular atrophy may be associated with hysterical paralysis or with the joint affections, usually the simple atrophy from disuse, but, more rarely, a degenerative atrophy with the degenerative reactions. The electrical changes mentioned under neurasthenia, as described by Rumpf and others, apply to hysteria as well as to neurasthenia, but their value, as has been said, is still uncertain. Œdema, including the "blue œdema" described by Charcot, and various cutaneous changes may occur in traumatic hysteria; but they are rare, and do not differ from similar conditions observed in the non-traumatic form. In one case the hair over a limited area on the scalp which had been the seat of severe pain began to grow out white, and when the patient came under my observation about an inch at the root of the hair was white, the rest of the hair being dark. It became entirely white, and then turned back to the natural color in the same way.

Respiratory and circulatory disturbances resemble those of neurasthenia already described. In one case, observed by one of my colleagues, there was extremely rapid respiration, 120 to 180 a minute. This persisted for some time, and then was temporarily cured after praying in a certain church. The rapid pulse and the circulatory weakness are very common.

The digestive disturbances in traumatic hysteria are also like those of neurasthenia. The typical hysterical anorexia and vomiting are rarely seen, but the loss of appetite may be great, and in a few cases the stomach can retain but little food.

We seldom see the anuria or polyuria so often described and occasionally met with in non-traumatic hysteria. As a rule the urinary symptoms are similar to those described in neurasthenia, and are of comparatively slight significance. Gilles de la Tourette and Cathelineau[1] have found in non-traumatic hysteria without attacks that the urine was normal as to amount and solid constituents. After an attack, however, the amount of urine passed in the next twenty-four hours is not increased, but somewhat diminished, the solid constituents (urea and phosphates) are one-third diminished, and relation between the earthy and alkaline phosphates, which is normally one to three, rises from one to twc, or even to equality.

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[1] Journ. Nerv. and Ment. Dis., May, 1891

THE "TRAUMATIC NEUROSES."

In view of the great prominence which this term has recently acquired in all discussions of the subject since the publication of Oppenheim's monograph, it is necessary to devote a brief space to a consideration of it.

Strümpell[2] states that in a part of the cases the trauma has merely a local action, and the nervous symptoms which result from it are also localized in the arm, the leg, etc. For such cases he uses the term "local traumatic neurosis." Under this head he classes the local hyperæsthesias, anæsthesias, paralyses, contractures, and the hysterical joint affections, excluding, of course, any affections due to material lesions of the peripheral nerves. As Strümpell himself admits that these cases are all local manifestations of hysteria, which is, of course, a general disease, the justification of the term seems questionable.

It is somewhat different with the "general traumatic neuroses" which he describes, and which have been more fully studied by Oppenheim.[3] According to the latter, nervous symptoms may develop either immediately after an injury or after an interval of some weeks or months. The earliest symptoms are usually subjective in character; pain, especially at the seat of injury, and often increased on motion; irritability, anxiety, and timorousness. Later on the mental symptoms increase until the patient falls into an hypochondriacal-melancholic state, with anxiety and abnormal irritability. This is often accompanied by insomnia. The intelligence is usually not much impaired, but it may be considerably affected. Vertigo and faintness are common, and convulsive seizures and other spasmodic conditions may occur. Tremor is often seen, and other conditions, such as convulsive tic, muscular spasms, chorea, reflex epilepsy, etc., sometimes develop.

Motion is often affected. This may be due in part to the inhibiting influence of pain, but there is generally an actual paresis. The paresis may take a generalized form, or the form of hemiplegia, paraplegia, or monoplegia. Paralyses limited to a given nerve distribution are never seen. The paralysis usually differs in some respects from paralysis from gross lesions. There may be an increased tension of the muscles, but this differs from the contractures following gross lesions. The tendon reflexes are often exaggerated, but never absent. Muscular atrophy and degenerative reactions are rare. Paralysis of any individual cranial nerve is almost never met with, but motions which demand the harmonious activity of different muscular groups are often impaired. The speech may be affected, but the affection is more akin to stuttering; aphasia and simple disturbances of articulation are never seen. The pupils may be unequal, but they are rarely immobile.

Sensory disturbances are common, but they never follow the distribution of the peripheral nerves or of the spinal segments, and they are often associated with disturbances of the special senses, especially with cont the field of vision.

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[1] La nutrition dans l'hystérie.

[2] Berliner klin., No. 3.

[3] Op. cit.

Vasomotor disturbances are very common. The vesical fun usually not much impaired, but there may be retention or inc There is usually loss of sexual power. The skin reflexes may be but they are usually diminished or lost, especially in the anæsthetic

The heart is very often abnormally excitable, and this may rare disease of the heart, dilatation, and valvular insufficiency. These c however, do not differ very materially from those already describ preceding sections, or in the sections to follow. Strümpell consider traumatic neuroses are chiefly hysteria and neurasthenia, or the mi hystero-neurasthenia, but he considers that trauma often gives then what distinct type, and he retains the word as a convenient term. heim also admits frankly that there may be a pure traumatic hyster traumatic neurasthenia, but that there are mixed forms, hystero-neu often complicated with other neuroses and psychoses, and that then obscure affections due to more material changes in the central ne tem. For all these, therefore, he would employ the term traumatic as a convenient, although not strictly definite term.

Other writers since then, with less knowledge, have adopted the have come to regard traumatic neurosis almost as a distinct dis exophthalmic goitre. This must be regarded as a mistake. Others objected so strongly to the use of the term that there has arisen a controversial literature on the subject, with the rather unfortunate weakening Oppenheim's position as to the genuineness and severit affections. Just as some men thought that, by proving that there spinal concussion, they could disprove the existence of all these affe others, by showing that there was no "traumatic neurosis," have slig real troubles. Upon the whole, the introduction of the term has h influence. It has tended to check the differentiation of the various traumatic nervous affections, and it has substituted a general term for lar terms, which always leads to confusion. Hysteria, neurasthenia, neuralgia, epilepsy, might all be classed as neuroses, and they all may be of traumatic origin, but it is better to employ the more precise tern ever possible. It is often very difficult to say whether a case is to be as neurasthenia, hysteria, or some form of cerebral or spinal sclerosis have already seen, the transition forms between hysteria and neurasth frequent, but, whenever possible, the more precise classifications sh employed.

TRAUMATIC SCLEROSIS.

In the section on pathology I have given the anatomical eviden which the claim that some of these obscure traumatic affections are due nite structural changes in the central nervous system rests. From that it will be seen that the evidence is still very slight. We must admit traumatic epilepsy the injury probably produces a neuroglia scler some cases injuries may give rise to certain lesions in the spinal cord subacute myelitis or degenerative areas, chiefly in the lateral columns

It is also generally accepted that trauma may give rise to definite st changes from direct violence, contusion, or crushing of the nervous hemorrhage, sand even infectious processes, with no discoverable cu wound. In various germ diseases the injury may make the part a diminished resistance, and it may then become a favorite seat for ment of the infectious process.

where trauma seems to form the only cause.

Notwithstanding the unsatisfactory character of the pathological evidence there are a good many cases, some of them resulting fatally, where the symptoms do not fall under the rubric of neurasthenia or hysteria. These cases point to a general disturbance in the cerebro-spinal system, and occasionally they present symptoms which are usually regarded as due to more definite structural changes in the nervous system than we expect to find in hysteria or neurasthenia. Out of one hundred and nine cases coming under my own observation, twenty-three have been considered to be due to such conditions. The symptoms, as will be seen from what follows, resemble very much the symptoms in the affections previously described, but the condition is much more serious, and in many cases other symptoms are added which resemble those of disseminated sclerosis. Dana,' who describes these cases under the term of the grave traumatic neurosis, inclines to a belief that there are definite structural changes in the central nervous system, but he admits with justice that it is impossible to draw the line sharply between them and the cases of neurasthenia and hysteria.

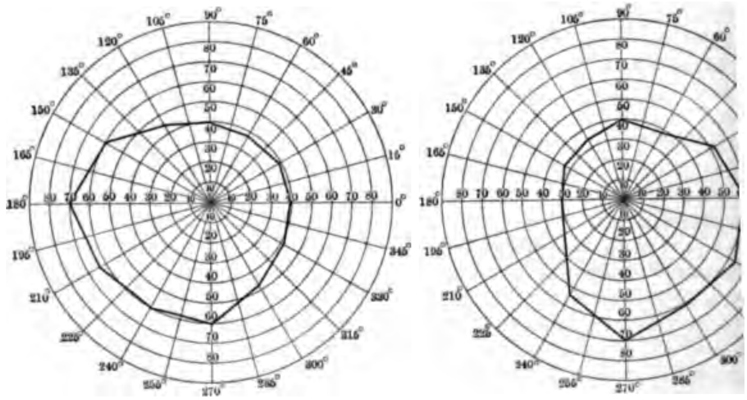
In the etiology of theses cases the psychical factor is less prominent, but the physical factor dominates, and the physical concussion often outweighs any possibilities of suggestion. In one case resulting fatally, after a period of a number of months, a man was caught in machinery and received physical injuries sufficient to break several small bones; a woman was dragged in a railway collision, her head and back bumping over the sleepers; she received injuries which resulted fatally after a period of twenty-one months; another man, while on a railway train, was struck by a bridge, the blow inflicting a severe scalp wound and causing unconsciousness for a number of hours. In other cases the physical force of the action was so great as to cause loss of consciousness.

The symptoms are usually of gradual onset, unless there be an initial period of unconsciousness. One of the most constant symptoms, especially with injuries involving the head, is headache. This is constant, not usually very severe, but occasionally exhibiting paroxysms of more intense pain. The pain is often localized at the seat of the injury, and it may be associated with local hyperæsthesia and tenderness. If not at the seat of the injury it is apt to be frontal or vertical; we seldom see the intense "pain at the base

neurasthenia are conspicuous by their absence. Actual delusions
cinations are rare. The sleep is usually very poor and is brok
dreams, especially by dreams of impending disaster.

With regard to the special senses, the smell and taste are often bl
sometimes lost. Auditory symptoms are not very important. Ti
common symptom, and the hearing may be diminished, with symp
cating a lesion in the labyrinth or nerve. Schultze[1] thinks that
of Ménière's disease are common. Only in one case have I note
deafness of probable nervous origin. The ocular symptoms are
greater importance. Strabismus is rare, but nystagmus is more
Inequality of the pupils sometimes occurs, and in several cases I h
the irritability of the pupils to light. Dilatation and contracti
pupils are rare. Diplopia has been complained of by a number of
and in one of my own cases which terminated fatally there was n
diplopia, which is more often an hysterial symptom, and which I
noted in traumatic neurasthenia. Failure of vision is not an u
symptom, sometimes with no errors of refraction and no change in th
A few cases are on record where there was optic atrophy, but none h
under my own observation. In a considerable number of cases
been a peripheral contraction of the field of vision, usually not
high degree, as shown in Fig. 53.

FIG 53.



Contraction of the visual field in traumatic sclerosis. (Personal observation.)

SENSORY SYMPTOMS. Sensory disturbances are common in this
affections, but they are by no means constant. In some cases the m
ful tests will reveal nothing, except, perhaps, local tenderness at the
the injury or in the back, where it is often due to a complicating tr
lumbago. More frequently there is a distinct loss of sensibility. Th
the form of diminished tactile sensibility, but the sensibility to pa
temperature and the muscular senses may also be involved. A dim
of sensibility to pain and temperature, with normal tactile sensib
rare; it probably occurs only in hysteria and in certain cord affection
lating syringomyelia, as in hemorrhage about the central canal. Th
nution of sensibility does not usually amount to absolute anæsthesia,
seldom has the distributions described under hysterical anæsthesia.
is usually a pretty general hypo-æsthesia, which is most marked in t

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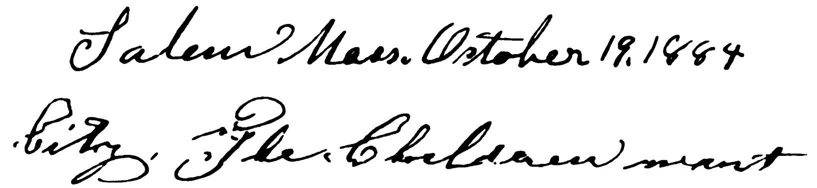
[1] Art. cit.

but it does not follow the distribution of anæsthesias from lesions of the spinal segments. There is seldom any definite line of division between the hypoæsthesia and the normally sensitive parts, but they shade into each other very gradually. The head is seldom involved. This form of anæsthesia is of interest with reference to Head's views as to a cerebro-spinal type of hysteria, which may be akin to disseminated sclerosis, but it does not strictly correspond to the anæsthesia that he describes.

Motor Symptoms. Oculo-motor symptoms have already been described. Paralysis is rarely seen, and, when met with, it is indicative of some focal lesion of the brain or cord. Ordinarily there is a general motor weakness, not amounting to definite paralysis, but involving both arms and legs. The gait is usually slow and feeble, and has often the characteristic features which complicating lumbago may give to it. It is also not infrequently uncertain, and the patient may stagger somewhat, but there is no real ataxia or spasticity. When the patient stands with the eyes closed there is often a pronounced swaying. The arms are weak, the grip, perhaps, not exceeding ten kilogrammes; all movements are slow, difficult, and uncertain, and there is often a distinct tremor of the hands, which in some instances may amount to a pronounced intention tremor. This tremor may show itself very markedly in the handwriting, Fig 54. The movements of the face are seldom affected, but a fibrillary tremor of the lips and tongue is quite common.

FIG. 54.


Salem, Mass. October 19. 1884
City. The Children must

Handwriting of patient with traumatic sclerosis before and after the injury, showing intention tremor. (Personal observation.)

Convulsive conditions are seldom seen. If convulsive attacks or temporary lapses of consciousness occur, they point to a neuroglia sclerosis, and the case must be regarded as one of traumatic epilepsy. Such cases are considered in another chapter. They are to be distinguished from hysterical attacks.

Muscular atrophy and electrical changes seldom occur; occasionally there is some general wasting, but not a degenerative atrophy.

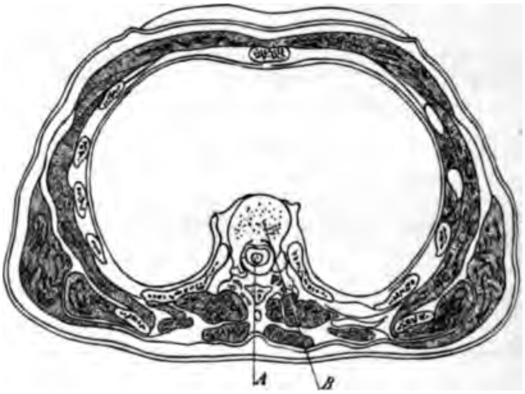
Reflexes. The skin reflexes are diminished when there is anæsthesia, but only in the anæsthetic region. The tendon reflexes vary; most commonly they are exaggerated, in rare cases even to the production of clonus, but occasionally they may be absent.

Other symptoms are not very remarkable, and they resemble those already described under neurasthenia. The cardiac-vascular disturbances are much like those of neurasthenia, but increase in the pulse-rate is less frequent, and symptoms referable to the heart's action, palpitation, cardiac distress, etc., are not so often seen. The appetite is often poor, nausea and vomiting are rather more frequent, and in some cases the control of the anal sphincter is diminished. Constipation is quite common. Vesical symptoms are rather more pronounced; it is not uncommon for patients to complain that it requires a greater effort to empty the bladder, and that there is more or less dribbling of urine after the act; but retention and incontinence are rare. The sexual powers are always diminished. There is usually a pretty constant failure of nutrition and a loss in weight.

TRAUMATIC SPINAL SCLEROSIS.

The spinal cord, as will be seen from the accompanying diagram, Fig 55, is deeply seated in the interior of the body; it is carefully suspended in a strong but flexible bony case, is stayed by the ligamentum denticulatum and the spinal nerves, and is protected by its membranes, a cushion of fluid and fatty tissue. Any blow on the spinous processes would naturally be transmitted through the laminæ to the bodies of the vertebræ. It is therefore not strange that many writers have maintained that it was impossible for injury to affect the cord unless there were direct violence sufficient to cause fracture or dislocation of the vertebræ, contusion, or crushing of the cord, or spinal hemorrhage. Such injuries give rise to definite symptoms of focal or transverse lesion of the cord, which are discussed in another chapter.

FIG. 55.



Horizontal section of the body through the eighth thoracic vertebra. (After BRAUNE.)
A. Spinal cord. *B*. Body of the eighth thoracic vertebra.

In the section on pathology reference has been made to the experimental and pathological evidence from which it seems probable that in some cases after severe injury there may develop morbid processes in the cord itself, of slow onset, without any injury to the surrounding parts, and localized chiefly in the white matter in the lateral columns. The clinical evidence of such conditions is even stronger.

The old term of spinal concussion, which was once in general use for all these conditions, has been generally abandoned by neurologists. There is, however, a condition which might almost merit the term. In a few rare cases paraplegia, sensory disturbances, loss of control of the bladder or rectum, loss of knee-jerk, etc., have developed immediately after a severe blow on the spine. These symptoms have disappeared entirely in a few hours or days. In such cases a disturbance in the cord seems the most probable explanation; but the nature of this disturbance is still hypothetical. The most plausible explanations are Schmaus's theory of a fatigue of the fibres, vasomotor changes, or slight meningeal hemorrhage. It may be borne in mind that the lower part of the cord is supplied with blood chiefly by the branches from the inter-vertebral arteries. It is not impossible that a severe blow may lead to a vasomotor paralysis of those vessels. The hypothesis of fatigue of the fibres seems, however, more probable.

He walks with short, scuffling steps, and with some unsteadiness, often requiring support. The gait is much like that of spastic paralysis. There may be a little wasting of the muscles in one or both legs, but it is general, and is not attended with degenerative reactions. The skin reflexes are not often affected, but the tendon reflexes are much exaggerated, and clonus may often be found. There is often some inco-ordination, but it is not very marked. There is often swaying when the eyes are closed. The weakness in the legs may increase to complete paraplegia.

The sensory symptoms are not usually so pronounced. Pain in the back, except as a result of traumatic lumbago, is rare. Pains in the legs, which may be sharp and lancinating like those of tabes, are not uncommon. Paræsthesiæ of various sorts, prickling, burning, crawling, etc., are often complained of. Anæsthesia is not constant, and seldom is pronounced. Anæsthesia in limited areas corresponding to the distribution of the spinal segments is not met with. There is most commonly a diminution of sensibility in all its forms, sometimes more marked in one leg than in the other; absolute anæsthesia is much rarer. Partial anæsthesia is also extremely rare.

There is generally difficulty in passing water, which may reach the point of retention or incontinence; more commonly there is great straining with some dribbling after the act, and occasional imperative desire to micturate. Constipation is common, and the patient has to strain greatly at stool; feces may accumulate in the rectum, and evacuation is imperfect; one patient complained that he had to use an enormous amount of paper to clean himself after a stool. Incontinence of feces is a later symptom. Impotence is a frequent symptom, but priapism is seldom, if ever, seen.

As has already been said, the majority of authors admit that various degenerative affections of the cord, notably progressive spinal muscular atrophy, may be due to injury, but these affections are discussed in the appropriate chapters.

Diagnosis. The diagnosis of traumatic nervous affections presents problems of a peculiar difficulty. Not only do we have to determine the character of an obscure nervous affection, but also, on account of the claims for damages that so often arise, we must decide whether there be any disease at all, or whether the patient be merely a simulator.

Neither of these points can be determined without a thorough and com-

In the differential diagnosis of the affections which have already been described, it is first of all necessary to eliminate the possibility of the existence of any focal lesion of the brain or cord, any systemic affection of the cord, or any disease of the peripheral nerves. This can usually be done by a consideration of the symptoms of these affections and by a careful examination. Traumatic lumbago, as has been said, is a frequent complication, and it usually can be readily detected by pain in the back, which is increased on movement, by the local tenderness of the muscles and their rigidity. Traumatic neurasthenia presents the characteristic symptoms of headache, depression, insomnia, incapacity for protracted effort, irritability, backache, muscular weakness, nervous dyspepsia, vasomotor disturbances, rapid pulse, exaggerated knee-jerks, "shifting" type of contraction of the visual field, together with the absence of the hysterical stigmata and any indications of focal or systemic disease. The diagnosis of hysteria must be based chiefly upon the discovery of the hysterical stigmata already described, especially the anæsthesia and the contracted visual fields. As has already been said, these two symptoms cannot be regarded as pathognomonic of the hysterical condition; but, in the majority of cases, a careful study of the symptoms already described will render the diagnosis of hysteria comparatively easy.

It is important to distinguish between the hysterical attacks and epileptic seizures, both of which may be of traumatic origin. In most cases there can be no confusion, but in some cases the distinction is by no means easy. In hysteria the patient is not apt to fall so as to hurt himself, the tongue is rarely bitten, consciousness is not so completely lost, there is rarely involuntary discharge of urine or feces; if the attacks be frequently repeated there is no rise of temperature. The urine is said, by Gilles de la Tourette and Cathelineau, to show certain characteristics already described.

As the very existence of the traumatic scleroses is still in dispute, the diagnosis is by no means easy. The history of a severe physical injury, the graver character of the symptoms, the mental failure, muscular weakness, the general hypo-æsthesia, and the physical symptoms will, on continued observation, give rise to the suspicion that there is a graver disturbance than simple neurasthenia. A confusion with hysteria is much less likely if due weight be laid upon the hysterical stigmata. If, in addition, there be optic atrophy, nystagmus, intention tremor without definite signs of hysteria, immobile pupils, loss of knee-jerk, degenerative reactions, and the like, the suspicions will become confirmed.

The diagnosis of traumatic spinal sclerosis is a simpler matter; the paraparesis, spastic conditions, bladder symptoms, sensory disturbances, and exaggerated tendon reflexes are at once suggestive of disease of the cord, and careful observation will enable the physician to eliminate mental and cerebral disturbances.

There has been much discussion of late as to the possibilities of simulation. We must, of course, admit that there are many men who will do anything but work in order to get money; and, consequently, there must be many men who would gladly simulate disease if, by so doing, they could get a large sum for damages from any corporation. It is unfortunately true that there are a certain number of lawyers and physicians who would join these men in such an attempt. It is, however, a harder thing to believe that such men, even when coached by such physicians, who are seldom very learned men, can successfully and persistently simulate the conditions already described. It is a singular fact that until recently very few neurologists have maintained that simulation was at all frequent, and the men who claimed that it was frequent have been, for the most part, railway surgeons, who have had but little familiarity with nervous diseases. Within a few years, as has been of them which have been published have not been able to stand careful criticism. In my own experience, although I have often met with cases which were said to be fraudulent, I have usually found evidence to prove the existence of disease. In less than 3 per cent. of the cases was I led to believe in simulation. The majority of neurologists in this country admit that simulation is very rare, although exaggeration is not uncommon.

The simulator is apt to present striking symptoms, and his efforts are usually so much exaggerated, and so incongruous as to be detected at once. No one familiar with nervous diseases can be deceived in him. All his symptoms are very much in evidence; they do not have to be sought for. It is also impossible to keep the attention fixed on any one subject for more than a very brief time; if the attention must be fixed on a number of different subjects at once, as, for example, when a man must feign a contracted visual field, anæsthesia, paralysis, and the like, the attention must inevitably be easily disturbed, and the expert can very soon catch the simulator off his guard and expose his deception.

In the majority of cases that come under observation the morbid symptoms must be sought for; they are not brought forward by the patient himself, and he is often unaware of their existence. It is not uncommon, for example, for a patient to say he has no trouble with his vision, yet, on examination, we find a slight contracture of the visual field; another may have normal sensibility to touch, with complete loss of sensibility to pain.

Careful examination will usually disclose symptoms unfamiliar to the laity, and often unrecognized by the patient himself, and it will usually reveal other symptoms, which have distinctly an objective character. The examiner must be familiar with the mental characteristics of nervous patients, the tendency to exaggerate, the variations that often occur in their general condition or in individual symptoms, and the seeming contradictions that these symptoms may present. Some striking examples of this have already been mentioned in the section on hysteria with reference to hysterical amblyopia and hysterical anæsthesia. In hysteria and neurasthenia, too, it is not uncommon for the symptoms to vary from day to day, and even from hour to hour.

To the person familiar with mental diseases the mental symptoms presented in most cases, such as have already been described, are easily recognized; yet to the casual observer they are not very striking. That they can be

The sensory symptoms also have a distinct objective value. It is not practicable for a simulator to tell whether a touch be on one side or the other of a given line, especially if that line pass horizontally about the body or a limb, or if it pass through the less sensitive parts of the body; hence, if there be a sharply defined anæsthesia, constant in its limitations, it is undoubtedly genuine. The simulator, moreover, will seldom adopt the more unusual forms of anæsthesia, such as the loss of sensibility to pain with retained sensibility to touch, or the slight blunting of the tactile sense. Moreover, when a sudden and very painful stimulus, such as may be conveyed, for example, by an extremely strong current from the induction coil through a needle point, is applied to any region of the body, especially the more sensitive regions, when the simulator is off his guard, it is impossible for him to refrain from some manifestation of pain; there, at least, will be certain muscular contractions or a quickening of the pulse. This was shown very strikingly by Pitres.[1] A man endeavored to feign disease, one of the symptoms being anæsthesia. He had previously earned his living by appearing as the anæsthetic man in a show, who thrust pins into himself without any manifestations of pain. The unexpected application of very painful stimuli revealed his simulation at once.

The increase of the pulse-rate on pressure over the painful region is objective evidence of tenderness, which is of great value. Paralysis with atrophy or with contracture can, of course, not be simulated; no voluntary contraction of the muscles is at all like the rigidity of contracture seen in certain forms of paralysis. Careful examination of an alleged paralyzed limb, especially if the patient himself be put through certain peculiar movements, and if the limb be moved about in different ways, will readily detect the existence of voluntary control of the muscles.

Although a tremor can be feigned under certain conditions, especially if the limbs be given a certain amount of support, if the limbs be unsupported it is impossible to continue a tremor with any definite rhythm, and the irregularities in the rhythm and in the degree of excursion of the limbs can at once be detected.

The circulatory symptoms, which have already been described, are of frequent occurrence, and they can, of course, be readily detected. I have already spoken of the distinction between the nervous increase of the pulse of the person coming under examination and the persistent increase so common in these conditions. Bladder symptoms are usually not of a very striking character. If there be distinct incontinence it can often be detected by carefully drying the parts with absorbent cotton and then noting if the urine escape drop by drop. This, of course, is a distinct proof of the genuineness of the symptoms.

Many neurasthenic and hysterical patients, no matter what the cause of their trouble, will exaggerate their symptoms. This is equally true in traumatic cases, whether there be a claim for damages or not; but it must be borne in mind that many, in fact the majority of patients with traumatic nervous affections who have a claim for damages, are more deeply interested in their personal sufferings than in any claim which they may have. The tendency to exaggerate can readily be detected by the man familiar with nervous diseases and with human nature, by a comparison between the complaints and the physical condition, and by applying the personal equation. Several careful observers, who have had the opportunity of examining all the claimants for damages for certain railway corporations, have assured me that the percentage of deliberate simulation was rare, and that the attempts at simulation were always gross exaggerations. I believe myself that the claim of the frequency of simulation is more often due to the ignorance of the physician than to any attempt to deceive on the part of the patient.

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[1] Op. cit., i., 79.

Prognosis. The prognosis of the affections described is of extreme importance, yet it is involved in much obscurity, and it is by no means easy to determine. In view of the immense importance which prognosis has in so many medico-legal cases it is by no means remarkable that, on the one hand, some men conclude that the great majority of cases recover completely, and that other men hold out gloomy predictions as to dementia, paralysis, and death. It is probable that a large proportion of the cases, taking all cases as they come, if they could be placed under proper conditions immediately after the injury, would do well. Unfortunately few cases are placed under such conditions; instead they are put under the worst possible conditions, and kept there for years.

Traumatic lumbago, when uncomplicated, is apt to be rather an obstinate affection, persisting for several months, and leaving its victim with a weak back; the symptoms being liable to occur at any slight strain or cold. When complicated with other nervous affections, especially under debilitating conditions, it lasts much longer. In rare instances it may persist, causing annoyance and some disability for years; in one case under my observation it lasted for twenty-eight years. Here there was probably some more deeply seated trouble, such as a chronic arthritis of the vertebral articulations.

The prognosis of traumatic neurasthenia is better than that of the other traumatic nervous affections. A certain proportion of cases get well if they can be placed under proper conditions. As in all forms of neurasthenia, however, the process of recovery is slow and tedious, and it must always be measured by months and sometimes by years. The prognosis in all forms of neurasthenia must vary with the duration of the trouble, and as the conditions in many of the traumatic cases are bad, as has already been shown, it is obvious that the prognosis must also be unfavorable. When any case of neurasthenia has continued under unfavorable conditions which tend to aggravate it, without treatment, for two or three years, the chances of recovery are greatly diminished. The chances of complete recovery in any case of neurasthenia that has existed over three years are slight. The patient may make a partial recovery, and perhaps be able to resume his accustomed work, but he has less endurance, his trouble is apt to recur after comparatively slight strains, he is nervous and irritable, his sleep is less sound, his judgment is not quite so good, he has tendencies toward headaches and digestive disturbances, he gets on less smoothly with his fellows, trifles annoy him, and, instead of being a capable business man and a genial companion, he is apt to be a nervous, querulous, disagreeable person—a " damaged man." The casual observer seeing him at his wonted tasks says that he is well, but his intimate friends and the neurologist can detect the change brought about by the accident. More than one case of traumatic neurasthenia can be cited where there was never any claim for damages, but where the symptoms have persisted ten, fifteen, or twenty years, causing various disturbances, from a mere change of disposition and diminished endurance to complete disability.

Even in severe cases, if treatment be instituted promptly, there is a fair chance of recovery after a number of months, but when the condition has lasted a long time it is probable that the changes in the brain become greater, and that the cells cannot be restored to their normal condition. Many cases, even after a lapse of several years, may make a partial recovery, which, under the excitement and stimulus of an award of damages and the relief from the anxiety of litigation, seems at first very great; but afterward there is a relapse, and the patient is seldom fully restored to health. A considerable number of cases persist partly disabled and semi-invalids, and others still remain complete invalids for life.

The prognosis of traumatic hysteria is better and worse than that of neurasthenia. The individual symptoms of hysteria are capable of complete and sometimes sudden cure, whence many have believed that the condition itself was cured. Hysteria, however, is on the whole a graver condition than neurasthenia, and it is to be borne in mind that when once a patient becomes hysterical the condition is apt to become permanent, although the individual symptoms may be relieved. Although the paralysis, contractures, or anæsthesia may wholly disappear, nevertheless the patient remains in an hysterical condition, and the same symptoms, or others even graver, may recur from the slightest cause. Moreover, in traumatic cases, we usually have to do with a combination of the two affections. The same, therefore, holds true of hysteria as was said of neurasthenia; a few cases, if put promptly under treatment, may recover completely after an interval of a number of months, as Dercum[1] has shown; the majority continue in a condition of partial invalidism. It must not be forgotten, too, that in rare cases both hysteria and neurasthenia may terminate fatally.

The prognosis of the traumatic scleroses does not differ from that of other forms of sclerosis; these cases gradually grow worse, and finally succumb, if not to the disease, to intercurrent affections.

Including all patients who have nervous disturbances after injury, in many cases, of course, the trouble is slight, and they recover completely. Such cases are, however, less frequently seen by the neurologist. In cases which present pronounced symptoms of nervous disease the prognosis must be regarded as grave. Out of fifty consecutive medico-legal cases that I have seen I have known of the death of four from the nervous disease within six years after the injury, and of many of the cases I have no present knowledge. Page[2] has tried to show that most cases recover speedily after damages are paid, but his statistics do not inspire confidence, and most neurologists recognize that the majority of severe cases never make a complete recovery.

Treatment. Every case of traumatic nervous disease ought to be put under systematic and thorough treatment from the beginning. In the section on etiology I have shown that this was seldom done in the medico-legal cases, but that, on the contrary, treatment was neglected, and that many factors were introduced which tended to aggravate the condition and to render it chronic. If proper treatment were promptly instituted the prognosis would be materially improved and many patients would doubtless recover who now remain invalids. Unfortunately, proper treatment is often not feasible, even in cases where there is no litigation. The patient may not be able to afford the expense of a prolonged and thorough course of treatment, or, if he be only partly disabled, being a wage-earner he will feel that he cannot afford to give up the time requisite for treatment, but he will insist on keeping on with his business and doing the best he can.

In medico-legal cases the opportunities for proper treatment are rare. No course can be more at variance with any reasonable therapeutics than to subject a case of severe neurasthenia or hysteria to repeated examinations by different experts, not for the benefit of the patient, but to satisfy the lawyers and to make a numerical showing of experts before a jury; to keep the patient harassed by excitement and vexations of a trial; to accuse him of fraud; to carry him to court, perhaps upon a stretcher, to be cross-examined and held up to the view of an intelligent(?) jury, who are to decide upon the genuineness of his complaints, the character of his disease, and his chances for recovery.

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[1] Alienist and Neurol., Oct. 1893.

[2] Art. cit.

In most of the affections which have been described the treatment requisite is the rest-cure, which, in all severe cases, must be carried out in all it details. This rest-cure has already been described in Chapter II., but it is necessary here to speak of certain minor details.

In a large number of cases traumatic lumbago is a complication so distressing as to require special treatment. Rest in bed often becomes necessary, the pain on motion being so great, but absolute rest by a spinal brace is not to be recommended; it will often give temporary relief, but it is merely a palliation, and the progress is apt to be slow under it. As in sprains elsewhere, it is better to treat the muscles and ligaments by permitting and encouraging a certain amount of motion. This motion can be best obtained by gentle massage of the injured muscles. This should be begun early, and it will usually be well borne. After a short time it will be found that the patient can bear more energetic, deep massage, and that he will improve under it. In addition to the massage much relief may be obtained by galvanizing the tender region, applying a large electrode with the positive coil, with a current of five to ten milliampères, over the spinal muscles. By massage and electricity considerable relief can usually be obtained, and many cases will be greatly benefited. Certain other remedies are also of advantage. Ironing the muscles with a flatiron, as hot as can be borne—a combination of warmth and massage—will give relief in cases where massage itself cannot be applied. Cupping, blistering, and counter-irritation by iodine give temporary relief, but they are apt to render the skin sensitive, so that massage cannot be used. The same may be said of the actual cautery, which, in more chronic cases, often gives very distinct temporary relief. As the patient's condition improves and his ability to move becomes greater, light gymnastics for the spinal muscles will often be of distinct benefit, but they should not be undertaken if they cause any increase of the pain which persists after exercise.

In hysteria and neurasthenia the rest-cure of Weir Mitchell is the only proper treatment, and, in all severe cases, it should be carried out in all its utmost details. Dercum has shown the benefit of this treatment when thoroughly employed from the beginning of the trouble, and, as was said above, many patients may recover under it who otherwise would remain chronic invalids. In the cases where this is not feasible, from the causes already mentioned, the physician is compelled to adopt a very much more unsatisfactory course, which often means more protracted suffering and greater expense for the patient, and also a poorer chance of complete recovery. When the complete rest cure cannot be instituted the patient should take as much rest as possible; he should lie down during the day, and avoid everything but the most necessary duties; he should be kept from all excitement and worry; forced feeding should be employed, milk, eggs, soup, prepared foods, etc., should be given in small quantities at frequent intervals. Galvanism and massage are of great benefit. In other respects treatment must be symptomatic. The bowels should be kept open, stimulants, analgesics, and hypnotics should be avoided as much as possible, and tonics freely given: iron and arsenic seem more beneficial than strychnine. Where there are pronounced cardiac symptoms digitalis and similar remedies may be necessary.

Page[1] has claimed that the symptoms in many cases were due not to the injury, but to the abuse of bromide of potassium. That the excessive use of the bromide is injurious no one will deny; but the symptoms from the abuse of bromide do not resemble the symptoms we see in these cases. The symptoms occur in cases that have never taken bromide, and many cases are benefited by a moderate use of the bromide. I have seen distinct improvement occur after taking ten to fifteen-grain doses of bromide three times a day. The bromides must be used with care, but they often relieve much of the nervousness and anxiety from which these patients suffer. They are best given combined with gentian and the tincture of chloride of iron.

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[1] Med. Times and Gaz., April, 1885.

The treatment of the traumatic scleroses does not differ from the treatment of other forms of sclerosis. Rest, seclusion, forced feeding, and tonics are the essentials. In other respects the treatment must be largely symptomatic.

In all medico-legal cases the physician should urge a settlement of the case rather than expose the patient to the anxiety and risks of a trial. An early settlement is of the utmost importance in affording the patient a chance for recovery, and it is better to settle for a moderate sum rather than to expose the patient to such risks in the hope of getting a little more money. If any system of procedure could be adopted in medico-legal cases whereby one competent expert could have the opportunity of seeing the patient as often as he might see fit, and could give an opinion which would be accepted as a judicial ruling, it would be much better for the patient than it is to be subjected to frequent examinations by incompetent men ; his chances for recovery would be greater, and the ends of justice would be better served.

CHAPTER V.

DIATHETIC AND TOXIC AFFECTIONS OF THE NERVOUS SYSTEM.

BY E. D. FISHER, M.D.

DIATHESIS is a tendency to disease, and therefore chronic by its very nature. We understand by it a condition, inherited or acquired, which under certain conditions, as errors of diet, exposure, etc., may manifest a definite class of symptoms. Rheumatism and gout represent such diatheses. The acute exacerbations in each of these diseases represent the overwhelming of the system by the distinct poison peculiar to each.

The relation of diathesis to nervous affections is of great importance in its bearing on a class of diseases which are often not the direct or immediate effect of the poison on the economy, but rather the accompanying or consequent injury to the nervous system.

We may define diathesis, according to Hutchinson, as "any condition of prolonged peculiarity of health giving proclivity to definite forms of disease." Diatheses represent morbid conditions of the blood. We have to consider them in their relation to the nervous system in two aspects: first, as to their primary effect as poisons *per se* within the economy, producing characteristic symptoms, and secondly, as to their secondary effect on the nervous system. It is, as a result of this secondary effect, that we find the more permanent and chronic changes induced.

RHEUMATISM.

Rheumatism may be regarded as a morbid blood state affecting the chemical processes of the body (Gowers.) It affects almost all the structures of the nervous system. The somatic signs of the disease are inflammatory states of the articular and muscular structures, accompanied by pain and high temperature.

Etiology. In the light of the fact that the rheumatic diathesis is peculiar to certain families, and that therefore heredity is an important etiological factor in the disease, it would seem that the theory of the disease, as due to some chemical change of nervous origin, causing malnutrition and nutritive changes, is the correct one.

Symptoms. The following symptoms occur as a direct or indirect result of the involvement of the nervous system.

Insanity, as a direct result of rheumatism, is rare. Clouston and Wiglesworth have reported several cases. The characteristic symptoms are delirium, hallucinations of sight and hearing, associated in some cases with choreiform movements. The direct relationship between the rheumatic poison and the appearance of the cerebral symptoms seems positive. It cannot, however, be considered as a metastasis, but rather the direct influence of the poison on the cerebrum.

Meningitis. There is usually present a marked delirium, which may be low and muttering or very active, and the patient may pass into a partial coma interrupted by delirium. Convulsions are not common, although they may be present. The temperature may rise to 106–7.° Post-mortem, inflammation of the meninges is found, generally simple in character, without pus formation. Hemorrhages have been observed in the cortex of the brain, and general congestion, giving a rosy appearance to the surface.

Hemiplegia. Cerebral embolism is a very frequent though late complication of rheumatism, due to the endocarditis which so often accompanies rheumatic fever. While we cannot consider it as directly due to the rheumatic diathesis, yet this disease is the most frequent cause of the cardiac complication. It is essentially a disease of youth, so much so that probably most cases of cerebral hemiplegia in the young are due to embolism.

Chorea. A considerable percentage of cases of chorea give a rheumatic history, with or without cardiac lesions. A large proportion, however, present no such connection. The course and symptoms are similar in both cases, the element of rheumatic disease not altering its characteristics. An anæmic condition is to be observed in most cases. Sir Dyce Duckworth goes so far in his statement as to say that both chorea and rheumatism are simply the result of the same specific agent, manifesting itself at one time in one form as chorea, and at another time as articular inflammation. Writers in this country do not find rheumatism so frequent an etiological factor in chorea as the French and English statistics would seem to indicate.

Associated with the chorea we may have a neuritis, general in distribution, or again, apparently, secondary to spinal cord disease. The paralysis, even when hemiplegic, is usually flaccid, the muscles are not rigid, and therefore the indication is that it is due either to involvement of the peripheral nerves or to the spinal cord in the region of the anterior horns.

Spinal Cord Diseases. Lesions of an acute nature, as poliomyelitis, are rare. In many rheumatic cases, however, exposure to cold appears to affect the nervous system so as to cause the production within it of a toxic agent capable of producing an inflammatory condition within the cord itself. It is not improbable that both poliomyelitis and general myelitis may be produced in this way. The relation of such a cause to chronic and systemic disease of the cord does not, however, seem equally clear. Meningeal inflammation, however, undoubtedly occurs, and when acute, is generally associated with the cerebral condition. It may not, therefore, be recognized. The symptoms are those common to meningitis—pain in the spine on movement, with extension into the extremities, giving evidence of irritation of the meninges and the spinal nerves at their exit from the canal. There is nothing diagnostic in the symptoms of their rheumatic origin, except their occurrence in the course of acute rheumatism. The prognosis is dependent upon the general course of the disease.

Neuritis. Multiple neuritis may be present, but is rare. A number of cases, however, have been reported. It is more commonly observed as affecting individual nerves. It is not uncommon to find the spinal accessory, the facial, the fifth, the ulnar, and sciatic nerves involved; more rarely, the vagus, the optic, and the oculomotor. No nerve is perhaps free from the affection. We must make a distinction in these cases, however, from those occurring around the joints in the course of articular rheumatism, as here the direct influence of the joint inflammation is probably the cause of the neuritis. It may also occur secondarily through the spinal cord (Charcot).

Neuralgia. Neuralgia is frequently associated with rheumatism, especially on exposure. The sciatic nerve is perhaps the most frequently involved. In these cases there is usually present some neuritis.

Pathology. In meningeal cases we find in the brain the pial vessels usually congested. The subarachnoid space and ventricles are filled with fluid, and perhaps discolored by blood. The cortex is congested and of a reddish color. Actual pus formation is rarely, if ever, present. The dura is not involved. The spinal meninges may show a like inflammation, and changes of an inflammatory nature in the anterior horns, with softening, have been observed. The change in the peripheral nerves is usually an interstitial neuritis, and the axis cylinder in most instances escapes. Parenchymatous degeneration, however, with destruction of the axis cylinder, may occur.

Diagnosis. The diagnosis is not difficult where a previous history of rheumatism is present; otherwise the symptoms are not diagnostic.

Prognosis. The prognosis is bad in cerebral cases if the temperature is high and delirium and coma are present. In neuritis it is generally favorable.

Treatment. In meningeal cases, with high temperature, the cold bath or douche is advisable, with the salicylates or salol. Acetanilide, antipyrine, and phenacetine may be prescribed to relieve the pain. In neuritis, associated with paralysis or wasting, strychnine, massage, and electricity are indicated. Remedies applicable to rheumatism in general, such as the alkalies or piperazin, are also of use here.

GOUT.

Gout is an acute or chronic diathetic disease manifested by local inflammation, with intense pain when acute, and often without either of these symptoms when chronic. It may be inherited or acquired. The attacks are associated with excess of uric acid in the organism, and deposits of urate of sodium in the articular cartilages and fibrous structures (Foster). It is, however, as a diathesis that we have to consider its relation to the nervous system. The predisposition to disease which this morbid blood state manifests, when once acquired, is permanent, and the more conspicuous outbreaks are to be considered as a temporary exaggeration of the peculiar morbid constitutional condition (H. T. Lyman). As in rheumatism many of the effects have to be considered from the standpoint of their indirect action rather than their direct influence upon the system. It is not only the effect of the uric acid itself, but its influence in causing other nutritional changes.

Etiology. Heredity is perhaps the most important factor in the list of causes. Malt liquors have a special influence, and also excess in eating when the habits are sedentary. Lead poisoning, especially in chronic cases, and when associated with the free use of alcohol, is also a provocative agent.

Pathology. The morbid conditions which have an especial bearing on the nervous system are not well defined. Uric acid and urate of sodium have been found deposited in the meninges of the brain and cord, and also in the neurilemma of the nerves; but the most common cause of the changes found in these organs is the sclerosis which affects both arteries and veins. We find especially the vessels of the base of the brain atheromatous, and an increased quantity of fluid in the ventricles and in the subarachnoid space. The vessels are at times partially obliterated, and it is possible that when the circulation is thus impaired that the direct action of the uric acid is manifested. The effect of gout on the vessels may be perhaps best studied in the eye. Dr. C. S. Bull, in an article entitled "Gouty Retinitis, Chorio-, and Neuro-retinitis," described the effects of gout on the vascular system as follows: (1) A high blood pressure in the arteries; (2) hypertrophy of the left ventricle; (3) hard, incompressible arteries undergoing atheromatous change. From these may result apoplexy, by rupture of the bloodvessel, aneurism, by dilatation of the vessel, or angina pectoris, with fatty degeneration of the heart. The first recognized departure from the normal condition of the vessel consists in stretching of the tunica media or elastic layer, and this process is the cause of a diffused primary arterio-sclerosis, characterized by dilatation and tortuosity of the arteries, by eccentric hypertrophy of the media, and by diffused compensatory fibrous thickening of the intima. Cerebral hemorrhage and thrombosis are common attendants on these vascular changes. In the spinal cord we find no special lesions other than those dependent upon the interference with the vascular supply. In some cases, as we have said, the uric acid deposit may give rise to direct irritation from the meninges, as is shown by shooting pains and spasms, or contractures of muscles.

Symptoms. The following nervous affections are met with:

Mental Disease. Mental disease as a consequence of the gouty diathesis is not uncommon, while at the same time no definite form of insanity can be ascribed to it. Melancholia is perhaps the most common form, although mania is not rare. Depression and loss of energy are almost always associated with gout, but this may increase to actual insanity, pursuing a course extending over months or years. It is most often seen in "suppressed gout" (Garrod), in which gouty attacks alternate with mental disturbances, and in which hallucinations, delirium, and convulsions are present. The poison of gout is especially observed in those hereditarily affected and in those of a neurotic disposition. Patients are more liable to present mental phenomena at the special periods of life, such as the climacteric, puberty, and old age. In women after the climacteric, we find melancholia with suicidal tendencies. The arterio-sclerosis with its accompanying vasomotor disturbances is the direct result of the action of the toxic material in the blood. No doubt such important factors as the cerebral anæmia, occasioned by the arterio-sclerosis, have their influence, or there may be special exciting causes, such as financial or domestic worry, or excesses of various kinds. Little can be added to that already stated concerning the morbid changes. Arterial disease with signs of congestion and serous effusion is found in the brain. The cerebral state is one of impaired mental activity, a tendency to apathy and loss of energy being marked; or again, there may be the almost opposite state of irritability and insomnia. There is often an inability to concentrate the attention on any subject, and a feeling, as one patient expressed it, as if he were only an on-looker on what was occurring. Vascular disturbances, often sudden in their onset, occasion vertigo, tinnitus aurium, and sensory impressions throughout the body, such as heat, tingling, and numbness, or neuralgic pains, which may secondarily occasion hallucinations of sight and hearing. Great depression, perhaps alternating with excitement and restlessness, is present, and a fear of impending evil—of the loss of money or the fear of committing some act to which the patient is impelled. Cephalagia is usually diffused in character, though at times it is unilateral, and then is probably dependent upon a neuritis. This condition may continue for a long period of time, changing as the blood state varies. The most common form of mental disease associated with suppressed gout, however, is melancholia, either simple, with stupor, or delusional. The close relation of the gouty poison to the cerebral state is clearly seen when an acute "external" attack, *e. g.*, acute involvement of a joint, entirely relieves the mental condition. Permanent dementia may result, dependent probably on the malnutrition and the consequent cerebral atrophy accompanying the arterial degeneration.

In the mental affections of gout the cause can only be determined or made probable by the history of previous attacks of gout or by a marked history of heredity. Many of the hereditary cases occur in the young and where no local manifestations exist. Such conditions most probably fall under the head of cerebral neurasthenia. It is not infrequent, however, to find fixed delusions, especially of doubt and fear, with suicidal impulses. Strict anti-gout treatment is always indicated.

Cerebral Neurasthenia. Lithæmia, affecting especially the cerebrum, and giving rise to a large class of indefinite symptoms, such as hypochondria, headache, depression, cardialgia and palpitation, various neuralgias, etc., seems to be a disease which has to-day been especially called to the attention of the neurologists and the profession in general. Many of the symptoms already detailed in considering mental disease due to gout can be applied here. Hereditary predisposition to nervous disease is perhaps the main factor in its evolution. Another important factor is malnutrition dependent upon poor assimilation, leading to non-oxidation of the nitrogenous elements of the food. These patients rarely have gout in the true sense, that is, it rarely shows itself externally except perhaps in the irregular form of some skin affection. They are the victims of ill-health; they suffer from a long continued impairment of nervous nutrition. On arising in the morning the depression is generally most marked. Their sleep, which may have been profound, has not given a sense of rest; headache, usually of a general character, and a feeling of constriction, are common, and may alternate with severe hemicrania, cardialgia, paroxysmal dyspnœa, pleurodynia, cramps in the muscles, tremor, etc. Indigestion, flatulence, gastralgia, and constipation are easily induced by slight errors of diet. Indisposition to exertion is marked. There is frequently a fear of doing anything or going anywhere.

The prognosis in these cases is unfavorable as to complete recovery, though marked improvement may be obtained by attention to the diet and living in the open air. General stimulation by massage and electricity is also indicated. (See also Chapter II.)

The morbid changes are usually not destructive in these cases. Rarely, it has been said, urate of sodium has been found in the meninges or on the surface of the cord. In chronic cases, degenerative changes are found in the nerves, and are the result of the general arterio-sclerosis, rather than of the gout *per se*. The symptoms do not differ from those of neuritis in general, and will not be detailed here.

Spinal Diseases. Gout may be considered as an etiological factor in both acute and chronic myelitis, more often in the disseminated than in the transverse form. It probably acts rather as a predisposing cause in most cases where exposure and overexertion or traumatism have been the immediate or exciting agents. Sudden hemiplegia or paraplegia, which not infrequently occur in the gouty, is the result of an acute overwhelming of the functional activity of the cord by the toxic agent. Spinal meningitis is rare as a complication; not infrequently, however, pain meningeal in character, due to gouty deposit in the meninges, is present and disappears upon treatment. The symptoms are fixed pain in the back, increased on movement and on pressure, and shooting into the extremities along the course of the nerves involved. There is little that is diagnostic except the history of the presence of the gouty diathesis, and its disappearance on the exhibition of the appropriate drugs.

Neuritis. Various nerves may be involved, but rarely do we find multiple neuritis. Paralysis of individual muscles or groups of muscles may occur. The cranial nerves are often involved. Optic neuritis has been observed by Hutchinson, Gowers, and others, and, as already referred to, by Dr. Bull. This writer says the most marked feature in the fundus is the development of the arterio-sclerosis and phlebo-sclerosis. The changes in the optic nerve seem to be almost entirely intra-ocular, and cannot be traced for any great distance back of the eyeball.

Neuralgia. Neuralgia is very frequent in gout, especially involving the fifth nerve, the sciatic, and the intercostals. The superficial distribution of the nerves of the skin are very often the seat of pain of an acute character, especially called out by sudden changes in temperature. Even the removal of the clothing, or entering a bath of too low or too high temperature, may induce this. Several such cases have come under my observation in which the pain has been associated with cramps in the muscles. Visceral neuralgias are very common. I believe most of these diseases are dependent upon a deficient blood supply to the nerves, owing to the arterio-sclerosis or to the vaso-motor disturbances. In the chronic cases, which are indeed the most common, as Gowers says, there is probably a neuritis present. A patient at present under observation with trigeminal neuralgia gave well-defined anæsthesia over the distribution of the superior branch of the nerve, which disappeared only upon treatment for the gouty diathesis. A second case with a similar affection showed a reflex spasm of the muscles of the ear, resembling a convulsive tic. Here a long history of gout revealed the basis of the disease.

General Prognosis. The general prognosis, other things equal, is favorable where strict attention is paid to the diet indicated for gout in general. In chronic cases with arterial changes there always exists the danger of cerebral hemorrhage. Chronic renal changes, it is almost needless to say, also influence the prognosis unfavorably.

Treatment. The treatment does not differ from that laid down for the ordinary management of gout.

URÆMIA.

Uræmia is a condition due to the absorption or retention in the blood of materials which have not been eliminated by the kidneys. The symptoms are as definite as those due to other toxic agents, *e. g.* opium. They are also as temporary, and cease when the poison is removed; and, as with opium, the system can learn to tolerate moderate doses with but slight reaction.

Etiology. Uræmia is commonly due to acute or chronic nephritis; perhaps it is more often associated with interstitial nephritis. There is frequently a history of a chronic lead or alcohol poisoning. Bouchard thinks it due to faulty elimination of the kidneys. Brown-Séquard also believes that there is a defective kidney secretion, causing a chemical modification of the blood. Experimentally kidney extract has relieved the uræmic symptoms where both kidneys have been removed.

Pathology. The changes most frequently observed in the brain are those associated with arterio-sclerosis of the cerebral vessels, with increase of fluid in the subarachnoid space and in the ventricles, with not infrequently a localized œdema over one hemisphere.

Symptoms. *Insanity.* Clouston ascribes mental diseases to affections of the kidneys, especially those of the maniacal type; while Griesinger considers Bright's disease rare as a causative agent. In the *Alienist and Neurologist*, 1890, Dr. Alice Bennett refers to a number of cases of insanity with Bright's disease as a causative agent. While kidney complications are not uncommon among the insane, and may modify the course of the disease, it is difficult to ascribe any well-defined form of mental disease to them. I would speak, therefore, of insanity with Bright's disease rather than of a special form due to uræmic states. A case of profound melancholia with delusions of persecution and suicidal attempt came under my observation, in which there was extensive disease of the kidneys, as revealed by autopsy. No lesion of the brain was present with the exception of atheromatous degeneration of the vessels at the base, and an increase of the fluids in the subarachnoid space and in the ventricles. It is impossible to differentiate these symptoms from those observed in any case of melancholia without affection of the kidneys.

The acute cerebral symptoms consist of stupor, headache, insomnia, dyspnœa, restlessness, muscular twitchings, general convulsions, and hemiplegia with coma. In the chronic cases we find alternate delirium and stupor.

Hemiplegia. Hemiplegia, or, more rarely, monoplegia of uræmic origin, has often been observed, and several of these cases have been reported with the usual symptoms—hemiplegia, aphasia, and exaggerated reflexes. Two such cases came recently under my observation, one of six months' duration, the other proving fatal within twenty-four hours. Careful post-mortem examination revealed no explanatory cause for the condition. It is possible, as has been stated by some writers, that we have to do with a serous effusion involving a limited area of the brain, consequent upon disease of the vessels insufficient to produce a hemorrhage or actual softening. There may be, however, more or less complete obstruction of the circulation, which, with the diseased coats of the vessels, allows an exudation through their walls of the fluid elements of the blood into the surrounding tissues. It is certainly difficult to believe that the poison in uræmia should select a definite area of the brain, except in association with disease of the vessels at that point. There may be all the usual signs observed in cerebral hemorrhage. The onset is, ordinarily, sudden, and there is marked coma. A diagnostic point is that the paralysis may entirely disappear in a few hours or days. It is commonly an accompaniment of chronic nephritis, where the arteries are the seat of sclerosis. If recovery from the hemiplegia is only partial, that is, if we find traces of paresis with exaggerated reflexes, we must ascribe it to softening rather than to uræmia. I think the majority of cases reported will fall under the latter head.

Convulsions. Convulsions are of frequent occurrence, and may be continuous or paroxysmal. There is usually coma between the attacks, from which the patient may be at times aroused. These seizures may be localized or Jacksonian in character, though rarely, being usually clonic and general in distribution, resembling those observed in general paresis. The onset of the convulsions or of the delirium may be sudden or gradual. In the former the pulse is of high tension. These attacks may continue for days or weeks, the pulse changing in character with the onset of the acute symptoms. In the fatal cases it becomes feeble and rapid. In chronic cases the high-tension pulse may not be present, but instead of this the pulse is rapid and compressible. It would seem, therefore, that this condition is not dependent on the uræmia—that is, the filling of the blood with poisonous materials—but rather due to the contraction of the vessels, as relief of this contraction by the administration of nitroglycerin, or by other means, causes these symptoms to subside. Again, this condition may occur even where the quantity of urea is not diminished and the specific gravity of the urine is normal.

Diagnosis It is frequently difficult to make a differential diagnosis from cases of cerebral hemorrhage, either involving the cortex or the ventricles, especially where hemiplegia is a complication in uræmia; or, again, from pachymeningitis hemorrhagica, especially as in these conditions the urine frequently contains albumin. In uræmic hemiplegia, however, when the onset is sudden, there is usually a rise of temperature, although this is absent in chronic cases, and may even be subnormal. The sudden clearing up of the paralysis is, however, in its favor.

Prognosis. The prognosis is entirely dependent upon the condition of the kidneys and the degree of associated cardiac and arterial disease. The attacks may be frequently repeated, and the patient is usually left weaker, and there is less reaction after each one.

Treatment The general treatment for nephritis must be followed. The kidneys should be stimulated by the administration of copious draughts of water, infusion of digitalis, etc. In coma and other exacerbations of the uræmic state, elaterium, gr. ⅙, may be given to secure profuse watery stools. By these means the elimination of the toxic substances in the blood is much favored. However, the most efficient remedy is pilocarpine hydrochlorate, which should be given hypodermatically in doses of gr. ⅛ to gr. ½. The immediate cerebral symptoms are to be relieved by attention to the heart and arteries. In acute conditions with high-tension pulse and convulsions, nitroglycerin, morphine, and chloral are indicated. When the pulse is feeble and of low tension, morphine seems to increase the coma, and nitroglycerin is contraindicated. We then use digitalis and strophanthus, which act unfavorably when the pulse is of high tension. This was admirably shown in a case where alternate conditions of high-tension pulse and a pulse without tension were present. Digitalis and strophanthus increased the cerebral symptoms when administered while the pulse was of high tension, but diminished them when the pulse was of *low tension.*

ALCOHOLISM.

Alcoholism is a term first used in 1848 by Huss, and comprises the phenomena traceable to the taking into the system of alcoholic liquors (Foster). It is a pathological state induced by the excessive use of alcohol, and is characterized by special symptoms due to disease of the brain, spinal cord, and peripheral nerves.

Etiology. The effect of alcohol on the nervous system varies with the manner in which it is taken. Those addicted to periodic excesses are not as liable to acute inflammatory lesions as those who habitually use alcohol. This may explain the fact of the greater frequency of multiple neuritis in women than in men, for women, if addicted to the use of alcohol, are apt to take it continuously in small doses and confine themselves to spirits rather than to malt liquors. The predilection of certain persons to be affected by alcohol is dependent upon some constitutional condition which it is difficult to explain. This is also observed in the part of the nervous system which may be selected as the site of the disease. Rheumatism, gout, lead, and heredity have an important influence in this respect. Not sufficient importance is placed on the degenerative influence of alcohol on the children of those addicted to its use. Conjoined with hereditary syphilis, it is productive of many lesions affecting the cranial nerves. Cerebral hemiplegia, otherwise rare in childhood, idiopathic epilepsy, and imbecility, with acute and systemic diseases of the cord, are not infrequently traceable to the same cause.

Pathology. While the peripheral nerves, and especially the nerve endings, are peculiarly susceptible to the poison of alcohol, the brain and cord are also involved. In acute alcoholism little pathological change is observed. In chronic conditions we find pachymeningitis. The pia is also frequently clouded or thickened, and there is œdema of the convex surface of the brain with effusion of fluid into the subarachnoid space, and into the ventricles, causing flattening of the convolution. Pachymeningitis hemorrhagica is not uncommon. The vessels also are the seat of atheromatous changes. The cells in the cortex show degeneration, their processes being lost. The association fibres also are degenerated. This is found in many of the older cases when dementia is present, and also in those with which multiple neuritis is associated. The cranial nerves, especially the pneumogastric and phrenic, give evidence of an interstitial neuritis. Sharkey refers to a case of paralysis of the phrenic and pneumogastric nerve, in which the whole trunk of the nerve was involved, and also the spinal cord. The spinal cord lesions are rare, and may be of inflammatory type, showing marked hyperæmia with capillary hemorrhages and softening. Dr. David Finlay has reported a case with autopsy in which the anterior horns were involved, the cells being shrunken and their processes lost. Chronic myelitic conditions are not infrequent, but are probably dependent upon the arterial degeneration, rather than upon the direct influence of the alcohol. In the nerve tracts of the cord we find interstitial, or, more rarely, parenchymatous disease of the nerve fibres. Gowers states that acute alcoholism more often causes parenchymatous inflammation, but we may have associated with it the interstitial form. The peripheral nerves are especially affected, and usually primarily. Both parenchymatous and interstitial changes are usually present. The lesions, as a rule, are symmetrical. The axis cylinder more rarely escapes than in lead, gouty, and rheumatic conditions. The cranial nerves are the seat of like changes, and the optic nerve may show granular degeneration and congestion with œdema. These changes are not observed in acute cases.

Symptoms. Insanity may occur in both acute and chronic alcoholism. Acute alcoholic mania often results from periodical excesses or from the sudden withdrawal of alcohol. The system is overwhelmed with the poison. Associated with this as a direct cause is exhaustion from malnutrition. The symptoms of *mania a potû* are too familiar to require a description in detail. Tremor is a marked feature, involving the extremities, and also the muscles of the face and tongue. There is excitement or delirium, accompanied by hallucinations of sight and hearing. The patient is constantly talking with imaginary persons. Sensory disturbance due to peripheral nerve irritation, not sufficient to cause loss of sensation, produces paræsthesiæ which frequently give rise to illusions of animals moving over the skin. It would seem also that many of the hallucinations of sight may owe their origin to the same cause.

Inco-ordination of voluntary movement is one of the earliest results of the abuse of alcohol, as shown in ataxia of the upper and lower extremities. It may affect the muscles of the eyes, causing vertigo. In one patient with marked vertigo the furniture in the room, such as the tables and chairs and pictures, all seemed to be rising from their places, so that he constantly requested that they be held down. Coma is at times complete, the breathing stertorous, and the reflexes lost. The patients can usually be aroused, but pass quickly back into unconsciousness. In the subacute or chronic state the condition may resemble general paralysis of the insane, with marked tremor, excitement, and delusions of grandeur.

Chronic Alcoholic Insanity. This is probably due to the secondary effect of alcohol on the vessels, leading to arterial degeneration and consequent organic changes in the brain structure, already alluded to. It tends toward dementia. There are most commonly present delusions, especially of suspicion of infidelity of wife or husband, delusions of persecution, with fear of poisoning and dread of impending evil. The prognosis is necessarily unfavorable.

Pachymeningitis. This is a common condition in chronic alcoholism. The dura and pia are involved, and the vessels diseased. The symptoms are those of irritation, stupidity, loss of memory, and frequently convulsions. There may be meningeal hemorrhage, usually subdural, accompanied by convulsions, hemiplegia, and coma. The differential diagnosis from intra-cerebral hemorrhage is difficult. A previous history of alcoholism and convulsions, especially if limited to one side, perhaps with a history of a slight injury to the head, will aid us. These symptoms may result long after the use of alcohol has been given up. An early diagnosis becomes important where an operation for the relief of the meningeal hemorrhage is contemplated. In a patient recently under observation, with an alcoholic history, and paralysis and convulsions affecting one side, the symptoms were entirely removed by trephining, a large subdural hemorrhage being found. See also Chapter XIII.

Epilepsy. Epilepsy is frequently the result of the meningeal complication. Epileptic seizures may occur during the excessive use of alcohol, and the epileptic condition become established, although alcohol be afterward abstained from. Children of alcoholic parents are especially subject to idiopathic epilepsy.

Spinal Diseases. Alcohol is rarely the direct cause of inflammatory affections of the cord; however, disease of the anterior horns and degeneration of the white tracts of the cord may occur. It is probable that some of the cases of alcoholic neuritis with symptoms resembling locomotor ataxia are also associated with disease of the posterior columns. Pal refers to a case with degeneration of Lissaur's tract in the lumbar region and of Goll's tract in the cervical region. Capillary hemorrhages may occur. A case resembling Landry's paralysis gave the following post-mortem lesions: hyperæmia of the cerebellum, medulla, and cord, with capillary hemorrhages in the posterior horns in the lumbar region, and in the anterior horns of the cervical region, third to sixth segment, with degeneration in the vagus, both phrenics, and the right sciatic nerve.

Chronic diseases of the cord, meningitis and myelitis, are more common. Without doubt alcohol acts as a predisposing cause to various lesions, especially if associated with lead or syphilis. As already seen, the degenerative influence of alcohol is not confined to the peripheral system. Buzzard indeed holds that the changes in the nerves are secondary to lesions produced by alcohol in the vasomotor centres of the bulb and cord, leading thus to anæmia and hyperæmia, and then to degeneration or inflammation.

Neuritis. Multiple neuritis is the form most usually found in connection with alcohol. The lower extremities are first involved, and later the upper. There is usually marked foot and wrist drop with atrophy. Pain is present, especially called forth on pressure over the muscles and along the nerves. Electrical reaction to faradism is lost, and partial or complete reaction of degeneration to galvanism is present. The reflexes are absent. The course of the disease may be very rapid, suggesting, as in one case referred to, Landry's paralysis; or, again, it may be very slow, resembling tabes. In severe and fatal cases the vagus and phrenic are involved. Contractures are common from overaction of the muscles not involved. The legs are usually flexed at the knee, and the hands present the claw-shaped appearance. There may be marked trophic changes present, as perforating ulcer, œdema, erythema, falling off of the nails, bullous eruptions, bed-sores, etc. Rarely do we find the bladder or rectum involved. The cranial nerves are frequently affected, although these lesions are comparatively rare, considering the large number of cases of neuritis. Ophthalmoplegia externa, in which the third, fourth, and sixth nerves are involved, has been observed. Thomsen refers to a case with degeneration of the neuclei of the sixth, fourth, third, and hypoglossal nerves. Mental symptoms are usually present, manifesting themselves frequently by loss of all idea of time and place. In such cases there is probably degeneration of the cells in the cortex of the brain and the association fibres. The pathological changes, as already stated, are those of parenchymatous and interstitial inflammation of the nerves. See also Chapter XXV.

Prognosis. For the prognosis of the diseases caused by alcohol the reader is referred to the various chapters in which they are considered in detail. The prognosis in neuritis is generally favorable, complete recoveries occurring where paralysis has existed for six months or a year. Naturally, however, if the vagus or phrenic is involved, cardiac failure or respiratory disease may cause a fatal termination.

Treatment. The general treatment of alcoholism consists first in the withdrawal of the poison. Acute conditions require sedatives to produce sleep. The usual exhibition of the bromides with chloral and digitalis is often the most effective. Somnal, trional, and sulphonal are to be used for the insomnia in preference to morphine. However, in very many cases they prove inefficient, and then sleep and cessation from delirium are best obtained by hypodermic injections of morphine, or of morphine with hyoscyamia, or hyoscine hydrobromate. Strychnine in large doses has been used with good results; however, in my experience the moderate exhibition of the drug—one-fiftieth of a grain—with digitalin, is to be recommended. Nourishment must be given as soon as possible. In neuritis, absolute rest, relief of pain—by antipyrine and phenacetine, and the exhibition of strychnine, with the use of massage and electricity as early as possible, and attention to the relief of deformities, constitute the appropriate treatment. Hypodermic injections of strychnine do not seem to offer greater advantages than the administration of the drug by the mouth, and there is some danger of suppuration following from the lowered trophic condition which exists.

MORPHINISM.

Morphinism may be defined as the morbid craving for morphine as a stimulant.

Etiology. The habit is usually acquired, at least in the Western Hemisphere, from the use of morphine to relieve pain, insomnia, etc. The predisposition varies with the individual. Hereditary influence is of great importance. Especially predisposing are the various neuroses, such as neurasthenia and lypemania. This is also true of phthisis and other acquired and inherited degenerative conditions. "The offspring of the victim to the morphine habit has a condition of the nervous system such as once subjected to the exciting cause develops the tendency rapidly" (Hoppel). The employment of morphine in intense pain, which occurs periodically, does not in my experience predispose to the habit; nor in these patients is the usual physiological [illegible] exaltation present. A not infrequent cause of the habit is dysentery or other intestinal disturbances of a chronic nature. Here the drug may be taken by the rectum, and will often be continued by that method. Its abuse is often associated with that of other drugs, especially alcohol, and of late years with cocaine.

Pathology. The pathological changes are not definite. In no case can we ascribe them directly to the drug. Russell, however, holds that after a few years organic visceral changes are set up.

Symptoms. The habitual use of the drug seems to establish an unstable condition of the nervous system. Mentally, we observe vacillation and lack of energy, apathy and procrastination unless the stimulant is taken. The will power is lost, and the patients become forgetful and unreliable. The moral nature is changed, and to procure the desired drug patients will lie and [illegible]. They are always to be distrusted. This is not peculiar to morphinism, but is found in all victims of drug habits. There is rarely a pleasurable [illegible] accompanying the taking of morphine in these cases; that is, [illegible] agination and pleasant memories are not present, nor does sleep result. Any great excesses occurring in the course of chronic morphinism may give rise to acute maniacal symptoms, hallucinations, and delusions of persecution. This was the case with an habitual morphine-taker, who, on what he called his "morphine drunks," claimed to have taken sixty grains in twenty-four hours. The most marked nervous symptoms are observed on the withdrawal of the drug. There is intense anxiety, a dread of some impending evil; the person may even become maniacal. There is marked insomnia and restlessness, with depression, and frequently suicidal intent. The mind is ultimately affected with a form of dementia, with chronic delusions of persecution. We can hardly say, however, that we have a distinct form of insanity due to morphinism. Certain it is that in many cases excessive habitual use of morphine is carried on for years with no mental deterioration.

Motor Symptoms. The physical signs of the disease are tremor and more or less paresis, which, indeed, in the more chronic cases is constant, but appears only in those of more recent date on the withdrawal or decrease of the amount of the drug. There is at times ataxia. The latter symptom was specially marked in a patient in whom the amount of morphine had been reduced from six grains a day to one-eighth of a grain. The reflexes are not usually affected.

Sensory Symptoms. Neuralgia of any special nerve is rare, but pains, diffuse and neuralgic in character, are common. This is frequently observed where the drug has been given a long time for some special pain, as in locomotor ataxia or neuralgia, making it difficult in such cases to differentiate the real pain from those of the drug. Possibly a neuritis is at the basis of this condition. Vesical and rectal paresis are common. Constipation or diarrhœa may be present. The pupils are usually contracted; this symptom is more decided when the patient is under full physiological effect of the drug. Vasomotor disturbances manifest themselves by a tendency to profuse sweating. This symptom also is chiefly noticeable on decreasing the drug. The pulse is rapid and compressible. General nutrition fails, so that emaciation is common. Trophic changes are present, as shown by a tendency to the formation of abscesses at the site of hypodermic injections.

Diagnosis. The diagnosis is not difficult. The tremor, the mental condition, the pupils, and the sweating are characteristic, especially when considered along with the knowledge of the habits of the patients.

Prognosis. The prognosis is almost always unfavorable as regards a cure of the habit, although its regulation and moderation can often be established.

Treatment. The various modes of treatment are: 1. Instant withdrawal; 2, gradual withdrawal by daily decreasing the dose; and, 3, the rapid withdrawal within a few days. I have seen all these methods successful. There is always great danger in asthenic cases from cardiac failure when sudden withdrawal is practised. In a patient addicted to the habit for many years several weeks after total withdrawal death suddenly occurred from heart failure. The autopsy revealed a fatty heart and a contracted kidney. The heart must be constantly watched, and, on evidence of failure, strychnine, digitalin, or nitroglycerin administered. The substitution of other drugs, such as chloral, codein, or cocaine, is not to be recommended. I believe in the gradual withdrawal, and that this is best carried out in some institution away from home. An important point to secure permanent recovery is to have the patient report regularly to the physician, as the tendency to relapse is common. It is not as difficult to entirely cut off the morphine as to prevent its being again used when the patient is exposed to the worry and cares of life.

CHLORALISM.

The habit is often acquired from the employment of the drug to produce sleep.

The symptoms of acute chloral poisoning indicate paralysis of the vasomotor centres. In severe cases death occurs from heart failure, with preliminary symptoms in the early stage of excitement and delirium, but later dyspnœa, vertigo, paralysis of the lower extremities, and paræsthesias. In chronic cases of prolonged use of the drug there may be marked nervousness, insomnia, mental weakness, depression to the extent of melancholia, tremor, general weakness, and palpitation. The sudden withdrawal of the drug causes an increase of these symptoms. Excessive nervousness and dyspnœa, with cardiac palpitation, may come on, and this may even pass into mania or melancholia with great agitation. The cerebral anæmia induced in chronic conditions results in dementia.

The somatic signs are those pointing to vasomotor disturbance, as shown in the lividity of the face and neck, and the appearance of urticarial erythema in the skin. There may be excessive diarrhœa from the same cause. The prognosis is largely dependent on the association of the habit with the use of other drugs. It also depends on the predisposition or hereditary tendency, as shown by the presence of acquired or inherited neuroses. Treatment consists in stimulation with coffee and strychnine, the use of tonics, and supporting measures generally.

For habitual users of the drug its gradual withdrawal is preferable—with constant cardiac stimulation, and combatting insomnia by less harmful drugs, as sulphonal, etc., with massage and electricity.

COCAINISM.

Etiology. The habit has been largely caused by the frequent use of cocaine in nose and throat diseases.

It is, however, rarely acquired unless there is a strong neuropathic tendency. The use of cocaine is common among those addicted to other drugs, as morphine, alcohol, chloral, etc. It is either taken as a substitute in the attempt to break up a former drug habit, or more often in the desire to find some new stimulant. The cocaine habit rarely exists alone. Medical men are said to be especially prone to it.

The production of toxic effects depends somewhat on the manner of administration. The hydrochlorate of cocaine has greater toxic effect than the phenate. Also when it is applied over a large area of the mucous membrane the results are more dangerous.

Certain persons have an idiosyncrasy toward cocaine—small doses producing almost immediate toxic effects. Again, on the other hand, excessive amounts may be taken before a result is produced.

Pathology. It is difficult to define the changes solely due to its chronic use, as many other causes are usually present capable of producing morbid conditions.

Degeneration in the ganglion cells of the medulla and spinal cord have been noted, and the coats of the arteries have been found diseased.

Few cases come under observation for autopsy, and as the symptoms are largely of a functional character we would hardly expect much organic change, especially when complications, such as the habitual use of other toxic agents, are absent.

Symptoms. Locally, paralysis both of the motor and sensory produced. The cerebral symptoms seem to depend on the vascul ance. There is usually excitation, the person becoming brilliant a This may be observed especially in those who have become addi use for nasal or throat complications, and is probably explainal ground of the relief of the arterial congestion present and the pri diac stimulation. The pupils are dilated.

Its persistent use causes early loss of mental and moral cont quently hallucinations of the special senses—hearing, sight and sn be present. There may also be delusions of persecution and delirii pulse becomes rapid and feeble, and vasomotor disturbances, show selves by sweating, are common.

In one case under my observation tinnitus aurium was constant subject to a paroxysmal increase, ending in slight epileptic seizures partial loss of consciousness was present. The convulsive seizures we limited to the muscles of the eyes, there being bilateral oscillati eyeballs with dilatation of the pupils.

There is evidence of gradual mental deterioration and loss of sel Zanchewski regards sexual depravity and loss of the sense of the time as important symptoms. He further says that cocainism may in chronic paranoia.

It is difficult to separate the various symptoms due to the use drugs, especially alcohol and opium, as in them we also find delusio picion and persecution, and the same tendency to deceit and lying the needed stimulant.

Prognosis. The prognosis is favorable when we have not to other habits.

Treatment. When the drug is stopped, the symptoms, under sti and sustaining treatment, strychnine, sparteine, digitalis, disappear. electricity, and exercise are indicated. Again, treatment at a sa away from home is advisable. I see no advantage in sudden wit believing, as in morphine, that the gradual decrease of the amou causes less constitutional disturbance, and is more lasting in its effe only applies when the patient can be treated away from home, or special observation. If he continues at his ordinary occupation it i cut off the drug as rapidly as possible, and in these cases strychnin useful, producing a feeling of rest and strengthening the pulse. La can be taken—$\frac{1}{6}$ of a grain, in divided doses, daily.

LEAD POISONING.

There is a marked tendency to the selection of nerve tissue by lead cially is this seen in the peripheral nerves. The brain and spinal c however, also affected. We have two conditions to consider: *First,* intoxication or overwhelming of the nervous system by lead presen blood, a condition which can be compared to acute alcoholism; and, a chronic absorption of the poison, which produces a class of sympt pendent to a large extent on degenerative changes induced in the system, and secondarily on the central and peripheral nervous system

Etiology. The usual cause of lead poisoning is exposure in the occupations in which the metal is employed. It is inhaled as d absorbed through the skin and intestinal tract by those occupied manufacture of white lead, by compositors, painters, etc. Lead pois also produced by the use of certain cosmetics and hair dyes, thou does not occur as frequently at present as formerly. Another source of poisoning is the drinking of water which has been allowed to stand in tanks lined by lead or in lead pipes.

Symptoms. *Insanity.* Mental disease may present itself in the following forms, according to Dr. Raynor, viz.: (1) that of coarse-lead poisoning, producing attacks of acute mania and conditions resembling general paresis, such as Tanquerel described under the term "lead encephalopathy;" (2) cases of minute and protracted lead intoxication, producing slowly-developing sensory hallucinations, noticeable by the absence of delusions of persecution, and by the persistence of hallucinations of vision; and (3) cases in which there is some coarse toxæmia. In the first form the gait and symptoms may closely resemble general paresis. It is not infrequent to find the alcohol habit associated with lead poisoning, so that the symptoms are frequently a combination of those belonging to both states. Probably the alcohol favors the more ready absorption of the lead. Certainly, in countries where malt liquors are largely used such symptoms are more frequent. While the symptoms in these cases often resemble general paresis, they differ in many respects from genuine cases of this disease. As a rule, we do not find the marked exaltation with delusions of grandeur. Delasauve, as far back as 1851, speaks of *pseudo-paralysie générale saturnine.* True general paralysis may, however, be caused by lead. Dr. Savage refers to a case of acute mania with hallucinations of sight and hearing, and finds three forms due to lead: (1) acute mania; (2) conditions of hallucinations; and (3) a condition resembling general paresis.

General Cerebral Affections. These manifest themselves in acute and chronic forms. In the former we observe cephalalgia, vertigo, epileptic convulsions, delirium, tinnitus aurium, diplopia, hallucinations of sight and hearing, and delusions of persecution with mania. Severe cases pass into coma. Chronic conditions are more liable to take the form of melancholia, with delusions of persecution, and a tendency to suicide.

Tanquerel divided saturnine encepalopathy into four forms: (1) the delirious; (2) the comatose; (3) the convulsive; and (4) the mixed. He found cerebral atrophy and an increase of the fluids in the brain usually present.

O'Carroll reports several cases, in one of which there was right hemiplegia with a tremor and aphasia. Urine normal; death with convulsions. Autopsy showed œdema of the brain, increase of fluids, with flattening of the convolutions. The cells of the cortex were not atrophied, but were pigmented.

Two theories for the production of the symptoms have been advanced: first, the direct influence of the lead in the small arteries and capillaries of the brain, causing anæmia, or secondly, œdema of the brain, causing compression of the capillaries. Probably both causes are active.

Hemiplegia. Hemiplegia following cerebral hemorrhage or softening may occur, due to endarteritis of the cerebral vessels. Not infrequently nephritis is a complication, and may give rise to accompanying uræmic symptoms. Convulsions of a hystero-epileptic type with hemianæsthesia and functional hemiplegia may be present. These transient conditions may also involve the optic nerves, causing amaurosis or even hemianopsia, and seem to point to a direct action of the poison. Permanent anatomical changes may occur in the optic and other cranial nerves.

Acute cerebral conditions may supervene in the course of chronic infection, and this has also been observed when the iodide of potassium has been given. Dr. McDonald reports that among those employed in the New Castle Lead Works, where previously no symptoms had manifested themselves, the exhibition of iodide of potassium induced marked symptoms of lead poisoning—convulsions, coma, etc. While epileptiform seizures are not infrequent, it is not probable that essential epilepsy is caused by lead. Hysteria is frequently present, but can hardly be considered as being due to the poison. In such cases it is probably latent, and only called out by the poison acting as an exciting agent.

Spinal Diseases. Poliomyelitis, chronic or subacute, is not uncommon. The disease is subsequent, as a rule, to the peripheral nerve affection, but may be primary. We find also diffuse scleroses of the nerve tracts of the cord. These degenerations are probably due to the changes in the bloodvessels, and the resulting malnutrition. Ascher holds that it may be through the cord that the nerves are affected. He reports a case of a typical character, in which there was paralysis and wasting of the forearm of the right side and paralysis and wasting of the shoulder on the left side. (*Berliner klinische Wochenschrift*, 1893.) He apparently considers the case as one of poliomyelitis.

Peripheral Palsies. The peripheral nerves are, as a rule, the first part of the nervous system to be affected by lead, and this is especially true of those supplying the extensor muscles of the hand and fingers. The selection may depend upon the more extensive use of these members. The disease is generally symmetrical, commencing first in one hand, usually the right, causing wrist-drop, and then extending to the left hand. Double wrist-drop therefore results. The paralysis may involve the shoulder. Considerable atrophy is observed. While not infrequently the patients state that the onset is sudden, this is not the rule. The hand and fingers are usually flexed, and very little power of flexion can be called out unless support be given to the paralyzed extensors, when the flexors are found to be normal. The supinator longus and triceps usually escape. Cases occur, however, in which the supinators and shoulder muscles are involved, including the deltoid and biceps. We may have the median and ulnar nerves affected, resulting in a wasting of the hand muscles. This is particularly seen in the ball of the thumb and the interossei. The lower extremities are only exceptionally affected. At times in acute cases, paralysis may be general, involving the muscles of the trunk also. Tremor is present where paralysis is marked, but it is rather one of weakness than an intention tremor. Fibrillary twitching is observed in the muscles as they atrophy. Sensibility is not much affected. Deep pressure over the nerve may elicit pain. Associated with the wasting there is frequently some swelling over the wrist and metacarpal bones. Electrical reactions show complete loss of faradism, and generally a partial reaction of degeneration, although, in some cases, there is simply a reduction of the response to both currents.

Diagnosis. Lead paralysis distinguishes itself from alcoholic neuritis in the absence of the extreme tenderness of the muscles and nerves observed in the latter, and also in the manner of its onset, the upper extremities being the first and usually the only parts affected. In arsenical paralysis the parts effected are first the legs, and often the loss of power is limited to them. In lead poisoning there is at times a close resemblance to poliomyelitis and progressive muscular atrophy. However, the distribution and manner of onset of the paralysis differ. In the latter affections both the extensor and flexor groups of muscles suffer. The history of exposure to lead and the appearance of the characteristic blue line along the gums aid in establishing the diagnosis. In atypical cases, however, especially, in affections of the anterior horns, it is necessary to remember that disease of the cord may be present, and, indeed, in a few cases the primary disease is in the cord itself.

As we have seen, there is often great resemblance at times to general paresis, and lead may be the direct cause of that disease; there is rarely, however, the same exaggeration of ideas and delusions of grandeur and general feeling of well-being, and the tremors do not, as a rule, affect the muscles of the face and tongue. The diagnosis in cases where the cause is obscure, as when water has been used from a lead-lined tank unknown to the physician, or cosmetics have been used, is often difficult. Uræmia may be mistaken for it, especially as the kidneys are often involved. Flint and Lauder Brunton, indeed, ascribe many of the symptoms of lead to uræmia. Epilepsy cannot be distinguished from the essential form, except through the history of lead. Sufficient has been said to call attention to the difficulty of diagnosis in cases when the anterior horns of the spinal cord are involved. It resembles at times very closely progressive muscular atrophy. It is in the irregular or atypical cases, naturally, where the difficulty is greatest. In all cases of toxic poisoning by arsenic, mercury, and alcohol, it is now established that while the peripheral nerves are most frequently involved, the whole nervous system may be affected primarily, so that brain and spinal diseases may occur. These paralyses were, not long ago, always considered spinal, and now the pendulum is again swinging back to that view.

In all suspected but doubtful cases of lead poisoning the urine should be chemically examined for lead. The administration of iodide of potassium for a few days favors the success of the test.

Prognosis. The prognosis is usually favorable when the individual can be removed from the continued influence of the lead; but where degenerative changes have occurred in the bloodvessels, or nephritis complicates the case, the result must be unfavorable. Again, second attacks are longer in recovering. Over-exercise without renewed exposure may cause a relapse of the symptoms, especially of the paralysis. However, even when the reaction of degeneration has been demonstrated, there may be complete recovery.

Pathology. We find evidence of lead in the brain tissue. This has been clearly shown by Blythe. Chronic meningeal changes, such as pachymeningitis, clouding of the pia with serous effusion in the subarachnoid space and ventricles, with cerebral atrophy, are frequently present. Atrophy of the optic nerve with evidence of interstitial neuritis is not uncommon, as well as affections of the fifth and seventh cranial nerves. The cord shows degenerative changes involving the anterior horns, in which we find atrophy of the nerve cells, with loss of their processes, and an increase in the neuroglia tissue. The vessel walls are thickened. The peripheral nerves are the seat of a parenchymatous and interstitial nephritis, the latter being the most frequent condition. The axis cylinder is usually not affected. Stieglitz found, in experiments on rabbits with lead, atrophic changes in the anterior horns, vacuolation of the ganglion cells, and corresponding degeneration in the nerve roots and peripheral nerves. In a case reported by me, atrophy of the cells in the anterior horns in the dorsal region and degeneration in Lissauer's tract and in the column of Goll were observed. The meninges are also thickened and the vessels are the seat of endarteritis. Buzzard's case, referred to by me, showed atrophy of the cells of the anterior horns in the cervical and dorsal regions. The author considers the case as one of progressive muscular atrophy, as the supinators had not escaped. However, the etiological factor in the case seemed to be lead, the patient being a sign painter.

In acute saturnism transient amblyopia without change in the optic nerve has been observed. However, neuritis going on to atrophy is not uncommon. The diagnosis of optic atrophy due to lead can only be made from the history of exposure to the poison, there being nothing diagnostic in the atrophy itself.

Treatment. This consists in the elimination of the lead by potassium iodide as rapidly as possible; if, however, acute symptoms supervene on the administration of the iodide it must be discontinued for a time. Opium relieves pain and also aids in the action of the iodide. Lead is passe the system through the kidney and the intestinal tract. Hot baths ar be employed. For the paralysis, strychnine and electricity, with mass indicated. Constipation, if present, should be treated by the admini of the sulphate of magnesia or sulphate of sodium, as they render suc poison as may still be present in the intestinal tract insoluble. Alu also be administered with the same end in view.

ARSENICAL POISONING.

Arsenical poisoning results from the absorption by the system of ar toxic doses.

Etiology. Arsenic is frequently introduced into the system in case tentional poisoning. Sometimes a toxic action follows its medicinal u number of cases of chorea have been reported in which arsenic has be in such large doses as to give rise to symptoms of poisoning. The san be said of cases in which various medicinal pastes containing arseni been used too freely. Arsenical poisoning also arises from the use of as a coloring matter in various arts and trades. It may occur fr atmosphere through absorption from wall-papers or tapestries. Much has been laid on these methods of slow poisoning by arsenic. Neurit consequent paralysis is, however, rare in these cases, most of the syn pointing to affections of the general nutrition. Arsenic has been fo the urine, however, thus proving the possibility of its absorption i manner.

Pathology. The usual changes are similar to those found in m neuritis. Parenchymatous and interstitial neuritis may be presen arsenical poisoning, as in poisoning by lead, the axis cylinder usually es especially when the poison has found an entrance by gradual absor Popoff found, in the case of a man dead of acute arsenical poisonin ease of the spinal cord in which the cells in the anterior horns ha their processes, and in which capillary hemorrhages were present. lesions are rare, and are usually the result of acute poisoning. How these results correspond with the changes found experimentally in the g pig, in which a few hours after the exhibition of arsenic there was evi of myelitis with hemorrhage.

Symptoms. *Cerebral Symptoms.* The brain, as a rule, escapes any del ous effects, and few symptoms can be ascribed to it. In acute cases, ever, loss of memory and mental confusion with delirium may be prese

Spinal Affections. The spinal cord is subject to degenerative ch as in poisoning by lead and alcohol. Myelitis may occur in the more forms of arsenical poisoning.

Peripheral Affections. The peripheral nerves are the chief seat c disease. The symptoms present themselves in the form of multiple ritis. A feeling of numbness or tingling is first noticed in the lowe tremities, and this is soon followed by more or less weakness, devel into actual paraplegia. The disease extends toward the upper extre involving the extensors of the forearm and hand—a similar distribut that seen in alcoholic neuritis, although more apt to remain limited lower extremities. There is considerable atrophy of the muscles with d ished response to faradism and galvanism, but there is rarely the re of degeneration. In marked cases there is loss of the reflexes, and tremor, though usually slight, is present, and is dependent upon the cular weakness. Contractions causing deformities, as talipes, or the claw-shaped hand, may occur, due to the contraction of unopposed muscles.

Sensory Disturbances. The sensory disturbances are of the character of diminished appreciation of pain and touch, with areas of hyperæsthesia and anæsthesia. On deep pressure over the nerves pain may be elicited, though it is rarely extreme. As a rule, there is no marked cutaneous hyperæsthesia. Ataxia is commonly present, and trophic changes following the course of the nerves, as herpes, have been observed. In the variety of cases where there is a slow and gradual infection, as from absorption through the atmosphere from tapestries and wall paper, there are associated symptoms, in which depression, general nervousness, prostration and cardiac weakness are marked, and where there are few signs of neuritis. There is loss of appetite, and, in fact, many symptoms of gastric irritation. These frequently disappear on change of surroundings, only to be renewed on return of the patient to the place of original infection, Their insidious character renders the diagnosis at the time very difficult.

Diagnosis. When the motor symptoms are not well defined the disease may be confounded with tabes, *pseudo-tabes arsenicosa.* In obscure cases, when a possible history of absorption from wall-paper and tapestries only is present, the general character of the symptoms indicating cerebral neurasthenia renders, with the absence of neuritis, the diagnosis very difficult, and the latter can only be made positive by the detection of the arsenic in the urine. Neuritis can be distinguished by the usual distribution of the paralysis, sensory disturbance, and by the condition of the reflexes from myelitis chronica or poliomyelitis. The vesical paralysis in the former and absence of sensory disturbances in the latter serve as additional points of differentiation.

Prognosis. The prognosis is favorable, other things equal, when the cause can be removed.

Treatment. The treatment consists in the exhibition of strychnine, the use of massage and electricity, and the removal of the cause of the disease. For the elimination of the poison iodide of potassium should be exhibited.

CHRONIC MERCURIAL POISONING.

Chronic mercurial poisoning results from the absorption of mercury in toxic doses.

Etiology. The usual cause is the absorption of the poison in connection with the various occupations in which mercury is employed, and especially where workmen are exposed to its fumes, as among those engaged in the manufacture of rubber. Among workmen in the mirror factories and among gilders and miners mercury is sometimes absorbed through the skin. Mercurial poisoning may follow the use of the metal as a medicine, as in the application of various ointments, in its internal administration, hypodermically, or by the mouth.

Pathology. The pathological changes are of the degenerative type, and are non-inflammatory. The peripheral nerves show segmental atrophy. The myelin is disintegrated, but the axis cylinder is rarely involved. There may be optic atrophy (Wising). Arterio-sclerosis is present, and in chronic cases we find, as in other chronic metallic poisonings, cerebral œdema, and increased fluid in the subarachnoid space and in the ventricles.

Symptoms. *General Symptoms.* Among the employés in factories who are subject to slow infection, a series of symptoms pointing to exhaustion of the nervous system are common. A loss of self-control is observed, and an increased emotional state, with a tendency to cerebral excitement, irritability, or marked depression. There is also more or less impairment of the approaching in extreme cases dementia.

Motor Symptoms. Tremor is observed, especially in the hands ai but later on it involves the muscles of the face, tongue, and lower ties. This may continue for years without increasing appreciabl tremor is at times convulsive in character, especially if the patient tionally excited. In extreme cases any act, such as eating, may these spasmodic attacks. This variety rarely comes under the ph observation except in the mining districts.

There is also muscular weakness, which may be general, or m chiefly the extensors. Again it may be localized, affecting the u tremities on one or both sides, or it may take the form of hemiplegia. bault reports a case of hemiplegia with hemianæsthesia. The tren be limited to the paralyzed part. Atrophy is rarely marked. The are usually flaccid, and the electrical reaction, although reduced, s qualitative changes to the galvanic current. The reflexes are gener mal, but may be exaggerated at times, and at others diminished The vasomotor changes are present, as indicated by extreme sweati many cases there are functional disturbances alone, resembling l This is especially observed in the emotional states and in cases whe plegia and hemianæsthesia are transient.

Sensory Disturbances. The sensory disturbances consist in the lowering of the sensibility to heat, touch, and temperature. They fol course of the paralysis, which would seem to indicate that the affectio to the peripheral nerves.

Special Senses. The special senses are often involved. Sometim is amblyopia. In some few cases there is atrophy of the optic nerve. tory hyperæsthesia is sometimes present.

Diagnosis. In lead and alcoholic poisoning the distribution paralysis is different, the tremor is never as well marked. Multiple s may be mistaken for it, but in mercurial poisoning we rarely find nys present, or the exaggeration of the reflexes, and the spastic states. In p agitans the tremor is controlled rather than increased by voluntary a

Prognosis. The prognosis is good, other things equal, when the can be removed from the source of infection. The longer a patient h exposed, the less favorable are the results.

Treatment. The treatment consists in the exhibition of the io potassium and the use of baths, massage, and electricity. Sulphur ba no longer recommended.

CHAPTER VI.

DISEASES THE DIRECT OR INDIRECT RESULT OF INFECTION.

By WILLIAM OSLER, M.D.

CEREBRO-SPINAL MENINGITIS.

Definition. A specific infectious disease, occurring sporadically and in epidemics, characterized anatomically by inflammation of the brain and spinal cord, and clinically by an exceedingly irregular course, the chief symptoms being fever, pain in the head and back, muscular spasms, and, in severe forms, delirium and coma.

Etiology. The history of the disease during the present century, from the date of the first recognized epidemic in Geneva, in 1805, is fully given in the works of Hirsch, of Stillé, and of Joseph Jones.

Children and young adults are most susceptible to the disease, but in some epidemics adults have been chiefly attacked. Males and females appear to be equally liable. The most severe epidemics have been in country districts. In 1873 the disease was very prevalent in the valley of the Ottawa River, and the villages and country districts suffered much more severely than did the cities of Ottawa and Montreal.

The affection has broken out simultaneously in regions far distant from each other. The concentration of population, as in large barracks and workhouses, favors the development of the disease; and in France during the fourth decade the numerous epidemics were almost confined to military hospitals. The outbreaks have occurred most frequently in the winter and spring, and have developed in exceptionally severe weather. Some writers have laid great stress on excessive moisture as a factor. The most serious outbreaks have been in towns and villages with very defective sanitary conditions. Poverty and overcrowding, with the coincident misery and squalor of large families dwelling together in small, imperfectly ventilated houses or in tenements, favor the development of the disease. Overexertion, as in prolonged marches, has been found by military surgeons to have an important influence.

The disease does not appear to be directly contagious. It is exceptional, indeed, to have two cases in the one house, and physicians and nurses are rarely attacked. On the other hand, there is very strong evidence in favor of the view that the poison may be transmitted by individuals from one place to another. Several striking instances of this are reported by Hirsch.

Evidence is accumulating in favor of the view that the micrococcus lanceolatus bears an etiological relation to the disease. Its presence has been demonstrated in the exudate of the meninges in many epidemics. It is also present in the meningitis secondary to pneumonia, and it has been found now in many instances of sporadic cerebro-spinal meningitis. The same organism has also been demonstrated in the meningitis developing in the course of diseases other than pneumonia and in that following injury. Altogether, the bacteriological observations of the past ten years point to the association of the micrococcus lanceolatus with both the sporadic and the epidemic forms of the disease. Other organisms have been met with in purulent meningitis, the staphylococcus pyogenes aureus, the typhoid bacillus, the colon bacillus, and other less definite forms.

There are insuperable difficulties in the way of a rational explanation of the conditions favoring the growth and development of the organism in the meninges. The micrococcus lanceolatus is a normal occupant of the body in a very considerable proportion of all individuals, at least 20 per cent., according to some authors.

The possibility of the disease being due to an auto-infection has been suggested through the nasal fossa by Strümpell and through the intestine by Flexner and Barker. There are serious difficulties, however, in the way of accepting such views which offer no explanation whatever of the epidemic prevalence, or of the remarkable facts quoted by Hirsch in favor of its transmission from one locality to another by a third person.

Morbid Anatomy. The patient may die before any inflammatory exudate occurs in the meninges, and then the condition is one of an extreme grade of hyperæmia and of a slight serous effusion. In well-developed cases on external inspection the petechiæ, sometimes herpes, may be noticed on the skin. On removing the skull-cap the dura is tense, and its inner surface hyperæmic; the sinuses full of firm clots. On exposing the cerebral cortex in severe cases the convolutions may be covered completely with a creamy exudate. Often this is patchy, most marked, perhaps, on either side of the longitudinal fissure and in the chief sulci. The cortical veins may be distended and prominent, and the smaller vessels of the pia are deeply engorged. Occasionally superficial hemorrhages are seen; the exudate is upon the pia mater; the arachnoid itself may be opaque. The effusion is usually abundant at the base of the brain about the chiasma and in the Sylvian fissures, and may cover completely the pons and medulla. The lesions are not confined to the meninges, but the cerebral substance is also involved, and the process is in reality a meningo-encephalitis. The gray matter is hyperæmic, juicy, and foci of infiltration and of hemorrhage may be seen. Abscesses of some size are occasionally found. The inflammation may involve the nerves at the base, which are surrounded with the fibrinous exudate. The ventricles may contain only an increased amount of serum, but in some instances the inflammation is most intense on the velum and choroidal plexuses. The ependyma is softened, ecchymosed, infiltrated with and covered by pus, and the ventricular contents may be of the same nature. In cases which have lasted for a long time there may be no longer any fibrinous exudates, but there are areas of meningeal thickening, adhesions, and most constant of all great increase in the serous effusion, which may cause great dilatation of the ventricles (hydrocephalus). Even in cases in which death has taken place so soon as the fifth week the ventricular effusion has amounted to three pints. The spinal meninges show the same lesions. Small hemorrhages are not uncommon. The exudate may be, in very acute cases, only a turbid serum, but more commonly a creamy, thick material, chiefly on the posterior part of the cord. Frequently it collects more particularly in certain regions, producing irregular bulgings on the arachnoid. The greater grade of exudation on the posterior surface is due entirely to the effect of gravity. In some instances the entire cord is imbedded in a thick, grayish-yellow lympho-purulent exudate. The substances of the cord may show the same changes as in the brain, namely, hemorrhages and infiltration with leucocytes, sometimes foci of such extent as to form small miliary abscesses. Microscopically the exudate consists of polynuclear leucocytes, lymphoid cells, larger cells with vacuolar nuclei, and even cells larger still, which in some places are very numerous, and even these contain other leucocytes and red blood cells within them. (Flexner and Barker.) The micrococcus lanceolatus may usually be demonstrated in the exudate. The changes in the other organs are those usually associated with fever. In very severe cases there may be hemorrhages on the serous membranes. The lungs often show changes, bronchitis, intense hyperæmia and œdema, and sometimes lobular, less frequently lobar pneumonia. Acute inflammation of the pleura and of the pericardium has also been found; endocarditis is rare; colitis may be present. The spleen usually varies in size according to the period at which death has occurred. It may sometimes be greatly enlarged.

Symptomatology. A stage of incubation with prodromal symptoms is rare. Loss of appetite, malaise, headache, pain in the limbs occur in a few cases. The onset, as a rule, as in pneumonia, is abrupt, and the patient, without preliminary symptoms of any moment, is seized with a chill or with violent headache, vomiting, and fever. Few disorders present so varied a symptomatology, and the cases are perhaps best described in groups characterized by special features.

1. *Fulminant form.* No acute disease—cholera scarcely excepted—may kill with such rapidity as cerebro-spinal meningitis. Cases are on record in which death occurred after an illness of ten hours, or even of five hours. This type is seen with variable frequency in different epidemics. In the recent one studied by Flexner and Barker ten patients died within forty-eight hours after the appearance of the symptoms. The onset is abrupt, with a violent chill, and without the slighest premonition the patient may be seized with agonizing headache, vomiting, high fever, active delirium, succeeded by great depression of the vital functions, gradual somnolence, sometimes spasms or rigidity of the muscles, or even general convulsions. Death may occur before the development of petechiæ on the skin, but in cases which last for more than twenty-four hours herpes and ecchymoses are almost invariably present. The fever in this so-called apoplectic type is not necessarily high. In many of the cases it has been quite moderate, 102° and 103°; the pulse may be rapid and feeble, but instances are on record in which it has been slow, falling to 50 or 60 in the minute.

2. *Ordinary form.* Commonly without any prodomal symptoms the disease sets in with severe chill, headache, and vomiting. The headache, usually severe and accompanied with great sensitiveness to light and to noises, may be diffuse over the entire head, or localized chiefly to the occiput or the forehead. It is one of the most constant features of the disease. The temperature rises rapidly and may reach 103° or 104°, sometimes higher, but occasionally even in severe cases the pyrexia is not at the outset or during the course of a high grade. The pulse is full and strong; later irregular, and when symptoms of depression occur feeble and rapid. The pyrexia, which has no fixed type, does not really bear any relation to the severity of the other symptoms. Vomiting, which occurs early, may cease within twenty-four hours, or in exceptional cases recurs throughout the course of the disease. An early and important symptom is a painful stiffness in the muscles of the neck and of the back, accompanied with aching, which often extends into the limbs. As the disease progresses, usually from the second to the fifth day, this stiffness becomes more marked, and there may be rigidity of the muscles of the back and neck, the latter causing marked retraction of the head. Opisthotonos is not common, but orthotonos, in which the trunk is rigid and firm, is not infrequent. Cases have been described in which the general rigidity and stiffness was such that the body could be moved like a

statue. Unilateral spasm of the back muscles, leading to pleurosthotonos, is rare. Except in early childhood and a short time before death general convulsions are not common. Tremor or clonic spasms may be present. Spasm of the muscles of the face may also occur. Lesions of the nerves at the base may cause paralysis of the muscles supplied by the third, inequality of the pupils, nystagmus, deafness, and disturbances of the sense of smell. Sighing, respirations, and Cheyne-Stokes breathing are met with in some instances. Intra-ocular changes are common, particularly passive congestion of the retinal veins and optic neuritis. Disturbance of sensation is common, particularly hyperæsthesia of the skin, and the patient may cry out when attempting to move the trunk or limbs. In part this may be due to hyperæsthesia, and sometimes to the tension spasm in the muscles. It may be very marked in the legs, and the slightest movement may be sufficient, even in a patient in a profound stupor, to cause reflex spasms.

The cutaneous features of the disease are important. The petechial rash, which has given the name "spotted fever" to the affection, is very variable. Stillé states that they were present in only 37 of 98 cases in the Philadelphia Hospital. With the ecchymoses there may be roseola and erythema, or these latter may occur alone. The distribution of the petechiæ may be symmetrical. In the epidemic at Lonaconing, studied by Flexner and Barker, the petechial eruption was comparatively rare, but an indistinct purplish mottling over the surface of the body was more common. Herpes is perhaps more frequent in this than in any other disease. It is seen first upon the face, either on the nose or lips, but often extends, and is symmetrical in its distribution. Urticarial forms of erythema, pemphigus, and in a few instances gangrene of the skin have been described.

As already mentioned, vomiting is an early and a prominent symptom. The tongue in protracted cases is dry and covered with sordes. Lacunar tonsillitis sometimes occurs. Difficulty in swallowing may be due either to the extreme retraction of the neck or to disturbed innervation. Diarrhœa is not common, but in four of the Lonaconing cases already referred to there was well-marked dysentery. The abdomen is somewhat retracted. Jaundice has been met with in a few instances. The spleen is sometimes enlarged, but, from the varying statements made with reference to it, it evidently is not a common feature. Of respiratory symptoms disturbed rhythm in breathing is common, particularly toward the close, and the respirations may be of the Cheyne-Stokes type. Epistaxis is a very frequent feature in some epidemics. Bronchitis, broncho-pneumonia, and pneumonia occasionally occur, and in the protracted cases there is much hypostatic congestion at the bases.

In the severer cases the urine is albuminous and may show the presence of hyaline and granular casts. Phosphates are often in excess, and blood may be present in the severer cases. Marked polyuria has been noted, and it has sometimes persisted for years. Glycosuria may also occur. The blood condition was carefully studied by Flexner and Barker, who found marked leucocytosis in all the cases. Neither the red corpuscles nor the hæmoglobin showed any special changes.

The course of the disease is extremely variable. More than one-half of the deaths occur within the first five days. Improvement is indicated by a fall in the fever, lessening of the spasm, and a return of consciousness. Convalescence may be extremely tedious, and after the acute symptoms have subsided may be interrupted by the complications and sequelæ to be mentioned shortly.

3. *Anomalous form.* (*a*) *Abortive type.* The attack may set in acutely with high fever, severe headache, photophobia, but in a few days all these symptoms subside and rapid convalescence is established. Strümpell distin-

guishes between the abortive form, setting in with great intensity, and the mild, ambulant cases, which have been described, in which the patients complain of headache, nausea, unpleasant sensations in the back and limbs, and stiffness in the neck. There may be no initial vomiting and very slight fever. Such cases could only, in reality, be recognized as due to the poison of the disease during the prevalence of an epidemic.

(*b*) *Intermittent type.* In this form the fever is of an intermittent type, assuming sometimes a quotidian, sometimes a tertian character, and in the intervals of the fever there may be almost complete freedom from the other symptoms of the disease. This is a form upon which we require further information.

(*c*) *Chronic type.* The attack may be protracted for two or three months, or even extend to six months, and may lead to the most intense marasmus. There are recurrences of the fever; thus Heubner gives an instance of a lad, aged seven years, who had repeated recurrences from the end of February until the end of June, and, though worn to a skeleton, he made a complete recovery.

COMPLICATIONS AND SEQUELÆ. Endocarditis and pericarditis are rare. Pneumonia, lobar or lobular, is a frequent complication in some epidemics. The percentage of cases is, however, very variable in different epidemics; in some a majority of the cases have presented this complication. It may be present only toward the close of an epidemic. Parotitis has been described, and occurred in a number of cases in the Lonaconing epidemic.

A remarkable complication is the arthritis first described by James Jackson, Sr., the number of cases varying considerably in different epidemics. In the Lonaconing epidemic twenty per cent. of the severer cases had joint affections, the knees, elbows, wrists, and ankles being involved. There were some cases in which, had it not been for the initial symptoms indicating a meningitis, the disease would have been diagnosed acute rheumatism. "Kernig has described a symptom which he thinks is pathognomonic of meningitis. In thirteen cases he observed a peculiar flexion-contracture (beuge-kontraktur) of the knee-joints, which could not be reduced when the patient was in the sitting position. In attempting to extend the knee, the leg could not be straightened further than a point where it made an angle of about 135° with the thigh, although when lying or standing this contracture was completely absent. If the patient lay on his side with the thighs drawn up the symptom was still present. It has been claimed that the same phenomena may be seen in many other conditions (old age, chronic alcoholism, etc.), but Kernig asserts that he has examined thousands of individuals with particular reference to this point, and has never found this contracture except in cases of meningitis."

The most important sequelæ are those affecting the special senses. Keratitis may develop with ulceration; less often iritis. The double optic neuritis may be followed by atrophy and blindness. Serious auditory lesions are still more common. Deafness may follow inflammation of the labyrinth, and in children this not infrequently leads to the condition of deaf-mutism. It is interesting to note that in the deaf-mute institution at Bamberg, of forty-two pupils in 1874, all had become deaf mutes from epidemic meningitis (von Ziemssen).

Mental feebleness and aphasia have occasionally followed the disorder. Headache may persist for months or years after an attack. Von Ziemssen regards chronic hydrocephalus as a frequent sequelæ, the symptoms being "paroxysms of severe headache, pains in the neck and extremities, vomiting, loss of consciousness, convulsions, and involuntary discharges of feces and urine." Paralysis of some of the cranial nerves may persist. Occasionally there is paralysis and wasting of the extremities due to multiple neuritis (Mills).

Diagnosis. During an epidemic the recognition of the disease is easy, and the description of the anomalous types is now so clear that they, too, are not often overlooked. On the other hand, mistakes frequently arise in the sporadic form, but cases of other acute infectious disorders are more often diagnosed cerebro-spinal meningitis than are cases of this disease overlooked. The disease must be recognized from

1. *Certain of the Acute, Infectious Diseases.* These are very likely to present cerebro-spinal manifestations which simulate those of the true cerebro-spinal fever. Typhoid fever, beginning with marked cerebral manifestations, delirium, tremor, and subsequently developing more or less rigidity of the muscles and retraction of the neck, may present a very deceptive picture. I have known at least three instances in which the diagnosis was made of cerebro-spinal fever, but post-mortem the lesions were those of typhoid fever, and the meninges presented only extreme congestion. The presence of rose spots, the development of tympanites and other intestinal features, and the gradual subsidence of the meningeal symptoms may lead to a revision of diagnosis. Many of these cases, unless an autopsy is secured, go into the mortality bills as cerebro-spinal fever. So also in typhus fever, the headache, backache, vomiting, hyperæsthesia, and the presence of roseola and petechiæ may for a time leave the practitioner in doubt. In certain of the severer types of smallpox the agonizing headache and the petechial rash may lead to the diagnosis of cerebro-spinal meningitis. A four-year-old child became ill suddenly with fever, pains in the back and head, and on the second or third day petechiæ appeared on the skin. There were retraction of the head and marked rigidity of the limbs. Cerebro-spinal meningitis existed in Montreal at the time, and both the physician under whose care the child was, and Dr. R. P. Howard, who saw it in consultation, agreed that the symptoms were highly suggestive of this disease. The cutaneous hemorrhages became more abundant, the spasm and rigidity were extreme, hæmatemesis occurred, and the child died on the sixth day. At the post-mortem there were no lesions of meningitis, and in the deeply hemorrhagic skin papules could be readily felt. The post-mortem diagnosis of smallpox was unhappily confirmed by the mother taking the disease and dying of it.

Other Forms of Meningitis. It is to be remembered that poliomyelitis may occur in epidemic form. In the remarkable outbreak which occurred in 1894 about Rutland, Vermont, and which is described by Caverly, of that town, many of the cases were thought at first to be cerebro-spinal fever.

(*a*) *Tuberculous.* Here the insidious onset and more protracted course are important points, and, as the meninges of the cord are not often affected, backache and rigidity and retraction of the head are seldom seen. The skin eruptions are also rare in tuberculous meningitis, and the presence of petechiæ and herpes is against the tuberculous form. On the other hand, local palsies of the ocular muscles, hemiplegia, and aphasia are more common, and, important of all, the determination of local tuberculous disease in other parts.

(*b*) *Pneumonic Meningitis.* As the membranes of the brain are chiefly involved there is commonly at first active delirium, frequently tremor and motor spasm, but not often great retraction of the muscles of the neck or back. In sporadic cases it may be very difficult to determine whether the pnuemonia has been a complication of the meningitis, or the meningitis a sequence of the pneumonia. There have been epidemics of cerebro-spinal meningitis in which a large majority of the cases were complicated by pneumonia.

3. *Certain toxic conditions,* particularly uræmia, may be associated with convulsions, rigidity and coma, and simulate in some degree cerebro-spinal menin-

gitis. The absence of fever and the conditions under which the symptoms arise, and in uræmia, the presence of albumin and tube-casts in the urine should make the diagnosis clear.

Prognosis. The mortality has ranged in various epidemics from 20 to 75 per cent. Hirsch states that of 15,632 cases 37 per cent. died. In children the death-rate is higher than in adults. Cases with deep coma, repeated convulsions, and high fever rarely recover. In the chronic form, even after the symptoms have persisted for months and there is extreme wasting with contractures, perfect recovery may occur.

Treatment. Considering the frequent association of the disease with filthy surroundings, an important prophylactic measure is the thorough cleansing of towns and villages in localities liable to the disorder.

As in other specific fevers careful nursing and feeding are the most important elements in the treatment. The room should be kept dark and thoroughly ventilated. The diet should consist of milk and strong broths. Many cases are very difficult to feed, and it may be necessary to use the stomach tube, or to resort to rectal injections. In the more chronic cases stimulants should be freely given.

In strong, robust patients with high fever and much mental excitement, abstraction of blood by wet cups, or even in suitable instances general blood-letting may be employed. The application of cold to the head and spine, which was recommended so strongly by the New England physicians in the first epidemics in this country, is of great service. The ice-cap to the head and the spinal ice-bag may be kept continually applied. With high fever and active delirium or coma the cold bath may be used, or cold sponging, or, if necessary, the cold pack. Counter-irritation is of doubtful benefit, and if applied at all the good effects are probably obtained by the light application of the Paquelin cautery.

Of the drug treatment of the disease we have no satisfactory knowledge. Opium has been much used by American physicians. It is particularly recommended by Stillé. Hypodermics of morphine may be used in reducing the violence of the muscular spasms. It should be freely used until the symptoms are controlled. Mercury, iodide of potassium, quinine, the salicylates, ergot, belladonna, and calabar bean are a few among the host of drugs which have been recommended.

TETANUS (Trismus—Lockjaw).

Definition. An acute infectious disease characterized by tonic spasms of the voluntary muscles, with marked exacerbations. As the disease shows itself first in the muscles of the jaw the names trismus and lockjaw have been applied to it.

The affection was well known to the ancients, and the descriptions given of it by Hippocrates and Areteus are particularly graphic. The disease commonly follows trauma (traumatic tetanus), but may occur spontaneously or after exposure to cold (so-called idiopathic or rheumatic tetanus), and, lastly, an important variety develops in newborn children (trismus neonatorum).

Etiology. The disease is more frequent in hot climates, and has prevailed extensively in the West Indies and in the Southern States of America. The mortality in some of the West India Islands from trismus neonatorum has been enormous. The colored races are more prone than is the Caucasian. The incidence of the disease in military campaigns has varied remarkably; thus in the Franco-Prussian War and in the Civil War in this country very few cases were observed. The disease may be endemic in certain localities.

Attention was early called in this country to its prevalence at the ea
of Long Island.

The disease occurs also in horses in which veterinarians have lo
nized its infectious nature, as cases are very apt to develop simulta
successively in the one stable. Cattle and sheep are also affected.

After the first month of life the disease is rare in children. I
most frequently in the third and fourth decades. Males are somew
frequently affected than females.

In a very large proportion of all cases there is a trauma. Since t
nized specific character of the malady, many now doubt if the di
occurs without a lesion of the surface, through which the poison may
duced. It may follow wounds of any kind, but is more common
tused or lacerated wounds, particularly when the nerves are involved.
of the extremities, particularly of the hand, are most liable to be
fected. The disease has also followed frost-bite and burns, the rem
tooth, and even the most trifling injuries, as the sting of an insect, o
splinter of wood. It is rare after surgical operations. Cases have
during the progress of vaccination. A special form of it is met wit
nection with the open surface of the uterus after parturition—the
tetanus—of which mention will be made later.

It is interesting to note that in a large proportion of all the cases t
been in the injury a possibility of contamination by the soil. "Sin
tion has been given to the point, it has been observed that in a co
proportion of the cases the injury involved contamination of the wo
soil, as in falls on the ground, a puncture by a broken stick or stak
had been in the earth, or by a splinter from a dirty floor. Such a
from the floor of a skittle alley, penetrating beneath the nail, has prod
one fatal case was due to a compound fracture of both femora, from
which the ends of the bones were covered with earth; gardeners h
fered from punctures by sticks. In most of these cases the tetanu
were found in the source of the contaminating material; their pres
plains the influence of these injuries. They have been found in spide
and tetanus has followed the application of such webs as a styptic (
lar custom in some places), and also the application of earth to a
(Gowers.)

The experience of the Civil War is interesting in connection with
stances of the disease in military surgery. Of 505 cases of tetanus
lowing was the distribution: upper limb, 137; distributed as follows
der, 31; arm, 37; coude, 7; forearm, 24; thumb, 4; hand, 37; low
292 cases: hanche, 2; thigh, 99; knee, 17; leg, 95; coudepied, 22; a
57. It is interesting to note that these figures do not confirm the usu
ment that tetanus more usually follows wounds of the hand than of t
The relative frequency of the disease after operations and wounds is
lows: in 29,980 amputations there were 116 cases of tetanus; in 465
tions there were 15 cases; and in 212,076 wounds there were 374 cas

The Tetanus Bacillus. The disease is caused by a specific or
discovered by Nicolaïer, and subsequently studied elaborately by K
The organism may be procured by inoculating an agar tube with pus
wound of a human being suffering from tetanus, which in the incub
35° to 37° C. shows the characteristic drum-stick bacilli. "The bacil
out into long threads at ordinary room temperature; but form the
teristic spores at 35° to 37° C. in the incubator in about twenty-four
They then appear as short, fine rods, with a large round knob on o
the knob constituting the resistant, glistening spore. The bacilli hav
pendent, but slow motion." (Bolton.)

The bacilli and its spores are widely spread in the soil of inhabited regions; of twenty-three specimens of soil taken from various parts of Copenhagen, sixteen proved virulent when inoculated into animals. The resistance of the organism is very great, and the virus has proved virulent in pus which has been dried for many months. The bacillus has been demonstrated in the dust from the floor of the wards of a military hospital.

Products of the Growth of the Bacilli. The filtrate of the culture three or four weeks old, entirely free from germs, contains the specific poison of tetanus, from which Brieger has separated two basic bodies, which are called tetanin and tetano-toxine. Brieger and Fraenkel have also separated a potent tox-albumen, but the true chemical nature of the poison has not been accurately determined.

With the products of the growth of the tetanus bacillus the disease is very readily produced experimentally, and comparatively small doses are required to kill a mouse—0.001 ccm. of the filtrate. Dogs, which are immune naturally, require a proportionately larger dose. The poison is not effective when administered through the stomach. The disease produced experimentally presents a picture similar to that seen in man. It is interesting to note that the cramps develop first in the muscles nearest the seat of inoculation. The effects are produced by the poison, not by the bacilli at the site of the inoculation. This is shown by the fact that the identical symptoms are produced by the germ-free filtrate of the pure culture, and also by the interesting experiments of Kitasato, who injected the tetanus bacilli into a mouse, at the root of the tail, and excised and burnt the seat of inoculation at various times afterward, as half an hour, an hour, an hour and a half, destroying in this way all the bacilli at the seat of inoculation. Only those animals in which the seat of inoculation was treated locally by incision and burning half an hour after injection recovered; the others died of the disease, showing that within an hour enough of the poison is absorbed to produce the symptoms and cause fatal results. There are several very interesting points still to be worked out about the production of the poison; thus it has been shown that when the cultures are grown at 20° to 22° C. they do not for several days produce any poison, and are no longer virulent. It is interesting, too, to note that these non-poisonous bacilli may become toxic when grown with other forms not capable of producing the disease. In accidental infection the local suppuration produced by other organisms inoculated at the same time may furnish the very condition favorable for the production of the tetanus poison. There is experimental evidence to show that the poison works like strychnine, and has its action upon the spinal cord.

Morbid Anatomy. The condition of the wound is variable. Very often the nerves in the locality have been found reddened and swollen, but in a majority of the cases they have been normal. No characteristic lesions occur in the brain or cord. Congestion of the bloodvessels, small hemorrhages, perivascular exudation, increased pigmentation in the ganglion cells have been described, but these changes are neither uniform nor distinctive. Minute ecchymoses are common on the serous surfaces. Œdema and hypostatic congestion of the lungs are frequently present. Rupture of muscle fibres may result from the intensity of the spasms.

Symptoms. Following an injury the first indications of the disease are usually manifest within ten days. In Yandell's statistics in two-fifths of the cases, and in Joseph Jones's statistics in four-fifths of the cases, the symptoms began before the fifteenth day. Slight stiffness of the neck and of the muscles of the jaw is the earliest feature, or the patient complains of difficulty in mastication, or that the movements of the tongue are not so free in talking.

In a few cases chilly feelings or even rigors may precede these ε and for a day or so there may be sensitiveness or even pain in tl Gradually the tonic spasm of the muscles increases until the co trismus or lockjaw becomes so marked that the jaws are separatec greatest difficulty. With the muscles of mastication those of the also involved, so that the angles of the mouth are drawn outward ward, producing the sardonic grin, *risus sardonicus*. With incr volvement of the muscles of the neck the head is drawn backwar muscles of the back become rigid, and the contraction may be enough to cause marked arching of the vertebral column. The body n remain perfectly straight, the condition known as orthotonos; or d upon the strength of the spasm of the different groups, opisthotonos, the back is arched like a bow; emprosthotonos, in which the bod forward; or pleurosthotonos, in which it is turned to one side. ' spasm may involve the muscles of the legs and arms, but as a rule and hands are not much affected. While the rigidity is tonic in there are frequent clonic exacerbations, which are apt to be e peripheral irritation, such as a draught of air, or a touch, or a nois exacerbations vary very much in frequency and severity, and they t proportion to the intensity of the disease. At first the rigidity is painful, but subsequently the suffering is extreme from the violent tion. In very severe attacks the thorax may be rigidly compresse muscles. The respirations are rapid, and spasm of the glottis m causing asphyxia. In the severe paroxysms sweating may be profu heart's action is increased during the paroxysm; the pulse may be fi to 160°. The temperature is very variable. It may be normal th or present only a very slight increase. In a few cases the disease is a with hyperpyrexia, particularly as an ante-mortem phenomenon, a are instances in which the body heat has reached from 110° to 11 urine is scanty, and according to Senator the amount of nitrogenou is not increased. There may be retention from spasm. The mind remains clear, except toward the close in protracted cases. Death m during a paroxysm either from asphyxia or cardiac dilatation. Th instance on record in which rupture of the walls of the heart occur ing the violence of the spasm. In other cases the fatal result is br by exhaustion.

There are certain varieties which are of interest. In the *head-t* Rose, which has followed in a majority of instances wounds of the f mus, dysphagia, respiratory distress, and facial paralysis are the pr features. On account, too, of the spasm in the deglutition musc consequent difficulty in deglutition, this form has been called also hydrophobicus. The cause of the facial paralysis is not known. Th be also paralysis of the eye muscles on the same side. This modifi not very common. Janin[1] has collected all the cases, thirty-one in up to 1892, and he regards it as a form in which the toxic mate chiefly in the medulla. The attacks recur at varying intervals, b may be almost continuous. In other cases they are extremely slig most serious attacks are those caused by attempting to take food, wh may be spasm of the muscles of deglutition and great interference w respiration.

Tetanus neonatorum, a rare form in temperature regions, and fort now rare in the trophies, was formerly very common in certain co particularly in the West Indies, where in some islands one-half of th drawn into a rigid straight line, strongly extended and immovable a statue, and will so remain immovable out of either of those postures, a considerable time, a minute or two." (Edition by Benjamin Rush, [illegible]

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[1] Paris Thesis. Du Tetanos Bulbaire, 1892.

In the Southern States of America the disease was formerly very common.

Puerperal tetanus is now fortunately rare, but formerly in some countries roved the most fatal of all forms of the disease. The disease begins, as a e, within five or ten days after parturition. In a number of cases it has wed abortion.

Whether tetanus ever occurs idiopathically has been doubted, but there cases on record, particularly in army practice, in which, without any arent external injury, the disease has developed after exposure to cold.

Mortality. The prognosis is always extremely grave, and two of the orisms of Hippocrates may be quoted at the present day, namely, "the sm supervening on a wound is fatal," and "such persons as are seized h tetanus die within four days, or if they pass these they recover." In traumatic cases the death-rate is not less than 80 per cent. In the cases ich develop without any wound the mortality is not nearly so high. From puerperal form recovery is excessively rare, and the form in infants is o very fatal. Death occurs from asphyxia, or from heart strain, which in e case has resulted in rupture of the wall. Favorable indications are late et, absence of fever, and localization of the spasms to the muscles of the ck and jaw.

Diagnosis. In well-developed cases following injury no possible difficulty uld arise. From strychnine poisoning, which presents very similar mus- lar spasms, it is differentiated by the following points: The anamnesis, e rapid development of the symptoms, and the greater extension and the lex spasms, as well as the fact that the jaw muscles in strychnine poison- g are not involved early if at all, and between the paroxysms there may be muscular rigidity. These points should suffice to distinguish it from rychnine poisoning. The question has been raised by the defence in murder ials, as in the celebrated Palmer case.

In hydrophobia, for which the head tetanus may be mistaken, there is no ecial spasm of the jaw, or any rigidity of the muscles, or paroxysms in hich the cervical and dorsal muscles are involved. In tetany the distribu- n of the spasm, chiefly in the hands and feet, the special involvement of splinter of wood, or from the earth in the part where the woun ceived, or portions may be inoculated into a mouse, which is the an susceptible, and which usually dies within a few days of the inocul

Treatment. *Immunization.* The present status of this question erence to tetanus may be thus stated: Animals which are very sli ceptible, such as the dog and hen, can be rendered immune by the of gradually increasing doses of the tetanus virus. The serum of a treated has the power of conferring immunity. For more suscep mals Behring employed the bouillon cultures, diluted with the a iodine trichloride, beginning with a culture containing 0.25 per cen percentage of 0.2, then a percentage of 0.15, and ultimately the culture. The serum of animals immunized in this way may be with 0.6 per cent. of carbolic acid, and may be used for immuniz animals not previously treated. The material used by Tizzoni and their observations upon man is the serum of the immunized dog trea alcohol, by which they obtained a material known as the tetanus an

Animals inoculated with fatal doses of tetanus poison treated serum of immunized animals have recovered. Up to the early part about a dozen cases of tetanus in man had been treated by the Catani antitoxin. The general expression of opinion by expert question of serum-therapy in tetanus is not althogether favorabl Klemperer and Levy in their recent works on clinical bacteriolog question whether a sure and undoubted result has been obtained in of the cases, as the healed cases were not so acute that the prognosis tirely doubtful. On the other hand, Remesoff and Fedoroff,[1] in report of a case supposed to be cured by the serum of an immunized claim in a review of the recorded cases that the duration of the di decidedly diminished by the treatment, that the temperature is redu attacks of spasms are less severe, sleep is promoted, and a very g provement in the general condition. Recent reports by Gilman Th and others are altogether more favorable, and the antitoxin should at the earliest appearance of the symptoms.

Surgical measures are usually employed at the site of the lesion, burning or excision of the scar. Unless done very promptly experi evidence, as mentioned above, would indicate that they are not of m portance, and this is borne out by experience. Excision of the nerve part and stretching have also been employed. Thorough cleansing a infecting of the wound, possibly excision, are the most justifiable re The general management of the case is most important. The patient be in a darkened room, attended by only one person at a time, and side communication should be forbidden. Veterinarians have long a ated the importance of complete seculsion, and in many of their well-eq infirmaries there is seen a brick, padded chamber, in which these ca treated. The question of feeding usually becomes very important, o the presence of lockjaw. The diet should be restricted to nourishing and milk, which usually can be taken through the teeth even when t spasm of the jaws. If necessary food can be introduced into the stom a catheter passed through the nose. The teeth have sometimes b tracted. In such cases we should trust for a time at least to the nutrit jections.

The drug treatment consists of the administration, in first place, of dies which relieve the spasm. For this purpose chloroform is the mos factory, though unfortunately the attacks recur as the effects pass ssium. The Calabar bean has been used successfully in some cases; also ne, which must be employed in large doses, as much as a quarter or half rain every hour or two. It is not, however, without danger. Among er means which have been recommended are the continuous warm baths, d the application of ice to the spine, and electricity and bleeding. In the erer spasms, leading to stoppage of respiration and cyanosis, artificial res- ation should be actively employed.

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[1] Centralbl. f. Bakt., 1894.

HYDROPHOBIA (Lyssa—Rabies).

Definition. An acute infectious disease of animals, dependent upon an known specific poison communicated to man by inoculation.

In animals the disease, known as rabies, is met with chiefly in the car- ora, particularly dogs and wolves. It is communicated from animal to imal by inoculation; hence, the extreme rarity of the disease in countries ch as North Germany, where the muzzling of dogs is rigidly enforced. It not infrequently communicated by inoculation to cattle, and occasionally horses and pigs. It is said that in the Western States the skunk is par- ularly liable, and a number of instances have been reported of the disease llowing the bite of this animal. In the dog the early symptoms are a ange in the disposition; it becomes quiet, dull and heavy, and very irrita- e toward strangers. The bark may have a peculiar ringing character. he animal does not take its ordinary food, but eats all sorts of articles, such straw and wood, and dirt. In some instances, the so-called furious rabies, ere is very much excitement, but in a majority of cases the symptoms are ther those of the dumb, or so-called paralytic, rabies, in which, after a ansient stage of excitement, there is unsteadiness in the legs, with increas- g weakness and ultimately paralysis and coma. The poison is present in e salivary glands, in the nervous system, central and peripheral, and in the ancreas, adrenals, and mammæ. The disease may be produced in animals y the inoculation of portions of these structures. In rabbits, the animals ually employed, inoculation is made beneath the dura, and death follows n from seventeen to nineteen days. The virus prepared for the protective culation is obtained by passing through a series of rabbits, when it is

part of the body which may happen to have been bitten. Wou
the face and head are especially dangerous; next in order of degre
tality come bites on the hands, then injuries on the other parts of
This relative order is, no doubt, greatly dependent upon the fact
face, head, and hands are usually naked, while the other parts are
c. The extent and severity of the wound. Puncture wounds are
dangerous; the lacerations are fatal in proportion to the extent of
face afforded for absorption of the virus. *d.* The animal conve
infection. In order of decreasing severity come: first, the wolf; se
cat; third, the dog; fourth, other animals." The average period is
from six weeks to two months. It may be prolonged to more th
months, and there are cases in which the incubation has apparently l
a year or eighteen months. Of persons bitten by rabid dogs only
percentage become infected; according to Horsley not more than
cent. The death-rate of those bitten by wolves is not less than 40
Bites upon the face are particularly dangerous.

Symptoms. In the premonitory stage there is usually some irritati
the bites, such as pain, numbness, or paræsthesia. There is loss of
headache, and depression of spirits. The patient may become very
and sleepless, and there is a constant sense of impending danger.
fever has been noted at this stage, and the pulse is accelerated. The
sensibility is greatly increased; a bright light or a loud noise is very
ing. Stiffness about the throat muscles, and slight difficulty in sw
may be expressed, and the voice is a little husky. In the second sta
period of excitement—there is great restlessness and hyperæsthesia.
afferent stimulant—*i. e.*, a sound or a draught of air, or the mere as
of a verbal suggestion—will cause a violent reflex spasm. In m
symptom constitutes the most distressing feature of the malady. The
which affect particularly the muscles of the larynx and mouth, are
ingly painful and are accompanied by an intense sense of dyspnœ
when the glottis is widely opened or tracheotomy has been performed."
sley). Any attempt to take water is associated with painful spasms
muscles of the pharynx and larynx, and of the elevators of the hyoi
This it is which makes the patient dread the very sight of water, a
given the name hydrophobia to the disease. These inspiratory and d
tion convulsions, as they may be called, are sometimes associated wit
iacal symptoms. In the intervals between the attacks the patient
quite quiet and the mind unclouded. Hallucinations and delusions
present, not infrequently associated with the idea of the presence of
in the room. He very rarely makes any attempt to injure the atte
and indeed may be particularly anxious to avoid hurting anyone.
may be, however, occasional fits of furious mania, and in the contrac
the muscles of the larynx and pharynx odd sounds are occasionally u
The saliva is usually abundant and tenacious, and flows from the
owing to the difficulty the patient has in swallowing it. The tempera
this stage is usually elevated, and may reach from 100° to 103°. The
may be afebrile throughout.

In addition to the local spasms of the respiration and deglutition m
there may be convulsive seizures of a tetanoid character. After lasti
a day and a half to three days this is succeeded by the paralytic st
which the spasms no longer occur. The patient becomes quiet, and
sciousness gradually supervenes. The heart's action gets more and
feeble, and death occurs by syncope. In animals the preliminary and
stages are absent as a rule, and the paralytic may be marked from th
the so-called dumb rabies. In man the paralytic form is extremely u

ne, though cases of it have been reported, and it may developed with a sudden paraplegia, and with symptoms resembling acute ascending myelitis.

Morbid Anatomy. The chief lesions are in the nervous system. Gowers, who has examined 9 cases, found morbid changes in 7. "These were indications of vascular disturbance; dilatation of small vessels, accumulations of leucocyte-like corpuscles around them and in the tissues, clots in small vessels evidently formed during life, and minute hemorrhages. These changes are met with in various parts, especially in the cortex of the hemispheres, the medulla oblongata, and the spinal cord. They are always most intense in the medulla, between the eminentia teres above and the decussation of the pyramids below, and especially in the neighborhood of the pneumogastric, hypoglossal, and spinal accessory nuclei. The accumulations of leucocytes around the vessels is a very conspicuous change. They surround the outer wall and may be so numerous as to fill up the whole space within the lympatic sheath; they may extend along the vessel for a considerable distance, and even pass into the adjacent tissue. The nuclei contain a much larger number of corpuscles than normal, and in places they may be aggregated and form dense masses, which, since the corpuscles may be regarded as identical with pus-cells, are, in fact, 'miliary abscesses.' . . . . In the spinal cord the alterations are usually much slighter, and are confined to encrustation of the vessels with leucocytes and to an increase in the number within the gray matter; in this the changes are usually greater than in the white columns, although they involve these in severe forms. They may, indeed, be so intense as to constitute a condition practically of acute myelitis. Even when the case has run so rapid a course as to be fatal in three days such myelitic changes may be conspicuous. The 'miliary abscesses' are very seldom seen in the cord. On the other hand, the central nervous system may present no other alterations than are common after death from asphxia, and this even in an animal inoculated with rabies, as well as in man. Outside the nervous system leucocytal infiltration has been seen in the salivary glands and in the kidneys." The mucous membrane of the larynx and pharynx is congested. In the dog the stomach not infrequently contains straw, hay, and foreign matter which the animal has eaten. There are no special changes in the abdominal or thoracic viscera.

Diagnosis. There is rarely any difficulty in distinguishing hydrophobia from other organic affections of the nervous system associated with spasms and cramp. In tetanus, spasms of the deglutition and inspiratory muscles occur occasionally, but the character of the wound, the short time which has elapsed before the symptoms develop, the presence of trismus and opisthotonos, and the absence of any aversion to liquid, render the diagnosis clear.

The greatest liability to error is in the so-called pseudo-hydrophobia, or lyssophobia, which is a neurotic or hysterical manifestation, and may closely simulate the true disease. A nervous person bitten by a dog, either rabid or supposed to be so, may display within a few months, or even several years after, symptoms resembling hydrophobia. He becomes irritable, depressed, and moody, constantly speaks of his alarming condition, and insists that he is certain to go mad. There may be hysterical paroxysms, in which he says that he is unable to drink, and shudders at the sight of fluid, grasps convulsively at his throat, and becomes emotional. A few years ago, when the newspapers were full of the details of Pasteur's treatment, a young man consulted me, who had been bitten a year or more previously by a dog which was still alive. Some of his fellow clerks had joked him upon the subject, and he had gradually become very much alarmed. When I saw him he was greatly excited, had pains in the throat, difficulty in swallowing, and in attempting to take a glass of water he would become greatly excited and alarmed, and would have a pseudo-convulsive attack. These sympt sisted for a couple of weeks, and ultimately yielded to treatment wi electricity. There have been instances published as lyssophobia i these seizures have become more frequent, and the patient has died ex Gowers holds that the majority of these cases are in reality genuine, remarks on this point are worth quoting: "There has more often bee dency to regard the genuine disease as imaginary than to mistake the for the genuine. This tendency is especially marked among critics w not seen the case, who do not scruple to cast doubts on the nature of ev cases. It is not certain that death has ever occurred from mere lyssop Nowadays the test of the nature of a fatal case can be readily made inoculation experiments are quite conclusive.

Dulles, in particular, has called attention to a number of diseases i symptoms of hydrophobia may occur, and he has rightly urged thorough and systematic examination of patients, and greater cau pronouncing upon the irregular symptoms, which, in nervous people, to follow the bite of a dog.

Treatment. The local treatment of the wound is important. If on a ligature should be placed above, and it should then be thoroughly ized. A bunch of lighted matches will serve the purpose, if nothing b at hand. Strong carbolic acid or nitrate of silver may be used. The should be encouraged to bleed, and it should be kept open for som Excision of the wound is also recommended. When the disease has dev the patient should be kept absolutely quiet in a darkened room, in ch a couple of nurses. There is rarely any necessity for restraint, and th sician can assure the attendants that there is no risk in their duties. is not a single instance on record in which the disease has been trans from patient to nurse. As a rule, the patient is readily controlled, an not require to be forcibly held or restrained. No attempts should be to force the patient to drink or to eat. Sometimes he can swallow r It is stated that the local application of cocaine allays the sensitiveness throat and enables the patient to swallow. If necessary, nutrient en may be given, or, if the patient cannot take water by the mouth, large tions may be given per rectum.

There is no medicinal treatment of any value. In the violent spas inhalation of chloroform may be tried. Morphine, chloral, bromide of sium, and curare have been recommended. The latter may be tried in of from a tenth to half a grain, repeated every half-hour until there is cular weakness.

PREVENTIVE INOCULATION. Pasteur found that the virus in the cords of inoculated rabbits, when preserved with careful antiseptic p tions, gradually diminished in intensity, so that the fourteen-day-old cor no longer poisonous. Dogs inoculated with portions of cords dried i way and of increasing intensity acquire immunity, and are in reality nated against the strongest virus, which would otherwise have proved Relying on these observations and on the fact of the long incubation p Pasteur began the inoculation in human beings bitten by rabid animals what is known as the simple method the individual receives an injecti the first day of a portion of the spinal cord of a rabbit which has bee served in the dry air for fourteen days; on the second day a bit of the c thirteen days old, and so on until the cord of the fifth day is used. In is called the more intensive method, on the morning of the first day a p of the cord (rubbed up in sterilized bouillon) of the fourteenth and thirt day is used, and in the evening the cord of the twelve and eleventh On the second day in the morning the cord of the tenth and ninth day is and in the evening of the eighth and seventh. On the third day the morning and evening injections are of the cord of the sixth day, and then one injection is made each day until the cord of the third day is used. Then a new series is begun, usually with the cord of the fifth day, and a third, or even a fourth, series of injections may be employed.

An enormous number (from 1886 to January 1, 1894, 14,430 persons) bitten by animals rabid, or supposed to be so, have been treated at the Pasteur Institute. Of these only 72 have died. A great difficulty has been that many persons bitten by animals not rabid have flocked to Paris for the treatment. In the preparation of the statistics these are carefully separated.

While the preventive inoculation is not invariably successful, as, indeed, is only natural, since many persons apply weeks or months after they have been bitten, it cannot be denied that the percentage of mortality in persons bitten by animals undoubtedly rabid is, after the treatment by the inoculation, very greatly lessened, and in some years has been nil; thus, in 1891, of 394 persons treated in whom the nature of the disease in the animal was determined with all possible certainty, not a single one succumbed. The possibility of communicating rabies has, of course, been urged, but it must be extremely slight, though there is one case in which this seems really to have occurred.

TETANY.

Definition. A paroxysmal affection, characterized by bilateral tonic spasms affecting chiefly the extremities.

Etiology. The disease occurs chiefly in young persons, and attacks males rather more frequently than females. Of 150 cases collected by Gowers from different sources, 76 were in males and 66 in females. The following are the most important conditions under which the disease develops:

a. In children associated with the debility of chronic exhausting maladies, such as diarrhoea. The carpopedal spasm, so frequently seen in connection with rickets, is regarded by some as a variety of tetany. It occurs as a sequence of the fevers; many cases have developed after typhoid fever, particularly in certain epidemics. A few typical instances have occurred in connection with pregnancy and lactation. Trousseau called the disease the rheumatic contraction of nurses. It may recur in successive pregnancies.

b. A remarkable association exists between tetany and the removal of the thyroid gland. Thirteen cases followed 78 thyroidectomies in Billroth's clinic, six of which proved fatal. It follows total, not partial extirpation. This is the most serious form of the disease. Removal of the thyroid in animals is also followed by tetany. Tetany and myxœdema may be associated, as in a case reported by James Stewart.[1]

c. The disease occurs in epidemic form, particularly on the continent of Europe during the winter months. Extensive epidemics occurred in Paris in 1855 and 1876, and a recent epidemic described by von Jacksch occurred in young men of the working classes, usually with slight fever. Fatal cases are rare in this form.

And, lastly, tetany has been met with in connection with dilatation of the stomach, particularly in the cases in which lavage has been practised. Death is very apt to occur in coma. A full record of the cases to 1892 will be found in the Paris Thesis by Vautier. In America true tetany is very rare, and it has not occurred in epidemic form. If, however, cases of carpopedal spasm be included the disease is not infrequent, and Griffith has been able to collect from the literature 72 cases, but the affection in adu all the cardinal symptoms, is rarely seen.

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[1] Transactions of the Association of American Physicians, vol. iv.

Symptoms. The following brief statement of a case which was wards in 1894 will illustrate some of the remarkable characters of t of tetany which recurs with pregnancy:

Mrs. P., aged thirty-three years; married at eighteen; first chi nineteen months afterward, and a second child two years after t While three months pregnant with the second child she noticed that he ached and felt tired, and two or three times a day would get quite stiff symptoms continued until a short time before her confinement, when much better. She remained free from the cramps until the ninth di labor, when they returned and were more violent. Not only were th closed in cramp, but the feet would also draw together. The attacks w accompanied by much pain. In the intervals the hands and feet usual. The attacks recurred for five months and then disappeared months. In November, 1882, after exposure in the snow, the spa appeared with greater intensity, and early in December she had an un severe attack in which the hands were closed, the elbows flexed, and th held close to the body. The spasms in the hands did not relax for a

In her third pregnancy, which followed in a short time, during tl five months she had no cramps, but in the last four months she ha daily. They again disappeared just before labor. She did not nur baby, and the fourth pregnancy followed in four months. She h cramps for the first four or five months, but they recurred as before the last four months. While in labor she had a very severe attack four or five hours. She afterward remained free until the ninth day she had a very severe attack.

In her fifth pregnancy, which followed in five months, the spasms oc at intervals throughout the entire period. In her sixth pregnancy, began eighteen months after the birth of the fifth child, she was free the first five months, then the cramps returned worse than ever. The and legs would get stiff and painful, and she had for the first time sp the larynx. During the last month of this pregnancy there were no at They recurred again on the ninth day after labor. From this time her seventh pregnancy, nearly three years, she was well, except at abo time of the menstrual periods, when she always had the spasms.

In her seventh pregnancy the attacks occurred as usual, but she was for a longer period before labor.

Since June, 1892, the date of her last confinement, she has had the at at intervals, usually about the time of the menstrual period.

The patient is a young-looking woman, well nourished, a little pale. mechanical excitability of the motor nerves was very great, the sli tapping in the course of the facial nerve was sufficient to produce contr of the muscles of the face on that side, and the electrical reactions, mentioned hereafter, were present in a typical manner.

The onset of the intermittent spasms may be sudden and unexpecte as a rule there are slight feelings of numbness or pain in the extremiti a feeling of lassitude and headache. The hands are usually affected and the spasms may be confined to them. The contraction begins in t terossei and the smaller muscles of the hands, which feel stiff and cra and gradually assume what has been known as the writing posture. fingers are closely pressed together, the thumbs adducted and pressed against the index fingers, or, in children, not infrequently flexed tight neath the fingers. The hand itself is generally flexed, and the elbow held in flexion. In children the arms are not infrequently folded ov cles. In the lower extremities the flexors of the feet and toes are in tonic spasm; the toes strongly flexed and the feet in the talipes equino-varus position. The thigh muscles are rarely involved. The muscles of the face and neck are less commonly attacked. In severe cases there may be trismus, and the angles of the mouth are drawn out. The trunk muscles are very rarely involved, but there is occasionally a slight degree of opisthotonos, and during a severe spasm the thorax may be fixed, and there may be slight difficulty in breathing. The skin of the hands and feet is sometimes tense and œdematous. The spasms are paroxysmal, continuing from a few minutes to an hour or more, occasionally lasting for several days. The contracture is, as a rule, painless, but when the spasms are intense the pain may be severe and cramp-like. The mind is clear, except in the extreme debility of children and in cases associated with dilated stomach, in which coma is a not infrequent accompaniment. The temperature is sometimes elevated during the attack, but it may be sub-normal. The pulse is usually accelerated. In the intervals between the paroxysms there may be a sensation of stiffness in the muscles. There are several important symptoms on the part of the muscles and nerves.

Trousseau found that pressure on the nerve trunks or on the vessels brought on an attack of spasm in the muscles of the limb. "So long as the attack is not over, the paroxysms may be reproduced at will, even though the patient has been free from them for twenty-four, thirty-six, forty-eight, seventy-two hours or more. This is affected by simply compressing the affected parts, either in the direction of their principal nerve-trunks or over their blood-vessels, so as to impede the venous or arterial circulation." (Trousseau.) In characteristic cases tight pressure round the wrist may be sufficient to produce cramp of the muscles of the hand.

There is a remarkable increase in the mechanical excitability of the motor nerves. A very slight tapping in the course of a nerve is sufficient to produce active contraction; if with the percussion hammer the slightest tap be made in the course of the facial nerve on the cheek, the muscles to which it is distributed will be instantly thrown into active contraction. This is known as the facial phenomenon, or Chvostek's symptom. It is not characteristic of tetany, and may occasionally be induced in tuberculosis. A very important sign is the great increase in the electrical excitability of the motor nerves (Erb's symptom). The current from a single cell may be sufficient to cause contraction of the face muscles. "Instead of the normal reaction 1, KClC; 2, AClC, KOC; 3, AOC, we have 1, AClC; 2, AOC; 3, KClC, or 1, AOC; 2, AClC, KClC, AOTe." (Gowers.)

And, lastly, the mechanical and electrical excitability of the sensory nerves is also greatly increased (Hoffmann's symptom), and the slightest pressure on the supra-orbital, the auricularis magnus, or the ulnar nerves (which in a healthy nerve would only produce a slight local sensation) is sufficient to cause paræsthesia in the parts to which the filaments are distributed. Other less constant symptoms are profuse sweating, œdematous swelling of the skin, herpes, urticaria, nutritive changes in the nails and hair, pigmentation of the skin, and in rare instances local or general atrophy of the muscles. The reflexes are usually normal, sometimes exaggerated. Psychical disturbances are rare, but epileptic attacks have been described. Though the spasms as a rule are intermittent, they are sometimes remittent, or they may be continuous, often in the same case. It is stated, too, that the spasms may persist sometimes during sleep. There are cases in which paræsthesiæ and stiffness may exist without the spasms. Gowers speaks of a variety met with in adult women in feeble health, who have a feeling of stiffness and tingling in the hands on awakening, which may last for a few minutes or longer. He says that there are instances in which this "sleep tetany" recurs through the day.

The course of the disease is very variable. The patient may hav two slight attacks and no recurrence, or it may last for many mon then again, as in the case above mentioned, it may recur throughout of years. There are instances in which it has recurred year by yea the winter months. In a few instances the disease proves fatal. T mentions such a case in which with very violent contractions, partic the muscles of the face and neck, asphyxia developed and cause More serious are the cases which follow extirpation of the thyroid a which develop in connection with dilatation of the stomach. Child infrequently die of the disease causing the exhaustion, rarely of the itself.

The anatomical condition is unknown, nor is its pathology as ye The occurrence in epidemic form has been held to show the infective ter of the disease. The occurrence after thyroid extirpation and in tion of the stomach suggest its dependence upon some toxic material nature of which, however, we have no clew. Bouveret and Devic i of tetany in dilated stomach have extracted from the stomach con material which they state produces a tetany-like group of symptoms mals. Recently Oddo and Sarles have reported a case of tetany in aged eighteen months, associated with retention of urine and anasarca. was, however, no albumin in the urine, but indican and an excessive q of earthy phosphates.

Diagnosis. Typical forms of the disease are very readily recognized. teria may simulate it very closely, but neither Trousseau's phenomen the increased excitability of the muscles and nerves is present. In r stances the disease might be mistaken for idiopathic tetanus when the are widespread, and in such a case the etiological factor would be mo portant. Some writers include with tetany all cases of carpopedal s in children. This has been done by Griffith in his recent paper. It i as he says, that there are numberless gradations between the conditi well-marked, widespread, intermittent contractions and the continuous termittent carpopedal spasms. While recognizing that there are ins in rickety children in which more extensive spasms occur than those ordinary carpopedal form and with the character of tetany (sometimes laryngismus), yet I think it is better to limit the name to those cases with the spasm show marked increase in the mechanical and electrical ability of the muscles and nerves.

Treatment. When the spasms are severe chloroform inhalations m used, as recommended by Trousseau. The entire range of antispasm may be tried, usually without any benefit. Such tonics as strychnine, ar and zinc are appropriate. Gowers speaks highly of the valerianate of with bromide of potassium, and for the nocturnal tetany a dose of dig at bedtime. Ice to the spine and electricity in its various forms may be u

Where possible the treatment should be directed to the underlying c tions. In the stomach cases, as the contractures very often follow di upon the use of the tube, this should be restricted as much as possible. the disease has followed extirpation of the thyroid, the extract of the may be given, or a portion of the thyroid may be transplanted.

DIPHTHERITIC PARALYSIS.

Nature of the Poison. Roux and Yersin showed by the inoculati animals with the cultures and with the toxins of the diphtheria b that a peripheral paralysis could be produced similar to that which occ tem. The observations of Sidney Martin on the character and actions of the chemical products of the diphtheria bacillus are most important. He was able to extract from the spleen and blood of persons dead of diphtheria an albumose and an organic acid. The albumose produced in animals, when injected subcutaneously, pyrexia, loss in weight, and paralysis, which anatomically was shown to be due to degeneration and atrophy of the axis-cylinder of the nerves. The loss of weight was a very striking feature in the animal. Very similar results followed the injection of the organic acid, but larger doses were necessary. Martin's conclusions are as follows:

1. "That the bacillus forms in the diphtheria membrane and in culture fluids products, viz.: albumoses and an organic acid identical with those found in the tissues of persons dead from diphtheria.

2. "That the physiological actions of single or repeated doses of these products, viz.: fever, emaciation, and progressive muscular paresis due to degeneration of the peripheral nerves, are the same as those of the corresponding substances obtained from the tissues, and as the phenomena of the disease itself.

3. "That the bacillus is therefore the primary infective agent in diphtheria.

4. "That it liberates in the membrane a ferment which when absorbed digests the proteids of the body, forming albumoses and an organic acid.

5. "That these are the immediate agents in the production of fever, paralysis, emaciation, and death.

6. "That the relatively enormous quantity of these products found in the spleen (in diphtheria as in anthrax) is explicable by the larger proportion of proteids normally present—stagnating, so to say—in the blood of the spleen than in the general circulation."

Anatomical Changes. The central nervous system is not, as a rule, involved. There may be a slight infiltration of the meninges, hemorrhagic foci, and the smaller vessels have been found blocked with micrococci. The diphtheria bacilli are not found in the nervous system. Many observers have described changes in the motor nerve cells of the anterior horns of the spinal cord, but they are not constant.

The nerves show important changes, which were first described by Charcot and Vulpian in those of the palate, by Ruhl in the spinal nerve roots, and in the peripheral nerves by Déjerine. The toxines produce either parenchymatous or interstitial neuritis, sometimes both. The alterations found have usually been in some proportion to the degree of the paralysis. The histological changes present nothing peculiar, being those described under the section of peripheral neuritis.

Hochhaus has called attention to the fact that the muscles are also involved. Granular and fatty degeneration is, of course, common in the muscles of the palate, but in the paralyzed muscles in other parts of the body there may be a very intense interstitial and parenchymatous myositis.

Symptoms. Paralysis follows diphtheria in a very variable number of cases, ranging from ten to twenty per cent. While it may develop as early as the seventh or eighth day, it is more strictly a sequel, not manifest until convalescence from the disease is well established. It may follow diphtheria of any part, and may occur after very slight forms of the disease. Children are very much less apt to suffer than adults.

The onset is usually slow, and not manifested by any recurrence or aggravation of the local throat symptoms, nor is there, as a rule, any fever. An interesting point, to which attention has been specially called by Bernhardt, Buzzard, and R. L. MacDonnell, is the loss of the knee-jerk during convalescence from diphtheria. It may be an early feature, while the local disease is still present, but more commonly it is found during convalescence.

It is important to bear in mind that it occurs in about two-thirds of
and that it is not necessarily associated with or followed by paraly

Local and general forms of diphtheritic paralysis are recognized.

LOCAL PARALYSIS. (*a*) *Palate, pharynx, and larynx.* By far t
common form is the gradual loss of power in the muscles of the pal
cated by a nasal tone of the voice and difficulty in swallowing. Th
in the voice is due to the fact that in the pronunciation of certain w
cavity of the nose is not shut off. In consequence also of this inab
patient cannot distend the cheeks or blow out a candle unless th
held. The difficulty in swallowing, manifest by regurgitation of
through the nose, is variable, being much more marked in some i
than in others. The palate is seen to be relaxed, hangs more vertica
cannot be raised. When touched the sensation is also much in
Atrophy of the muscles follows the paralysis, and the reaction of de
tion has been obtained, though with difficulty. This, the slightest a
transient form of diphtheritic paralysis, may disappear spontaneously
two or three weeks. Occasionally unilateral facial paralysis occurs w

When the muscles of the pharynx are involved, which is fortuna
so common, the act of swallowing is accomplished with difficulty, o
treme cases may be impossible, so that the patient has to be fed with

Involvement of the laryngeal nerves: "Paralysis of the upp
(superior laryngeal nerve) is more frequent than that of the voca
(inferior laryngeal nerve). In the former case the epiglottis stand
against the base of the tongue, and does not descend over the opening
the act of deglutition, in consequence of the weakness of the dep
The upper part of the larynx is insensitive, although when a foreig
reaches the vocal cords pain is felt. Hence, food is apt to get into the
and to cause coughing. The voice is hoarse, probably in consequ
paralysis of the crico-thyroid muscle, but the vocal cords move as usu
other cases there is paralysis in the region of the inferior laryngeal
and phonation may be impossible. The laryngoscope then shows imm
of the cords and sometimes a preponderant weakness of abduction, s
the cords are not separated during inspiration. In one fatal case, at t
of the first week, swallowing was impossible, and there was complete mot
sensory paralysis of the larynx." (Gowers.)

(*b.*) *Special senses.* Paralysis of the eye muscles, intrinsic and ext
is not uncommon. Loss of the power of accommodation, due to affect
the ciliary muscle, and loss of the light reflex may be present. Ptosi
external and internal strabismus are occasionally seen, and in rare ins
complete ophthalmoplegia. There may be contraction of the fields of

It is much rarer to have the other special senses involved, but cases
record of loss of the sense of taste, of smell, and of hearing.

(*c*) *Cardiac nerves.* Various forms of arrhythmia are not uncommon.
heart's action may be slowed to twenty or thirty beats per minute. In
cases there may be tachycardia, or the two conditions may alternate
same patient. In other cases the pulse is irregular in volume and in rh
Fatal syncope may occur, either at the height of the disease or durin
valescence. Occurring during the fever the child may, after an exagge
of the symptoms, present unusual pallor; the pulse may either be wea
rapid or may be not more than forty or fifty; the extremities are col
temperature sinks, and death takes place within a few hours with
features of collapse. More often the fatal event occurs during convales
even as late as the sixth or seventh week after apparent recovery.
attack may occur abruptly while the child is in bed, or may follow a s
exertion; more commonly there have been symptoms pointing to dist

cardiac rhythm, or there have been fainting spells. In some cases vomiting has preceded the attack. There are not often physical signs other than slight increase in the area of dulness and the presence of a gallop rhythm. These serious symptoms are ascribed to a neuritis of the vagi or of the intrinsic heart nerves. Possibly in some of the cases the lesion, as pointed out by Mosler and by Leyden, is an infectious myocarditis.

GENERAL PARALYSIS. The multiple form of diphtheritic paralysis is by no means uncommon. It usually begins with an affection of the palate, or with the loss of accommodation in the eye, and an absence of tendon reflexes. It is, as a rule, bilateral, involving the legs first, and the patient complains that they are heavy and stiff, and that he tires easily. Gradually the weakness progresses, and the paraplegia may become complete, or it may involve chiefly the extensor groups of muscles. The paralysis may extend and involve the arms and face and render the patient completely helpless. The muscles usually waste, and there is a diminution or even complete loss of the faradic irritability. The sphincters may be involved, though they are often spared, even when the paralysis is extensive. Disturbance of sensation in the form of numbness, tingling, and anæsthesia may develop. Anæsthesia may be present as a very special feature; thus Hallager[1] reports an instance in which some paresis of all four extremities occurred with anæsthesia of the distal parts, in the arms not extending above the elbow, and in the legs to the middle of the thighs. The muscles of respiration are usually spared, but the intercostal muscles and the diaphragm muscles are occasionally involved, and the muscles of the neck and back may be so weak that the patient can neither turn over nor hold up his head.

The outlook, of course, in these very severe cases is serious, and yet is not in many cases so bad as some of the symptoms indicate. Of 13 cases of the severer forms of multiple diphtheritic paralysis collected by Cadet de Gassicourt only 6 died.

The duration of these severer forms is very variable, from several months to an entire year. I remember the case of a medical student in whom the peripheral paralysis did not disappear for more than fifteen months.

The prognosis in the local palsies is good, except in the instances in which the pharynx and larynx are seriously involved, and in the cases with affection of the heart. The most dangerous are those in which a rapid and widespread palsy supervenes shortly after the primary disease.

Diagnosis. The diagnosis is rarely doubtful when the history of an attack of diphtheria is clear. In many instances the nature of a throat or nose trouble has been made manifest by the onset of a paralysis having the characters of that which so often follows diphtheria. Rarely could the absence of knee-jerks and the slight inco-ordination lead to a diagnosis of locomotor ataxia, nor is the gait, when the extensors of the feet are chiefly involved, at all like that of true tabes. There are instances on record in which hysteria, complicating the diphtheritic paralysis, has caused anæsthesia, either total or hemiplegic.

Treatment. As in all forms of peripheral neuritis, there is naturally a strong tendency to recovery, and the main indications are to support the patient's strength and to keep up, as far as possible, the nutrition of the muscles by electricity and massage. The special treatment is that of the ordinary forms of peripheral neuritis. We know of no measures which can directly counteract the poison in the system. When the palate is paralyzed there is rarely great difficulty in giving abundant nourishment. It is to be remembered that in this state solid and semi-solid foods are better swallowed than liquids. When the pharynx is seriously involved the patient fed per rectum or with a soft stomach-tube, the greatest care being that particles of food do not get into the larynx.

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[1] Neurologisches Centralblatt, Bd. ix.

It is too soon to say how far the new antitoxin treatment (the which seem so favorable) will diminish the liability to these mo sequelæ of the disease.

Disorders other than neuritis may follow diphtheria; thus multip sis may develop in children, as noted particularly by Marie.

Hemiplegia following diphtheria is not usually due to neuritis, but encephalitis or to cerebral embolism from heart-disease. Of 160 ca fantile hemiplegia in Wollenberg's statistics, three followed diphther one of my series of 120 cases followed this disease. Seifert[1] has repo interesting cases, both in children about the age of ten, who had ordinary palsy of the throat following diphtheria. In one hemiplegi oped suddenly; in the other more gradually. He has collected only from the literature of complete hemiplegia after diphtheria, two o were fatal, both from hemorrhage. As in other instances, the con probably due to an acute encephalitis, setting in with convulsions an Caspar Sharples has reported the case[2] of a boy, aged thirteen ye during an attack of diphtheria was seized with right hemiplegia and

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[1] Neurologisches Centralblatt, No. 12.

[2] Medical News, August 4, 1894.

CHAPTER VII.

CHOREIFORM AFFECTIONS.

BY WHARTON SINKLER, M.D.

CHOREA.

UNDER this head are included a number of different spasmodic affections, embracing the ordinary form of chorea known generally as Sydenham's chorea, hereditary or Huntington's chorea, chorea of pregnancy, hysterical chorea, post-hemiplegic chorea, senile chorea, chorea major, and the so-called electric chorea.

SYDENHAM'S CHOREA.

SYNONYMS: Chorea minor, St. Vitus's dance, and St. Anthony's dance. This is the variety of chorea commonly met with, and is what is usually referred to when the term St. Vitus's dance is used.

Definition. Chorea minor is an acute functional disease, occurring principally in children, and is characterized by irregular and inco-ordinate muscular contractions and twitchings, which are ordinarily beyond the control of the patient's will, and cease during sleep. There is present in most cases more or less psychical impairment.

Sydenham's original description of chorea is so graphic and clear that it is worth repeating. He says (*Entire Works of Sydenham*, London, 1783, *Schedula Montoria*, etc., page 562): "This disorder is a kind of convulsion which chiefly attacks children from ten to fourteen years of age. It first shows itself by a certain lameness, or, rather, unsteadiness of one leg, which the patient draws after him like an idiot, and afterward affects the arm of the same side, which, being brought to the breast or to any other part, cannot be held in the same posture a moment, but it is distorted or snatched by a kind of convulsion into a different posture and place, notwithstanding all his efforts to the contrary. If a glass of liquor be put into his hand to drink, he uses a thousand odd gestures before he can get it to his mouth, for not being able to carry it in a straight line thereto, because his hand is drawn different ways by the convulsion. As soon as it has happily reached his lips he throws it suddenly into his mouth, and drinks it very hastily, as if he meant only to divert the spectators."

Etiology. In studying the causes and conditions which influence the development of chorea we must remember that in infancy all of the attempted movements of the child are irregular and inco-ordinate; and even when the infant is not attempting to perform a voluntary act there are more or less irregular movements taking place in the extremities. If a young infant attempts to grasp an object, its hand is unable to reach it without a number of efforts, which are spasmodic and inco-ordinate, and like the movements of a choreic child.

Heredity. It is not often that one can trace chorea through a family, but it is fairly common that more than one case occurs among brothers and sisters. I have seen five cases of chorea in a family of six children, and there was no principle of imitation involved in this instance, as all of the cases occurred at different times. In 797 of the cases studied at the Orthopedic Hospital and Infirmary for Nervous Diseases at Philadelphia, no heredity could be traced in 631; in 33, one of the parents had suffered from chorea; in 96, a brother or a sister; in 30, an uncle or an aunt; in seven there was a history of chorea in the grandmother.

The parents of choreic children are frequently sufferers from some form of nervous affection, such as migraine or neuralgia, but at times there is no neurotic family history to be traced.

Age. This plays an important part in the affection. The majority of cases occur between the ages of five and fifteen years. In the cases of chorea in the note-books of the Infirmary for Nervous Diseases, which I have examined with the assistance of Dr. J. H. Rhein, we found in 961 cases that 816 were between the ages of five and fifteen years, the greatest number occurring at the age of nine; that is, 113 cases. Gowers believes that most cases occur at the age of thirteen years. Age of incidence in 937 cases: First hemidecade, one to five years, 107. Second hemidecade, six to eleven years, 453. Third hemidecade, eleven to sixteen years, 309. Fourth hemidecade, sixteen to twenty-one years, 68. Total, 937. The disease may occur at any period of life. I have recorded two cases which were apparently congenital, and have seen one case at the age of eighty years. It is rare under five years. In the 961 cases above referred to, but 53 occurred under that age. In one case, the choreic movements were observed at one month; in another the child, a girl, was attacked at a year and a half; 8 cases occurred at two years, 17 at three years, and 27 at four years.

Moyer (*Medical Times and Register*, May 13, 1893) reports a case which began at ten months, but it was evidently not Sydenham's chorea. The patient was fifteen years of age when examined, and the movements were like those which are associated with infantile diplegia.

I have seen a case of chorea which had existed since birth, and it was attributed to a fright which the mother of the patient received during pregnancy. Similar cases have been reported by others.

Sex. Gowers gives the proportion of the sexes as three girls to one boy. This ratio was obtained from the examination of a combination of statistics amounting to 1365 cases, and is the same which I found in the 961 cases which were treated at the Philadelphia Infirmary. Of the 961 cases which I analyzed, 696 were females and 265 were males, or a ratio of almost exactly three to one. In adults, if one excludes the chorea of pregnancy, the influence of sex is less marked. Contrary to what one would expect, the approach of puberty in girls seems to make no increase in the proportion of cases in that sex. Of 309 cases in the series which I have examined, there were 219 girls to 90 boys, a ratio of 2.43 to 1, between the ages of eleven and fifteen, inclusive, the period at which the catamenia is likely to appear. Osler states that after puberty the proportion of females to males becomes greater, but this does not agree with my statistics.

Race. It is exceedingly rare in the negro race, and Dana states that in New York it is more common in children of the German, Hebrew, and Portuguese races. Ogle (*British and Foreign Med.-Chir. Rev.*, vol. xli.) says that chorea is common among Jews, and quotes Addison, Steibel, and others as making the same statement. It is difficult to estimate the influence of race, unless one knows the proportion of the different nationalities in a given place, but, from my own experience at the Infirmary for Nervous

Diseases, I am satisfied that in Philadelphia it is more frequent in children of American parentage than in foreigners. Mitchell made extensive inquiries in regard to the extent of chorea in negroes, and the testimony which he obtained was "that chorea is seldom met with among pure-blooded negroes." I have met with five cases of chorea in negroes, in two of these only were the patients full-blooded negroes. In the majority there was some peculiar combination of influences brought to bear to produce the attack. One of the cases was a mulatto woman aged nineteen years; her parents were dead—both of them light colored. The patient had an attack of rheumatism one year previously. She had been married three months, and was in the fourth month of pregnancy. Four weeks before she had got into an altercation, and was struck on the left hand with a stick, and was hurt considerably, so that the hand was swollen and sore at the time she was seen by me. One week after the injury choreic movements began in her left hand, and they soon extended to the shoulder and then to the leg. The movements involved the whole left side when seen, and were increased when she made a voluntary effort. In this patient there were three predisposing causes for chorea. First, rheumatism, second, pregnancy, and, third, the injury associated with considerable mental excitement at the time of its receipt. Another case was that of a mulatto girl, aged twelve. She was one of five children, all of whom were healthy, except two, who suffered from rickets. She was small for her age, and had never menstruated. In September, 1893, she had an attack of tonsillitis, which was followed by subacute rheumatism, in which all of the large joints were involved. On December 10th, two months after the attack of rheumatism, she presented herself with marked evidences of chorea. It began at first with difficulty in articulation, then the right side of the body became affected, and later the whole body. The patient had some soreness of the joints, and there was a loud and well-marked mitral systolic murmur. The patient slept badly, was restless, and frequently awoke crying. She was irritable, cross, and peevish. Under the use of the salicylate of sodium she made a rapid recovery. Other cases of chorea in negroes have been reported. Skinner (*Phila. Med. Times*, 1875) records a case of a girl of eighteen, of pure African descent. Roy (*New York Med. Record*, May, 1892) also reports a case in a negro. Sachs says he has seen several in New York.

Climate and Social Station. Chorea occurs more frequently in the city than in the country, although country children by no means possess an immunity from it; neither is it any respecter of persons as regards social station, although some writers think that it occurs more frequently among children of the poorer classes. It is difficult to make any accurate estimate in regard to this point, for the majority of cases which occur among the better classes are treated by the family physician and do not come under the notice of a specialist, while among the poor they are usually brought to dispensaries for treatment, and these fall under the notice of a neurologist.

Eskridge[1] found that the climate of Colorado at altitudes varying from 4400 feet to 10,200 did not predispose to chorea; but he found that at the higher altitudes the disease was more difficult to cure, and it was necessary to send cases to lower levels before they got well.

Season. Chorea occurs most frequently in the spring. Gerhard[2] in a study of 80 cases, most of which were observed at the Philadelphia Infirmary for Nervous Diseases, found that of 68 cases, 39 occurred in the spring, 10 in the summer, 7 in the autumn, and 12 in the winter. Mitchell[3] has also written on the relation between season and attacks of chorea. Morris J. Lewis has written an elaborate paper on the relation of chorea and rheumatism.[1] He has examined 1383 separate attacks of regard to the month of the onset of the disease. Of these 717 are note-books of the Philadelphia Infirmary for Nervous Diseases, and occurred in Boston, and were collected by James J. Putnam an Coombs Knapp. Lewis found that of these cases, there occurred in 106, February 101, March 172, April 159, May 160, June 150, July gust 106, September 76, October 74, November 54, December 99—to

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[1] The Climatologist, August, 1891.

[2] American Journal of the Medical Sciences, July, 1876, p. 99.

[3] Lectures on Diseases of the Nervous System, Philadelphia, 1881, p. 128.

I have studied all the cases of chorea in the note-books at the phia Infirmary for Nervous Diseases up to date, and find that in 812 in which the month of onset is noted, there occurred in January 6 ary 59, March 79, April 93, May 74, June 93, July 87, August 74, ber 52, October 35, November 33, December 67, as will be seen b companying diagram.

FIG. 56.

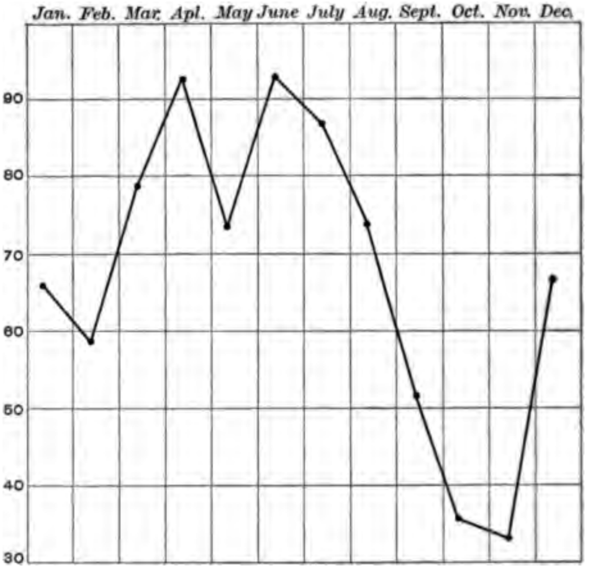

Diagram showing month of onset of 812 cases of chorea which were observed at the Phil Infirmary for Nervous Diseases.

Dr. M. Allen Starr[2] records 325 cases of chorea which he had obse New York, in which he had noted the month and day of onset of the Of these the largest number, 49, occurred in the month of April, a smallest number of cases occurred in October, November, and Deceml

Other observers have differed somewhat in regard to the season at the greatest number of cases occur, but I believe the discrepancy d upon the smaller number of cases which have been studied by them.

Koch,[3] in a study of 267 cases, found that it was most prevalent in Dec 22 per cent. of his cases having occurred in that month; and Dana[4] sa in New York there is almost an equal number of cases in the autumn and the spring.

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[1] Transactions of Association of American Physicians, 1892, vol. vii., p. 249.

[2] Ibid., p. 262.

[3] Archiv für Klinische Medicin, 1888.

[4] Text-book of Nervous Diseases, p. 437.

Lewis,[1] in a study of the relation of weather to chorea, thought that the weather influenced the production of the attacks, and believed that the cases were rather more frequent when the mean relative humidity and barometric pressure are low. More extended observations, however, have made him regard the influence of weather as less marked than he formerly thought.

Rheumatism. The relationship between rheumatism and chorea has been recognized for many years, but great differences of opinion have prevailed as to the point. The English and French writers, with but few exceptions, have upheld the view of the relationship, while the German authorities thought that the connection was very small. I have seen too many cases of chorea which immediately followed an attack of acute rheumatism not to believe that association between the two diseases is real and not accidental.

Osler[2] has analyzed 554 cases, in which 15.5 per cent. had a history of rheumatism in the family. In 88 cases, or 13.8 per cent., there was a history of articular swelling, acute or subacute.

Of 927 cases which I analyzed at the Philadelphia Infirmary for Nervous Diseases there was a history of rheumatism, acute or chronic, in 187 cases, or 20.1 per cent. In addition to this, there were 38 cases in which the children were said to have had "growing pains." In 79 cases, or 8.5 per cent., there was a distinct history of acute articular rheumatism. The intervals between the rheumatism and the attack of chorea varied from six years to immediately preceding. In 38 cases the attack of rheumatism had occurred within one year of the chorea; in 8 cases acute rheumatism immediately preceded the chorea, and in 7 the two affections were coincident.

The statistics of the Collective Investigation Committee of the British Medical Association give a percentage of 26 out of 239 cases in which there was a history of "joint affection."

In many cases of chorea there is a history of vague general pains, or flitting joint pains, which are probably not rheumatic. Osler suggests that they may be analogous to the joint troubles of scarlet fever, puerperal fever, or gonorrhoea.

Heart Disease. In a large proportion of children suffering from chorea there is some abnormal condition of the heart. In many cases there is distinct irregularity in the heart's action, and in others reduplication of the second sound is heard. Some writers believe that endocarditis is generally present in this disease, and is one of its causes, through the washing of small vegetations from the valves into the brain. Of course, it is difficult to determine in how many of the cases met with the cardiac affection has antedated the chorea. The British Medical Association Collective Committee found that in from one-quarter to one-half of the cases heart-disease precedes chorea.

The following table from Dr. M. Allen Starr[3] shows the connection between rheumatism and endocarditis in 2476 cases of chorea:

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[1] Medical News, Nov. 15, 1886.

[2] Practice of Medicine, p. [illegible]

[3] American Text-book of Diseases of Children.

TABLE I.—SHOWING THE RELATIONSHIP OF CHOREA, RHEUMATISM, AND ENDOCARDITIS (STARR).

Author.	No. cases chorea.	Rheumatism.	Cardiac.	Reference.
Groendal	52	37	Majority.	Wien. med. Woch., March 26, 1891.
Meyer	121	11	15	Berl. klin. Woch., July 14, 1890.
Koch	267	48	37	Arch. klin. Med., 1886.
Peiper	30	14	6	Deut. Med. Woch., July, 1888.
Sée	196	134	...	La Méd. Moderne. October, 1891.
Leroux	80	5	5	Rev. Mens. des Mal. de l'Enf., June, 1890.
Dale	20	3	8	Lancet, October 31, 1891.
Herringham	80	37	20	Lancet, January 12, 1889.
Garrod	80	36	45	Lancet, January 12, 1889.
Cheadle	84	62	...	Lancet, May 4, 1889.
Brit. Col. Invest. Com.	439	116	141	British Med. Journ., February 28, 1857.
Gowers	100	24	40	Dis. Nerv. System, vol. ii. p. 550.
Sachs	70	8	12	Keating's Cyclo. Child. Dis., vol. iv. p. 843.
Dana	130	7	8	Arch. of Pediatrics, April, 1888.
Sinkler	279	37	82	Pepper's System of Med., vol. iv. p. 442.
Starr	448	83	83	
	2476	662 26 per ct.	502+	

Nutrition. In the majority of cases of chorea which I have seen, the children are well nourished and do not look anæmic. The British Medical Association Investigating Committee inquired into the food of 437 cases, and found that in 384 it was sufficient, in 48 not quite sufficient, and in the remainder it was insufficient.

Anæmia is sometimes met with, and in some cases the number of red corpuscles in the cubic millimeter has been found far below normal. In the 439 cases of the Collective Investigation Committee, 92 were recorded as being anæmic.

Rachford[1] believes that in most cases of chorea the underlying cause is a "scrofulous anæmia."

Infectious Diseases. Of the cases which I have studied, remarkably few have had a history of antecedent infectious diseases.

Osler found a history of scarlatina in 25 per cent. of his cases; and of 853 cases which I examined there were 268, or 31.4 per cent., in which there was an antecedent history of scarlet fever. Of these cases, however, but eleven occurred within a short time of the onset of the choreic attack. It is highly improbable that there is any relationship between this disease and scarlet fever.

I find no record of any relationship between other infectious diseases and chorea, except Sturges's statement that he had found a history of previous whooping-cough more frequently in choreic than in other children.

Trowbridge (*Alienist and Neurologist*, January, 1892) believes that a relation exists between chorea and epilepsy. He gives a table of fifteen cases in which these diseases were associated at different times in the same individual.

Fright is a frequent cause of chorea; but it is probably not so common as is popularly supposed. On inquiring into the history of an attack, it is frequently attributed by friends of the patient to fright; but it is often found that there is no direct relationship between the fright and the attack. Sometimes the supposed fright has taken place several weeks before the onset of the disease. Gowers believes that fright is a cause of one-fifth of the cases of chorea, and in 859 cases which I have examined, in whom that point was recorded, there was a history of fright in 203, or 23.5 per cent. In order to consider fright as a cause of the attack of chorea, it is necessary that the interval between the alleged cause and the attack should be less than a month.

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[1] Medical News, April 22, 1893.

Traumatism is occasionally the origin of chorea; and a splinter in the flesh or the extraction of a tooth has been apparently the exciting cause in some instances. In these cases there has, however, been some accompanying mental disturbance or a predisposition to St. Vitus's dance, which has made the traumatism effective. Reflex irritation has been known to develop the disease. Jacobi reports a case caused by nasal irritation; and Leonard records an instance of chorea in a girl in whom there was adhesion of the preputium clitoridis, which was cured by freeing the adhesions. Other reflex causes, as adherent prepuce in the male, are occasionally met with, but they are rare, and I have never seen a case in which masturbation could be regarded as a cause of chorea.

Overstudy is, I believe, a frequent source of chorea, although there is a diversity of opinion in regard to school as a factor in this disease. Sturges speaks of "school-made chorea;" and refers to the influence of school as operating rather through bad ventillation of the school-room and injudicious discipline of teachers, rather than by overtaxing the brain by hard study. He speaks of nine out of twenty-five cases which he had observed in which the attack was directly traceable to school.

My own observation has led me to believe that a large number of choreas in children are the result of either overstudy or overanxiety, in connection with examinations or standing in the class. I have frequently found, on making inquiry, that the patient held a high place in her class and was anxious to keep her position; and a number of cases have developed during the time of the examinations in the public schools.

Morris J. Lewis, in his paper above cited, states that he doubts the influence of overstudy as the exciting cause of chorea; because, in his table of 1383 cases, collected in Boston and Philadelphia, the greatest number, 172 cases, occurred in March; and the school examinations do not occur until June. But in his table the number of cases in April, May, and June are almost as great as in March—159, 160, and 150, respectively—and in my table of 812 cases, all of which were seen at the Infirmary for Nervous Diseases, there were as many cases in April as in June, and more than in March. Among the cases seen at the Infirmary for Nervous Diseases, school as an exciting cause was mentioned in 42, and this does not by any means represent the children in whom the effect of ambition and overstimulation to do well was unobserved by the parents. Hamilton[1] found 20 per cent. of all school-children in New York either choreic or suffering from some similar disorder.

Pregnancy. The majority of cases in adults, exclusive of the hereditary form, are connected with pregnancy. This form is known as chorea gravidarum. It is most common in the first pregnancy, and seldom occurs for the first time after twenty-five years of age. It generally makes its appearance during the first five months, although it may occur during the latter months of gestation. Chorea in pregnancy very closely resembles the same disease in childhood, and frequently has predisposing causes in common, such as rheumatism and fright. The patients have frequently had chorea in childhood. A young woman who recently came under my observation had five attacks of chorea in childhood, married at twenty-eight, and became choreic in the third month of pregnancy. She was treated with vigorous doses of arsenic, and the choreic movements were entirely cured in ten weeks. She was safely delivered at term.

If chorea begins during the latter months of pregnancy, it is liable to continue to the end, and is more likely to be fatal. Gowers and Sachs cases of chorea which have begun after delivery, or after abortio former has made an analysis of 28 cases which he has collected, ar that the ages at which they occurred is as follows: Seventeen y eighteen years, 3; nineteen years, 3; twenty years, 8; twenty-one twenty-two years, 2; twenty-three years, 6; twenty-four years, 1.

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[1] American Psychological Journal, February, 1876.

Chorea of pregnancy has been recorded by some writers as a v disease; but this has not been my experience. I have seen at leas cases of chorea of pregnancy, and have not seen one terminate Barnes (*Transactions London Obstetrical Society*, vol. x., 1889) colle cases of chorea of pregnancy, of which 17, or 30 per cent., were fatal. probably represented the worse cases, and which were recorded beca were severe. An interesting case came under my observation in the wards at the Philadelphia Hospital, in which pregnancy apparently c attack of chronic chorea which has existed for years.

Mary G., aged thirty years, single, no neurotic family history. The has had scarlet fever, measles twice, and diphtheria. When she was n years of age she dreamed one night of seeing her dead mother stan her bed, and when she awoke she was in such agitation and fright fi vividness of the dream that she could not remain in her room alone. T morning, on awaking, she had a tremor, confined to the left side. Si later the whole body became involved in choreic movements; and the violent and extreme in character. After she had been choreic for fou she became pregnant; but she was unaware of her condition unti months advanced. Soon after this the movements increased in viol such an extent that she was unable to do any work, or even to sit still f than a few minutes. She was admitted to the hospital when she was fifth week of pregnancy. The movements were then so excessive t had to be kept in bed constantly. She was delivered at eight mont stillborn child weighing three pounds and twelve ounces. From the her delivery the choreic movements lessened, and in a few months th ceased entirely. She has been perfectly well for two years, and is n ployed in the hospital as a helper, and is active and efficient. Chore recur in several pregnancies, but this is unusual.

Ocular Defects. It has been asserted by Stevens and others that of refraction are the cause of many cases of chorea. De Schweinitz n study of fifty cases of children at the Philadelphia Infirmary for N Diseases, and came to the conclusion that while hypermetropia and metropic astigmatism are the preponderating conditions in the eyes of c children, being found in about 77 per cent. of the cases, evidence lacking that the refraction error is the basal cause of chorea.

Of the cases which have come under observation at the Infirma Nervous Diseases, a large part have had their refraction errors correcte there has been in no instance immediate improvement until medicina hygienic treatment was pursued.

Occasionally, optic neuritis is met with in a patient suffering from c Gowers speaks of this, and de Schweinitz has observed the same con Oliver[1] says that in almost all cases of chorea there exists a low form of neuritis and retinitis.

Among the cases at the Infirmary for Nervous Diseases, there was a h of intestinal worms in but a small percentage; in fact, there were but children who were said to have worms at the time of application for ment. On the other hand, Ogle[2] states that fourteen of eighty cases se him had lumbricoids at the beginning of treatment. The same writer relates the case of a girl of nine years who had an attack of chorea of extreme severity. There was a bed-sore on the sacrum, and the elbows were much chafed. Pieces of tapeworm had been passing per anum for three months. A dose of Filix Mas brought away seven yards of a tenia, including the head, and within twenty-four hours the child was quiet, except for an occasional twitch. In ten days she was entirely well, but a distinct mitral murmur remained.

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[1] Keating's Cyclopædia of the Diseases of Children, vol. iv., p. 197.

[2] Brit. and For. Med.-Chir. Review, vol. xli., p. 233.

Malaria has been suggested as a cause of chorea. Vought[1] reports a case in which choreic movements followed a fright. There was a history of malarial poisoning, and an examination of the blood showed the plasmodia characteristic of malaria. The patient was then treated with quinine and arsenic, and recovered. The cure seems hardly speedy enough to make one regard the treatment as the direct cause of improvement, three months having elapsed before the child was well. In another case the same writer found in the blood of a choreic boy "pigmented intercellular bodies." A malarial origin may explain the benefit which has resulted from the administration of large doses of quinine recommended by Wood.

Symptoms. The earliest change to be observed in a child threatened with chorea is a general restlessness and inability to sit still. There may be an occasional shrugging of the shoulders or a restless movement of the body, without any special or definite movements being noticed. Sturges[2] thinks that the best indication of the approach of an attack of chorea is to be found in the hands, and he suggests the following test: "The child is told to hold both hands open, with extended arms and the palms toward you. If that be done steadily, both hands upright and both alike, no finger or thumb quivering, no falling back or hanging forward of either hand, nothing to choose between the position of the two, then the child may be acquitted of chorea." Frequently, however, the choreic movements in a child are first observed by the teacher at school, who finds that the little pupil is unable to sit as quietly as formerly, and that there are some occasional irregular movements of the hand or body. The child's writing becomes bad, and the pencil is often dropped from his hand, at the same time there may be emotional disturbances. The child becomes irritable and peevish, and wakes in the night with attacks of crying. There may be more or less joint pains, which are considered by the parents "growing pains," and headache is more or less common. In a few days the irregular movements become more marked. The child grows awkward in the use of his hands, drops his knife at table, or spills a cup of milk, and on being reproved becomes more awkward in his movements. As the disease progresses the movements become more constant and more violent. They are irregular and jerky, and the want of rhythm is a striking characteristic. At first the movements are confined only to the arms, or, perhaps, to one only; but in a short time they extend to the legs, and then to the face. The extent and severity of the movements vary in different cases, and it is convenient to divide the cases into two groups, the mild and the severe.

In the mild cases the movements are only moderate in extent and are, to a certain degree, under the control of the patient; for example, a voluntary effort will, for the time, arrest the movements, and fixing the child's attention will also have the same effect temporarily. In these cases it is often possible to arrest the movements by making the child fix his eyes upon some object, like the tip of a finger, a few inches from his nose. The child is able to go about and to perform the various voluntary movements without trouble. He can dress and undress himself and feed himself, and frequently is able to continue his studies at school without interruption.

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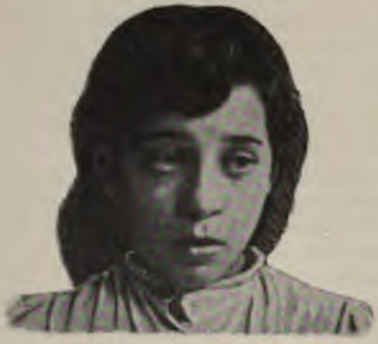
[1] Familiar Forms of Nervous Disease, Starr, p. 238.

[2] On Chorea and St. Vitus's Dance, p. 82.

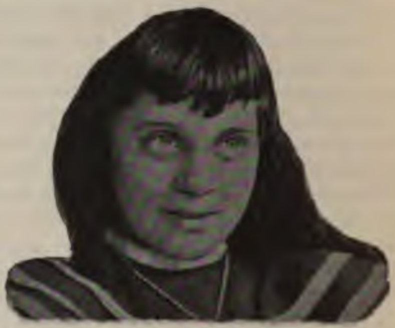
In the severe form, the movements become rapidly general, and are so violent and constant that the patient is in a continual state of muscular contortions. Every muscle seems to be alternately thrown into contraction, the body is twisted and turned, the limbs jerked about, and the features distorted, so that it is almost impossible for the child to remain seated upon a chair. In extreme cases the patient has to be kept in bed, and even then there is a continual state of muscular agitation, the whole body being thrown about so violently that unless the bed is enclosed in high-padded sides the patient will throw himself out of it. A patient who was under my care a couple of years ago had movements of such extreme violence that, although she was placed in a bed with padded sides, two feet above the bed, she sometimes managed to throw herself over the sides on the floor. In such cases the child is covered with bruises, the skin around the mouth is abraded from the continual protrusion of the tongue and running of the saliva, and the lips are cracked and ulcerated.

FIG. 57.



Sarah J. R. during attack of chorea.

FIG. 58.



Sarah J. R. after recovery from chorea.

The speech is frequently affected in both the mild and severe cases. The patient speaks in a thick and indistinct manner, as if the tongue were beyond control or were too large for the mouth, and sometimes the words are jerked out spasmodically, as if the patient was obliged to take advantage of momentary opportunities to utter a few words at a time. In mild cases this merely amounts to sometimes breaking off in the middle of a sentence or the lengthening or cutting short of a word. In some cases the patient is unable to articulate at all, owing to the inability of the child to control the tongue and other muscles concerned in articulation. Sometimes there occurs a condition known as laryngeal chorea, in which peculiar explosive sounds are made. At times these are like a bark, and in some cases a word is involuntarily uttered. Echolalia, or the repeating of the last syllable of a word, and coprolalia, the use of filthy or obscene language, are occasionally met with in chorea, but these cases are commonly hysterical. The muscles of the eyeball in rare instances are affected, and it is said that momentary diplopia sometimes results from spasms of these muscles. The features of choreic children undergo extreme distortion, with uncouth grimaces, and during the intervals between facial movements the face assumes a blank and almost vacant expression, which is characteristic of this disease. I have endeavored in the accompanying illustrations, taken from photographs, to give a reproduction of the choreic face.

The muscles of respiration, especially the diaphragm, are sometimes also affected, as well as the voluntary muscles. The respirations are irregular; sometimes short, sometimes deep, with occasionally a few rapid, shallow respirations, and then a long, sighing respiration. Occasionally the heart's action is irregular, but it is doubtful if this is a true choreic disturbance; it is probably dependent upon the irregular respirations.

The disease reaches its maximum severity in about two weeks, and by this time, if the case is a severe one, all the muscular movements are constant and severe. If the patient is told to protrude his tongue, it is thrust out after a moment of hesitation, and then withdrawn so rapidly that one has hardly time to see the organ. If the mouth has been open the jaws snap together with violence, and there has been a case recorded in which the jaw was broken through the violence with which the teeth were brought together.

The irregular movements may at first involve one side only, and afterward extend to the entire body, or the movements may be general from the onset. There is considerable difference of opinion as to the side most frequently affected. Some writers hold that the right side is most frequently attacked, and others maintain that the left side is most likely to be disturbed, on account of this side being the weaker. My own observation leads me to believe that both sides are about equally affected.

Sturges believes that the right hand is much oftener affected than the left, because he considers that in school-children this hand is habitually overtaxed. Gowers, out of 64 cases of hemichorea, found 31 on the right side and 33 on the left. Seè found that in 97 of 154 cases the movements were either confined to the left side or were most marked on that side. It is probable that if a sufficiently large number of cases were examined it would be found that both sides are equally affected. In the majority of cases the disease does not remain confined to one side, especially in cases which are severe. After a few days both sides become involved.

The voluntary movements in cases of chorea are distinctly inco-ordinate or ataxic in character, and are usually interrupted by the involuntary movements. There is distinct lack of power in the patient to use the will in making voluntary movements. This condition is sometimes mistaken for paralysis; but it is not from absence of muscular power that the movements cannot be made. A patient is told to perform some act, for example, to take up an object from the table. He hesitates for a moment, as if waiting for the involuntary movements to subside, and then darts down upon the object and seizes it. In extreme cases the patient is often totally unable to force the muscles into obedience by a mandate of the will. In a case I studied recently I told the child to put the palms of her hands together. She made no attempt to move the arms for a few seconds, and then they were thrown about in an inco-ordinate manner. There was another pause in which the patient was apparently steadying herself for another effort, and then the hands were brought almost together for a fraction of a second, and the arms dropped to her lap. The child then said with tears, "I can't do it."

During sleep the choreic movements cease entirely. Occasionally a case is reported by a parent in which the child is said to have movements in sleep, but it is doubtful if this is true. The sleep of the choreic child is frequently restless, but the choreic movements do not continue. However, Osler has recorded the case of a child in the Johns Hopkins Hospital in whom choreic movements were observed during sleep.

The electrical condition of the muscles has been studied, and there is no distinct reduction in irritability. Some writers have noted an ove response to the galvanic current; but this is admitted by these writei inconstant.

Sensibility is unaffected, except in hysterical cases, and in ordinar of St. Vitus' dance there is no change in tactile sensibility or in tl sense, nor is tenderness over the spine or nerve trunks met with.

The muscular spasms in chorea occasionally induce a sense of fatigu it is very seldom that patients complain of being tired. They seem to a great extent, unconscious of the extreme character of the movements children who are suffering from chorea of extreme severity generally den they feel tired. No muscular pain seems to be developed in these although the joint soreness and stiffness, already alluded to, are som complained of.

The mental state in the mild type of chorea is seldom changed, that the patient may be more irritable than usual and less able to s but in severe cases there is usually distinct mental deterioration. If the is let alone it is not often that he complains, and I have often been sur to see a child lying in bed, thrown about from side to side by the movei but apparently satisfied, and making no murmur or complaint.

The muscles of deglutition are sometimes disturbed, so that the pati unable to properly masticate his food, and swallowing is occasionally dif The principal trouble, however, is that the child is interrupted in his att to take food by the involuntary movements which occur.

The temperature in ordinary cases of chorea is generally normal occasionally it is elevated. Gowers states that he has seen it reach 102°. fatal cases, however, the temperature has been known to reach a high p

Muscular weakness frequently exists in cases of chorea, but usually not amount to more than a condition of paresis. The weakness is show a dragging of the leg in walking, or by enfeeblement of the hand in u it for various purposes. The loss of power, however, in all cases, is apparent than real, and, as already observed, it is frequently the case the inability of the child to use the limb is due to lack of will-control the muscular movements, rather than true paralysis. In some cases paresis is a more prominent symptom than the movements (the para chorea of Todd), and it is not unusual to have a child brought for t ment with the statement that there is paralysis of one side, and on invest tion it is found that there is a choreic condition.

It has been a question among authorities whether the muscles of org life are ever involved in chorea. The action of the heart is said by s writers never to be affected, but there is no doubt that in many cases heart's action is irregular and disturbed, although no more so than one m with in patients who are suffering from some other nervous disorder.

HEART SYMPTOMS. As just remarked, irregular and frequent ac of the heart is often met with, and sometimes the action is tumultu There is seldom, if ever, any true disorder of the rhythm. Heart m murs occur with great frequency. In the majority of cases these hæmic, and are dependent upon the anæmic and weakened condi which is usually found in these cases. A systolic murmur is freque heard at the base, soft in character, and varying in degree at different ti Of 813 cases at the Infirmary for Nervous Diseases, in which the condi of the heart was noted, there were 300 in which a murmur of some k was detected. The most frequent of these murmurs were systolic, and v heard at the apex. There were 128 cases of this class. In 65 cases t was a systolic murmur heard at the base, and in only three was a diast murmur noted. In but one case was an aortic regurgitant murmur no

Gowers states that he met with only two instances of aortic regurgitation among about 250 cases of chorea which he had observed. In the large majority of the cases studied at the Infirmary for Nervous Diseases the murmur was soft and was usually hæmic.

Endocarditis occurs with considerable frequency in chorea, but, as a rule, it is not of an extreme kind. It is to be expected that organic heart lesions will occur in a disease which is so closely allied to rheumatism as is chorea. Osler (*American Journal of the Medical Sciences*, 1887, vol. ii.) examined a number of cases at the Infirmary for Nervous Diseases, two years and longer, after an attack of chorea. He found of 110 patients that 54 presented some signs of organic heart disease. In many of these cases the murmur was only slight, and in but few were there any general disturbances resulting from the valvular disease.

Pericarditis occasionally occurs as a complication of chorea. I reported a case in the *University Medical Magazine*,[1] in which a boy of six years, during the course of an attack of chorea, had a violent attack of pericarditis, from which, however, he recovered; but a marked friction sound remained. This child had suffered from an attack of acute rheumatism, preceding the chorea, and had a mitral systolic murmur when first seen. We must remember that in some cases the organic disease of the heart antedates the chorea; but, in many cases, it is evident that the murmur has developed during the course of the disease. In all cases it is difficult to say whether the murmur is functional or organic, as the murmur is seldom harsh or loud, and, frequently, the only way to determine whether the disease is organic is when it persists after the patient has recovered.

The urine in chorea has been examined by a number of authorities. It contains usually an excess of urea and phosphates. The amount of these salts depends upon the severity of the attack. In a patient recently under my observation, in whom the movements were of a most violent character, the deposit of urates and phosphates was enormous. The specific gravity is high, 1024–1030, and the urine is of small volume each day. The amount of the chlorides is diminished in proportion to the urea.

Gowers mentions a peculiar pigment, "urohæmatoporphyrin," which was discovered by McMunn in the urine of rheumatism, as having also been found in chorea by Garrod. Herter[2] refers to the peculiar red color, eosin-like, but which he says is not due to urobilin.

Mental disturbances are frequently met with in choreic children, although in the majority of cases there is no grave trouble. The temper is almost invariably irritable, and there are frequent hysterical outbreaks, and the child is likely to be averse and headstrong. There is a marked change in the mental powers, the child is unable to apply its mind to study or to fix the attention for any length of time. Occasionally there is distinct evidence of insanity in a mild degree, and melancholia has been met with. In rare instances there is an outbreak of acute mania, and when this occurs it is associated with a very violent form of chorea, giving rise to what has been described as chorea insaniens. We should not overlook the fact of the mental deterioration in cases of chorea, and it is of great importance to impress upon the minds of the parents that the children are not responsible for many of their acts, and too much should not be expected of them.

Convulsive attacks are rare as a complication in chorea. Gowers speaks of a case in which the choreic movements were severe, but which had occasional attacks in which there was apparent loss of consciousness and peculiar convulsions, in which the head was turned to the right, the right arm and leg presented violent choreic movements, while the left arm was stretch the fingers everted, and the whole limb in a state of cataleptic r Epileptic seizures are also rare as a complication, although they are oc ally met with, and sometimes one sees a patient whose attacks of e date from an attack of chorea. Gray[1] mentions cases which he has which there was temporary loss of consciousness, like petit mal. Th says, may develop into a grave form, with symptoms of meningitis or e alitis, from which the patient dies.

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[1] Vol. ii., 1890, p. 482.

[2] Diagnosis of Diseases of the Nervous System, p. 556.

The tendon reflexes in chorea are frequently disturbed to some ext have made a study of this condition and find that, while in a majo cases the knee-jerks and other reflexes are normal, in quite a consid number the knee-jerks are either absent or they may be described as cious; that is, the knee-jerks may be absent at one moment and next an involuntary movement of the child causes a re-enforcement, a response to a tap upon the patella tendon is prompt and energetic. are some cases, however, in which the knee-jerks cannot be developed any circumstances. In 614 cases in the series at the Philadelphia Infi for Nervous Diseases the reflexes were normal in 239; in 180 the increased; in 56 they were diminished; and in 36 they were absent altog In 42 cases it is recorded that the knee-jerks were capricious; in 36 were irregular, but diminished; and in 8 they were increased, but irre In 14 cases the knee-jerks could be elicited on one side only.

This irregularity in the knee-jerks is an interesting phenomenon, contrary to what one would expect. At first sight it would seem pro that the knee-jerks would always be increased, on account of the co re-enforcement which should be present from the ever-moving muscles we found that in only 29 per cent. of the cases which we examined we knee-jerks increased, and in 145, or nearly 24 per cent., of the cases the jerks were either absent or diminished in some degree. D. B. Lees[2] re a case of chorea in a child in which the patellar reflexes were absent believes that this demonstrates that the motor cells in the cord are af in this disease.

Trophic changes are shown in chorea in the ease with which slight scra or injuries become irritated and inflamed. It is very common in chi who are suffering from an aggravated form of chorea to find them co with sores and suppurating scratches, which heel slowly. I saw recentl old woman, who had violent hemichorea, in whom the whole side was cov with bruises, and the hand and arm were of a deep purple hue, loo almost like beginning gangrene. The other arm and hand were discol also, but in a lesser degree. Callosities occur on the knuckles, elbows, knees from the friction to which they are exposed in extreme cases, and are greater than would occur in a healthy child from the same amou irritation. Lloyd[3] mentions the case of a young girl, who, in the t month of pregnancy, became choreic. In the fifth month an abscess of breast developed, and, although incised several times, sinuses formed, a did not heal for several weeks. It was thought to have resulted fro injury in some of the patient's violent movements. Ogle[4] reports one ca which there was a bed-sore on the sacrum; and another, a child of s years, in whom, during an attack of chorea, there were subcutaneous abs of the heels, shoulders, and thorax. The case terminated fatally. In a t patient, a girl of seventeen years, there was inflammation of the alv e eruptions which accompany rheumatism are met with in the course of attack of chorea. Osler says that he has seen erythema nodosum and puric urticara. He also says that an aggravated condition of rheumatic rpura, known as Schönlein's peliosis rheumatica, sometimes occurs in orea. English writers speak of subcutaneous fibrous nodules, such as are ociated with rheumatism, being seen in chorea. The Collective Investiga- n Committee of the British Medical Association reports 7 cases out of 439 which they were met with. These eruptions are further evidence of the nection between rheumatism and chorea.

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[1] Nervous Diseases, p. 406.

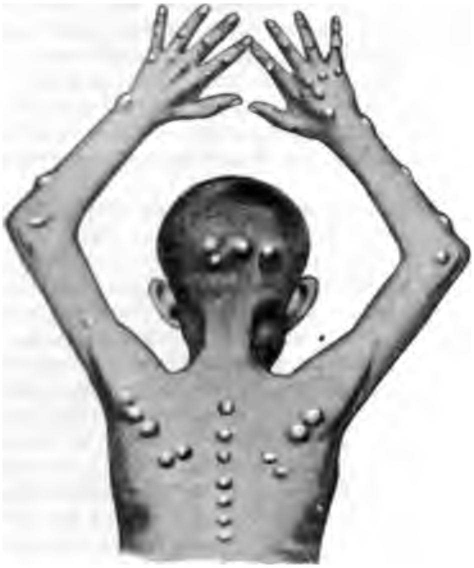
[2] Clinical Journal, London, March 8, 18

[3] Hirst's System of Obstetrics, vol. ii., p. 596.

[4] British and Foreign Med.-Chir. Rev., vol. xli., p. 233.

Dr. C. H. Brown (*Journal of Mental and Nervous Disease*, August, 1893) rte a remarkable instance of subcutaneous nodules in a case of chorea

FIG. 59.



trophied, its impulse was diffused, and its action markedly exaggerated. was a mitral regurgitant murmur heard. One hundred and fifty tun actual count, varying in size from the head of a pin to an almon discovered over the dorsal surface of the whole body, and were esp marked over the joints. They were subcutaneous, of a soft consisten under the microscope were found to consist of "young granulating con tissue."

Duration. The duration of an attack depends upon the severity case, and varies from three weeks to three months. The average dura agreed by writers on this subject to be ten weeks. The attack ma into months, and it is not unusual to see cases which have lasted six n and more. These cases, however, have almost invariably begun with toms of great severity. The violent movements rarely last more than three weeks, and under favorable circumstances do not persist even a as that. Cases are met with which have lasted for two or three years. however, are rare, and the statement of the parents in regard to the du of an attack is to be taken with allowance for unintentional misstate They frequently state that the child has suffered from chorea contin for several years, but have failed to take into consideration the rem which have lasted for eight or nine months of each year. Mild attack last only for two or three weeks. Occasionally cases become chroni continue during the entire life of the patient. Gowers quotes a case had begun in youth and continued until sixty-six years of age, and I myself seen two cases which began in infancy, and persisted in one case the thirtieth year and in the other until the thirty-fifth year. The should be borne in mind, however, that the more severe the attack the l will its duration be.

Recurrence. Relapses are comparatively frequent in chorea. O 961 cases of the Infirmary series, 341 had more than one attack. Thi slightly larger proportion than that given by Gowers, who states that third of the cases which he had analyzed had two or more attacks. O 341 cases who had more than one relapse, 219 suffered from two attack had three attacks, 29 had four attacks, 13 had five attacks, 2 had six att 2 had seven attacks, 1 had eight attacks, 2 had nine attacks, and 1 was to have had twelve or more attacks. Girls are rather more liable to r rence than boys. The danger of a relapse usually ceases after si years of age in either sex. It is unusual in boys to have a relapse after tw years of age, but in girls the relapses continue until they are sixteen or se teen. Evidences of organic heart trouble are more frequent in cases w have relapses than in first attacks, and it is quite common to find pat who return with a second attack of chorea to have cardiac murmurs. interval between the first and second attack is usually nine to twelve mon occasionally, but rarely, the relapse occurs after two or three months; bu a rule, the recurrence takes place at about the same period of the year as in which the first attack took place. This statement is not in accordance Gowers's observations, who says that there is no uniformity in the time of year in which recurrences take place, although he admits that there ar markable exceptions. In my own observation, however, a patient who had an attack one spring is liable to have a return the spring following, if this period of the year is escaped on the first year, it is likely that on second year there will be a recurrence at the same season.

Fright is an occasional cause of the recurrence, but in many cases no di cause is to be traced, unless it may be the influence of overstudy, or of depressing influences of the combined causes connected with school, w have predisposed to a first attack. Successive attacks of chorea are usu much like the first, but they do not invariably affect the same side as any previous attack. A patient seen recently had the first attack involving the right side, and the second attack in which the left side was only affected. Succeeding attacks are rarely as severe as the first, and yield more promptly to treatment; occasionally, however, the second or even the third attack may be of great severity.

Morbid Anatomy and Pathology. There is scarcely any disease in which the pathology is so unsettled as in St. Vitus' Dance, notwithstanding the fact that a great number of carefully made autopsies have been recorded. Almost every observer, however, has reported different conditions. Sée collected 84 cases in which post-mortem examinations had been made, but in 16 of these no changes in the brain or nervous centres were found. In 32 there were discovered lesions of some kind in the brain and nervous centres, and in the remainder some inflammatory changes were found in the serous membranes. In 29 of these cases there were evidences of cardiac disease. This writer believed that in but few cases of chorea was death the result of heart-disease, but considered that the majority of deaths should be referred to nervous excitement and anæmia. On the other hand, of 80 fatal cases collected by Sturges, the heart was healthy in only 5. The embolic theory which was advanced by Hughlings Jackson has been accepted by many writers as a plausible and attractive view. The fact of the frequent association of endocarditis with chorea, with the possibility that minute vegetations may be washed into the capillaries of the brain, gives strength to this theory. It does not prove it, however, because in a large number of cases of chorea in which death has occurred no endocarditis has been found, and Gowers and others have examined the brain of many fatal cases of chorea without finding emboli.

The experiments of Angel Money on animals also lent some force to the embolic theory. This writer injected lycopodium into the carotids of animals, producing thereby movements like chorea, and after death he found capillary plugging in the brain and cord; the symptoms, however, in the subjects of Money's experiment were different from those in true chorea, and, moreover, it is well known that many lesions in the brain give rise to choreoid movements. The symptoms of capillary embolism in the brain, such as vertigo and drowsiness, are absent in chorea, as has been pointed out by Sturges. Golgi found calcification of the cells of Purkinje in the cerebellum of one case.

Lastly, the theory of microbic infection, as a cause of chorea, has been advanced within the past few years. Donkin and Hobb (*Medical Times and Gazette*, Nov. 1, 1884) found rod-like bodies in the blood of a patient dying of chorea, and Richter (*Western Lancet*, vol. xii., p. 529) discovered cocci in the blood of another case. Berkeley, in a valuable paper on chorea insaniens (*Johns Hopkins Reports*, vol. i., 1891), strongly urges the infectious origin of chorea, and in the fatal case which he described, in which the most elaborate and thorough microscopic and bacteriological examinations were made, evidences of microbic infection were discovered. Pianise[1] accepts the theory of an infectious orign as the result of bacteriological examinations in a case of acute chorea which terminated fatally. This author obtained a bacillus from the cervical cord of the patient, which, inoculated in dogs, caused the appearance of the disease. Dana is also an advocate of this view, and supports it by the report of the following case (*American Journal of the Medical Sciences*, January, 1894): The patient was thirty-four years of age, and had repeated attacks of chorea following acute rheumatism at the age of fourteen years. The last attack was of eight months' duration, and was excessively violent u death of the patient, which resulted from exhaustion. At the autopsy leptomeningitis of the convexity of the brain was found, and menin the upper part of the spinal cord. There was also a slight degree of m encephalitis. Hyaline bodies were found in the brain cortex, and dip in small numbers were found in the proliferating tissues between the m and the brain. There was no endocarditis, and the heart was otherwise

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[1] Annual Medical Sciences, 1894, vol. ii., C. p. 29.

Dana's deductions from this case are that it shows that there is relationship between many of the chronic spasmodic disorders of in types and the chorea of Sydenham. Secondly, that it confirms the vi generally accepted that chorea is a vascular and humoral disease, and t that it gives weight to the belief that there is in some cases, at least, a n which produces the disease. He thinks that there is nothing which explain the phenomena of the disease so well as to suppose that the virus producing chorea is a microbe, and perhaps some special form diplococcus.

It seems to me that the explanation of all these differences in th mortem findings which have been recorded by numerous obser to be found in the fact that the autopsies have been made in different forms of chorea, and usually in cases of long standin that the observers have examined their cases from varying stand In nearly all of the fatal cases of chorea which have been examine mortem, totally different conditions have existed from what are present ordinary chorea of childhood. In many some grave complication like carditis or meningitis was the cause of death. In Berkeley's case the p died of chorea insaniens, which is altogether unlike Sydenham's cho many respects, and in this case not only were there many symptoms of o disease during life, the patient having had a distinct attack of men followed by choreic movements four years before the final attack, but autopsy endocarditis was found, and the microscope revealed leptomeni In Dana's case the patient had had repeated attacks of chorea, the last of had persisted for eight months, and he died at the age of thirty-four As was to have been expected, coarse brain lesions were found. C leptomeningitis and some meningitis of the upper part of the cord present. The age of this patient and the duration of the attacks m unlike a case of Sydenham's chorea; nevertheless the findings were of as pointing to the seat and nature of the lesions likely to exist in the c of childhood.

We must bear in mind the fact that there are several separate for chorea, and the pathology of each must necessarily be more or less diffe also, we should not forget that coarse brain lesions in many regions giv to choreic movements which do not constitute typical chorea. It is impo to believe that an attack of chorea which has been brought on by fright which has been completely cured in seven or eight weeks, can have bee result of structural changes such as have been described by the writers q above.

In chorea most of the symptoms point to the cerebral cortex as the s the disease, with the motor region principally involved. This view is out by the fact that in many cases the movements are markedly unila and this could hardly be the case were the spinal cord the seat of the dis besides, in chorea, the movements cease during sleep. The face is frequ affected, and the arm more frequently than the leg. The intellectual dis ances which are present in chorea also point to the brain.

The pathology, then, of chorea may be stated as follows:

That in acute cases the disease is due to nutritive changes in the cort the brain, the motor region being more affected than elsewhere; that the cause of these nutritive changes may be either fright or mental strain, like overstudy; and in some cases the changes are due to an alteration in the blood. This blood change is the result of some toxic condition, which, while not identical with acute rheumatism, is, no doubt, allied to it.

It is more than likely that in some cases there is an infectious process from a bacillus; but in the present state of our knowledge we are without facts to prove that transient nervous diseases are caused by microbic infection. In acute chorea there are no gross changes in the nerve centres, and in many cases even the microscope will not reveal any departure from health. If the disease lasts long enough, changes will invariably be found by the microscope. The most constant of these are vascular. The capillary vessels of the cortex will probably be found dilated, and there may be plugging of the calibre of the vessels. Hyaline bodies are to be looked for in the cortex, and the nerve cells will be more or less altered. In some cases lesions will be found in the pyramidal tracts, lenticular nuclei, and in the spinal cord.

Diagnosis. There is seldom any difficulty in recognizing a case of chorea, as the symptoms are usually characteristic, and atypical cases are rarely met with. Imitative and hysterical chorea may be mistaken for that of Sydenham; but in the first the history of the patient and the character of the movements will show the true nature of the affection. Hysterical chorea is almost always seen in patients over seventeen years of age, and the movements are more rhythmical and appear to be under the influence of the patient's will, as they increase in severity whenever the patient is under observation; moreover, the general aspect and conduct of the patient shows the presence of hysteria. The athetoid movements in infantile hemiplegia somewhat resemble chorea, but the presence of paralysis and contractures and the different character of the movements in the former, together with the chronicity of the affection and the want of influence of treatment, make the diagnosis clear. Chorea can scarcely be confounded with epilepsy, although Sachs has referred to a case of petit mal, in which the patient was supposed to have chorea. The irregular movements in disseminated sclerosis may be mistaken for chorea, and this affection is also met with in children. The movements, however, consist in an intention tremor, and there is frequently nystagmus; the onset of the disease is gradual; it is of long duration, and is not benefited by treatment.

Prognosis. This is almost invariably favorable. Death from chorea is rare. In the report of the British Medical Association Investigating Committee there was found a mortality of only 2 per cent. Guillemot (Thése de la Faculté de Paris, *Annual Medical Sciences*, 1894, vol. ii., c. 30), in a study of 720 cases, observed 18 deaths, or 2.5 per cent., and in none of his cases was there a death under seven years of age. In Philadelphia in seventy-four years, from 1807 to 1881, among the deaths from all causes there were but sixty-four attributed to chorea. Of these thirty-eight were under twenty years and twenty-six over that age. It is seldom fatal in young children, and death, as a rule, is the result of some intercurrent disease, like endocarditis or meningitis. Hutchinson (*Philadelphia Medical Times*, vol. vi., page 535) recorded a fatal case in a boy of twelve years, who died two days after his admission to the Pennsylvania Hospital. The chorea began after several days of headache and rheumatic pains. The movements were excessive, and the child died apparently from exhaustion. In this case the heart was found diseased, the aortic valves especially being involved. In chorea insaniens the mortality is great. Fatal cases usually are met with in first attacks, but Sachs mentions a case which occurred in his own practice of a child who died in his second attack.

Occasionally, an attack of chorea runs a rapidly fatal course. Cc Beale[1] have reported the case of a girl of nine years who died after an of but five days' duration. After the first two days the movemen excessively violent, and the child's face wore a terrified expression. was a systolic murmur heard at the apex. The pulse ranged from 160 per minute, but the temperature never rose above 101°. There was ı culty in deglutition. At the autopsy the lungs were congested, and vege were found on the mitral valves at the insertion of the cordæ tendineæ brain and cord appeared healthy, but the pons and medulla were anæ

Recovery usually takes place after a few weeks, and the child ther as well as ever, although it is not infrequently the case that for several ı after the child is apparently well there may be occasionally detected movements or some awkwardness in the actions, if the child is clos served. In giving a prognosis as to the length of an attack we m guarded, as the circumstances surrounding the case influence recovery l The care and attention which can be given to a patient make a great ence as to the duration of the attack.

Treatment. Like all diseases which are either self-limited in durat are incurable, chorea has almost an unending number of remedies. hardly necessary to go over the long list of therapeutic agents whicl been employed, but I will refer only to those which have borne the experience and are principally used at the present day. The first anc important factor in the treatment of chorea is complete rest of min body and the removal of all excitement and external irritants. Ever should be carefully examined for reflex irritation, and should any be p it should, if possible, be removed at once. An examination of the e important in all choreic children, and although it is rarely the case th rection of ocular defects has an immediate influence on the choreic ı ments, the course of a case is tedious and prolonged unless any existin strain is removed. Genital irritation should be looked after, and an adl prepuce or clitoris should be relieved.

In almost all cases it is essential that the child should be removed school and study, and disturbing influences, like exciting games, shou forbidden. The other children in a family are often irritating to a whose nervous system is so much disturbed as it is in chorea, and it i quently of great importance to remove the patient from home, or, at any to seclude him as much as possible. But little physical exercise shou allowed, and in many cases absolute rest in bed is requisite. In the vi forms of chorea the patient is compelled to keep in bed, as he is unable up; but even where the violence of the movements does not necessitat recumbent position, rest in bed, with quiet surroundings, is often the plan of treatment. Should the patient be kept in bed, it is well to gentle massage given once or twice a day, as it keeps up the nutrition ol muscles and also has a soothing influence. It is of great importance to a mental irritation and restlessness by means of a cheerful room and a ple nurse. In cases in which the movements are excessive care must be tak prevent the patient from injuring himself. He should be put in a bed padded sides, and it is necessary to have these extend high above the tress, so that the patient may not throw himself out of bed in his contort This accident has come under my observation, even where the sides ol bed were two feet high. If a suitable bed cannot be obtained, the pa may be placed on a mattress upon the floor. Dr. John Abercrombie has gested the use of a hammock in which the patient may be slung. The inj which may be received by a choreic patient by striking himself against hard objects sometimes take an unfavorable course, owing to the cachectic condition of the patient, and may run into blood-poisoning and cause death in this way. Should bed-sores occur it may be necessary to use a water bed, but the water bed is not always a satisfactory arrangement for a choreic patient, on account of the ease with which it is shaken by every movement.

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[1] British Medical Journal, April 14, 1888.

One should not forget the risk of taking the temperature of a choreic patient in the mouth for fear of the thermometer being bitten.

The utmost cleanliness should be observed in the patient and his surroundings by means of daily bathing and frequent changes of the bed linen, and the bowels should be kept open and the skin in a healthy condition. In many cases the skin is harsh and dry, and it is beneficial to give an occasional hot-air bath or to administer diaphoretics. The patient's hair should be cut short, so as to avoid the irritation which arises from combing it, and closely cropped hair also keeps the scalp cooler. Plenty of wholesome food should be provided, and in cases in which deglutition is interfered with minced meat, broths, and milk should be given freely. Cocoa or chocolate is taken readily by children and are extremely valuable articles of diet.

Many patients do very well without the use of drugs, provided rest, quiet, and seclusion can be enforced. In hospitals it is seldom necessary to administer medicines, except when the movements are extremely violent. In private practice it is difficult to enforce absolute rest, except in those cases where the means of the family will permit of the employment of a trained nurse. The advantages of rest are often seen in cases which have resisted treatment by drugs for weeks, and who are cured after a few days of absolute rest in bed. Drugs, however, are not without distinct influence on the course of chorea, and we have daily evidence of this in the cases which are cured at the out-patient services of our hospitals. The duration of an attack is undoubtedly cut short by certain medicines, and we frequently see cases which have had no treatment and have remained stationary, or have been growing worse for months, which are cured within a short time by the administration of suitable remedies.

Among the various drugs which have been given in chorea arsenic is, by far, the most efficacious. There can be but little question as to the marked curative influence which it has upon the disease. Among the other remedies which have been used with success may be mentioned strychnine, the salts of zinc, the sulphate, oxide and bromide, nitrate of silver, belladonna, cimicifuga, conium, antipyrine, and salicylate of sodium. The use of sedatives in ordinary cases of chorea is not only unscientific and irrational, but is dangerous and cannot fail to leave ill effects, which must unfavorably influence convalescence. Dujarden-Beaumetz (*Bulletin Gener. de Therapeutique*, March 15, 1894), in a review of the therapeutics of chorea, gives his own experience and that of others. Antipyrine in doses of 4 grammes (60 grains) per diem is recommended in chorea of rheumatic origin. According to Legroux this drug brings about a cure in from fifteen to twenty days of treatment. It is to be observed however, that the author states that the result is best attained if the remedy is used in the declining period of the malady. In serious cases antipyrine is not sufficient, and then chloral in large quantities is advised. Bouchet has given it in doses of from 3 to 5 grammes in twenty-four hours (45 to 75 grains). Bromide of potassium in large doses, 30 to 60 grains a day, is also recommended by these writers. Dujarden-Beaumetz has tried exalgine in doses of 45 grains daily, and Dana and Löwenthal have given it in still larger quantities, 60 to 100 grains daily. Morphine has been advised as a routine treatment, but I place it in the same category with the drugs named above. Churton recommends its administration as follows:

Begin with ⅙ grain (0.01 gramme) hypodermically, immediately following by inhalation of chloroform for a few minutes. Gradually increase the morphine to ½ grain, (0.03 gramme), always following with chloroform. If necessary the chloroform may precede the injection. (T. Churton, *British Medical Journal*, March 24, 1894.)

When we consider the dangerous results which have followed the use of all these drugs, especially exalgine, it seems to be unjustifiable, to say the least, to administer these remedies in such doses.

Ratchford (*Medical News*, April 22, 1893), believing that the pathology of chorea is a scrofulous anæmia, recommended large doses of the iodide of iron. I have used it, however, without as good results as from arsenic. Pianese, whose views on the infectious origin of chorea have been referred to, treated 13 cases with salol, and found in his experience that it is superior to all other drugs; he also considered the favorable effect of this remedy an argument in behalf of the infectious origin of the disease. Wood (*Boston Medical and Surgical Journal*, August 24, 1893) advanced the theory of the spinal origin of chorea, and gave large doses of quinine, because he found that in dogs this drug produced a marked inhibitory influence upon the cord. Potts has recorded a series of cases which were successfully treated by quinine, and I have seen one or two in which the effect of quinine was good, but it is by no means universally applicable.

The best mode of administering arsenic is to give Fowler's solution in ascending doses. In children of seven years, 5 drops three times a day may be given as an initial dose, and, if no idiosyncrasy against the drug is manifested within three or four days, the dose can be increased by 1 drop three times a day until physiological effects are seen. Children frequently bear from 15 to 20 drops three times a day before saturation occurs, and it is very rare to meet with any grave effects when the maximum dose is reached. Vomiting or diarrhœa, with puffiness of the face, headache, or pain in the stomach, are indications for stopping the arsenic. When toxic symptoms occur the remedy should be suspended for two or three days, and, as by this time tolerance of the drug is usually established, the patient may begin again with the dose which was borne before the toxic symptoms were shown. In the majority of cases, however, at this period the choreic symptoms are so much diminished that it is well to resume the arsenic in small doses. If arsenic is not well borne, it is better to increase the dose more gradually, giving but 1 drop additional each day. At the Infirmary for Nervous Diseases we are in the habit of giving the parents of each child who is taking arsenic for chorea, a printed slip of directions for the gradual increase of the dose and the indications which should cause the withdrawal of the medicine. For example, a child is told to begin with three drops three times a day. On the second day the dose is four drops in the morning, three drops at noon, and three drops at night; on the third day, the dose is four drops in the morning, four drops at noon, and three drops at night; the increase to be continued in the same way.

It is quite frequent to meet with various inflammatory conditions of the skin from the large doses of arsenic which are given in chorea, and arsenical conjunctivitis is comparatively common in children who are taking the drug. We must bear in mind, however, that untoward effects have been reported from arsenic given in medicinal doses, and the patient should never be allowed to take arsenic in ascending doses unless he is under the supervision of a physician.

Bars showed a case before the West Riding Medico-Chirurgical Society (*British Medical Journal*, 1893, vol. i., p. 239) in a boy aged twelve years, who was totally disabled by multiple neuritis which followed the administration of liquor arsenicalis in the treatment of an attack of chorea. The maximum dose of arsenic which was given was twelve drops. The symptoms subsided speedily on the withdrawal of the arsenic, and the boy was discharged from the hospital perfectly well; but a month later he was readmitted with loss of power in the arms, legs and trunk, and with some anæsthesia and tenderness over the nerve trunks. The tendon reflexes were absent. Bars considered the polyneuritis due to the arsenic which had been administered.

As soon as the acute symptoms have subsided the dose of Fowler's solution should be reduced to two or three drops three times a day; and as soon as the symptoms have entirely ceased the arsenic should be withdrawn entirely.

When arsenic is not tolerated by the stomach, I have found cimicifuga in doses of from ten to fifteen drops of the fluid extract, three times a day, useful, and the fluid extract of conium pushed to fifty or sixty drops three times a day is a good remedy. Salicylate of sodium has been recommeded in cases of rheumatic origin. I have given it a faithful trial in a large number of cases without finding it of special value. It is decidedly inferior to arsenic, even where there is a clear rheumatic history preceding the attack. Of course, should there be any symptoms of acute rheumatism present, the salicylates are indicated.

During convalescence iron may be given in full doses, and should be continued until the child is entirely restored to health. In all cases in which there is marked anæmia, iron should be given from the beginning; but, unless the patient is distinctly deficient in red blood cells, there is no advantage in giving iron in conjunction with arsenic. As far as my observation goes, there is no specific action in iron. Cod-liver oil is often advantageous in children who are badly nourished and feeble. General sustaining remedies are more or less useful in all cases.

During the decline of the disease light gymnastics and Swedish movements may be used with advantage, but it is not desirable to employ these during the acute stages of the disease.

In severe cases with incessant and violent contortions, in which there is sleeplessness and sometimes delirium, it is essential that rest should be obtained. Chloral, in moderate doses, usually answers admirably in these cases. Hyoscine hydrobromate is sometimes better than chloral in producing quiet sleep. If these remedies fail, then morphine should be administered hypodermically.

A case of excessively violent chorea is reported by Himmelsbach, of a young girl, aged eighteen years, who was admitted to the Buffalo General Hospital with such an extreme case of chorea that nourishment could not be administered by either the mouth or the rectum for some days. Chloral and hyoscine both failed to give relief, but after a hypodermic injection of a quarter of a grain of morphine the patient at once improved.

Occasionally inhalations of chloroform or ether may be necessary, and the former is most efficacious. Sometimes the wet-pack is very soothing in these cases. In cases of extreme violence it is of the utmost importance to use supporting measures and to carefully watch the heart's action, lest there be failure of this organ. Should the case become chronic, change of air to the seashore or to the mountains often will result in a speedy cure.

CHOREA INSANIENS.

Chorea insaniens, or maniacal chorea, is a variety of chorea which should be considered separately from the common type.[1] It occurs chiefly in females at or soon after the age of puberty, or as the result of pregnancy. The mental disturbance may precede the choreic movements, or, what is more common, the maniacal symptoms occur after the choreic movements become violent and excessive. Chorea insaniens, however, must not be confounded with the form of chorea which occurs in the chronic insane. The attack may begin with movements of moderate intensity, which become rapidly excessive, but frequently within a short time the muscular spasms are violent in the extreme, and delirium develops speedily. If the attack should be acute and severe and the temperature runs high, the case is likely to terminate fatally within a few days. In milder cases there are merely delusions of a slight form, and the patient has extreme loquaciousness similar to that seen in acute mania. In some cases the excitement subsides in a few days, and leaves the patient in a dull, listless and depressed state, and sometimes with persistent delulsions. A patient in this condition may refuse all nourishment, which then has to be administered by force. This state may pass away by degrees, but in some cases it has lasted for months after chorea has ceased. Golgi has reported a case in which after the subsidence of the acute stage there was gradually increasing mental failure during a period of ten years, and death at the end of this time.

TABLE II.—CHOREA INSANIENS—41 CASES.

Age.	Male.	Female.	Recovered.	Died.	Insanity contin'd.	Reference.
14-25	5	18	8	10	5	Gay, Brain, vol. xii. p. 149.
23	...	1	1	...	...	Clouston, Mental Diseases, p. 324.
Adult	1	...	1	...	...	“ “ “ “
23	...	1	...	1	...	Bucknell and Tuke, Psychological Med., p. 372.
17	...	1	1	...	...	“ “ “ “ “ “
24	...	1	1	...	...	“ “ “ “ “ “
19	1	...	...	1	...	Powell, Brain, vol. xii. p. 157.
20	...	1	...	1	...	“ “ “ “
18	...	1	...	1	...	Osler, Practice of Medicine, p. 935.
70	...	1	1	...	...	Ferrier, Lancet, 1891, vol. i. p. 1379.
20	...	1	1	...	...	Banks, Dublin Hospital Gazette, vol. vii. p. 58.
22	...	1	1	...	...	L. Meyer, Archiv für Psychol., vol. vii. p. 585.
19	1	...	1	...	...	J. Russell, Med. Times and Gaz., 1869, vol. i. p. 64.
20	1	...	1	...	...	“ “ “ “ “ “ “ “
...	...	1	1	...	...	“ “ “ “ “ “ “ “
56	1	...	...	1	...	B. Lewis, Med. Times and Gaz., 1876, vol. ii. p. 280.
27	...	1	...	1	...	Berkeley, Johns Hopkins Reports.
7	1	...	1	...	...	Gay, Brain, vol xii. p. 149.
18	...	1	...	1	...	Goodell, Amer. Journ. of Obstetrics, vol iii. p. 140.
	11	30	19	17	5	

Gay[2] records a case of chorea insaniens, and has made an analysis of 23 cases. I have been able to collect 18 additional cases from other sources. Of these 41 cases 11 were in males and 30 in females. The ages varied from seven to seventy years of age, the majority of the cases, however, being between seventeen and twenty-seven. In the case recorded by Gay of a boy of seven years, it is rather doubtful if the patient had genuine chorea, and the writer himself admits that the case was open to question. Seventeen of these cases terminated fatally, a mortality of 40.1 per cent., and 19 recovered. In five cases the insanity continued.

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[1] Chorea insaniens was described by Marcé, Mémoire lu à l'Académie Impériale de Médicine, April, 1859, who says that this name was first used by Bernt, of Prague, in 1810.

[2] Brain, vol. xii., 1889.

Osler decribes the case of a girl of eighteen years of age, who was employed in a hotel. She was very much frightened by seeing two men fighting, and dropped a tray of dishes which she was carrying. For this she was severely reprimanded, and within two days developed choreic movements. She almost immediately became maniacal. The temperature rose to 105°, and death occurred at the end of ten days. There is no note of an autopsy having been made.

Berkeley[1] records the results of an elaborate examination made in a case of chorea insaniens. The patient was twenty-seven years of age, and had a history of rheumatism at sixteen years of age. When she was twenty-three she had a second attack of rheumatism, after a period of intense application to her studies. After she had been ill for a few days her mind became affected; she was delirious for five weeks, and during three weeks of this time she had choreic movements. The mental symptoms and choreic movements disappeared entirely, and the patient remained well except that she had headaches. The fatal illness began suddenly with jerking of the muscles, which awakened her in her sleep. Within a few days mental symptoms developed. There was extreme restlessness and wild delirium, and the temperature rapidly rose, reaching 105°. Death occurred at the end of eighteen days. During the illness a parotid abscess developed. On the admission of the patient to the hospital there was a papular bronze-colored rash over the body, which was scaly in places; ulceration of the pharynx, and enlarged cervical and inguinal glands. On the under surface of the clitoris was "an elevated and excoriated non-indurated sore." At the autopsy were found acute endocarditis, catarrhal pneumonia of both lungs, and an abscess of the parotid gland. Neither the brain nor membranes showed gross changes, but under the microscope a number of lesions were found in the brain and membranes. These were principally vascular; and Berkeley believes that the disease in this case was of infectious origin.

Clouston[2] considers that maniacal chorea and rheumatic insanity are analogous conditions, and that the mental disorder is the result of rheumatic poison in both, although he admits we may have choreic insanity both in early youth and in advanced life without any acute rheumatic symptoms. The delirium in these cases he points out as being of an inco-ordinate jerky kind, like the muscular movements.

Pathology. Chorea insaniens is usually due to either a blood-poison like rheumatism, or the absorption of septic material, or it may occur in connection with pregnancy. Several cases have been recorded where the disease was the result of fright. In the few autopsies which have been recorded there has been found meningitis or vascular changes in the brain, and endocarditis has been met with.

The prognosis is grave, especially in connection with pregnancy, but the younger the case the greater is the prospect of recovery. The duration of the disease varies from a few days to several weeks, and occasionally a chronic condition remains which lasts for years.

Treatment. This consists in quieting the mental excitement and in giving the patient rest. Hypnotics are necessary, and various forms have been used with different degrees of success. Morphine has been found useful in many cases, and in other instances chloral and hyoscine hydrobromate have been of greater efficacy than opiates. It is important to keep the patient from injuring herself by keeping her in a bed with padded sides, and so strapping the patient down is found necessary, as such cases frequentl themselves by the violence of their movements. As much food as given should be used, and if the patient is unable or unwilling to voluntarily nourishment must be administered by the stomach-tube enemata.

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[1] Johns Hopkins Hospital Reports, August, 1891.

[2] Clinical Lectures on Mental Disorders, p. 319.

CHRONIC CHOREA.

Chronic chorea embraces four classes of cases: First, a form which is tially chronic from its beginning, and may be congenital or have its at any period of life; secondly, the hereditary form; thirdly, senile and, lastly, those cases which begin with an acute attack, but become c and last for months or years, although ultimately they recover. Th form includes all of the chronic choreas which are usually met with in and depends almost invariably upon some gross lesion of the brain or cord. We meet with two types of the affection: the cerebral and the

The first form may begin at any period of life, and is frequently c ital. The movements are more rhythmical and more continuous than th Sydenham's chorea, and have been said by some writers to resemble the which occurs in dogs. This is more especially the case in the spinal f chronic chorea. In this variety there is no mental disturbance, and but movements of the face and head. In the cerebral type there is more to be mental enfeeblement, and disorders of speech are liable to occur. progress of the disease is very gradual, and the patient may live to a vanced age without any material increase in the symptoms. As a rule, ever, the patients do not live beyond fifty years of age.

There is no doubt that many cases of post-hemiplegic chorea or inf diplegias are confounded with chronic chorea, but the distinction can re be made if the history and symptoms of the case are closely followed. chell and Burr[1] have related the case of a young man, nineteen years o in whom the choreic movements began in infancy and were probably genital. His maternal grandmother and mother suffered from chorea d most of their lives. The movements in this case involved all of the l trunk, and face, and in it there were occasional spasmodic attacks, ac panied with rigidity of the legs. The patient's condition varied: at tim was well enough to earn his living, while at other times there were ex bations in which the movements were quite violent. Chronic chorea is i able, and no medication seems to alleviate the severity of the movem except for brief periods.

HEREDITARY CHOREA.

SYNONYMS: Huntington's chorea and chronic progressive chorea.

Hereditary chorea was first described by Dr. C. O. Waters in a lett Dr. Dunglison[2] in 1841. This letter contained an excellent description remarkable and undescribed form of St. Vitus' dance, which he says somewhat common in the southeastern part of New York, and was k among the common people as the "Megrums." He notes the characte features of the disease, namely, heredity, the fact of its rarely occurrin fore adult life, and its incurability. He also refers to the fact of there in all cases a state of mental deterioration sooner or later; in short, in this brief communication, Dr. Waters gives as lucid a description of the disease as has been presented by anyone.

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[1] Transactions American Neurological Association, 1890, p. 8.

[2] Practice of Medicine, vol. ii., p. 245.

Irving W. Lyon[1] also presented a report on chronic hereditary chorea. His cases, like those Waters, were residents of the State of New York; and he also alludes to the fact that the families in which these cases occurred were popularly called "Megrim Families."

Huntington,[2] however, wrote the first paper on this disease which attracted the attention of the medical world. His account was one of great interest and clearness, and he believed that this form of chorea which he described was peculiar to the eastern end of Long Island. His father and grandfather, who had practised medicine in this locality for seventy-eight years, had been acquainted for many years with certain families in which chorea had existed for generations. The name Huntington's chorea has been used in connection with this disease ever since this writer's account of the disease was published.

The cases of this malady observed in America have been chiefly in the States of New York, Connecticut, New Jersey, and Pennsylvania. From the fact that these are adjoining States, it seems probable there has been some common ancestry in all of these cases.

East Hampton, according to King,[3] was settled in 1649, and choreic families have resided there since then, and several emigrations have occurred, one of them to Delaware County, New York, where Dr. Waters's original case lived.

A number of cases, however, have been reported in Germany and France since the publication of Huntington's paper, and a complete list of these will be found in my paper on "Hereditary Chorea."[4]

Lanois has written an admirable article on hereditary chorea,[5] in which he records cases of his own, and also a review of the entire subject. Huet's monograph contains the complete literature of the disease.[6]

In hereditary chorea there is almost invariably a history of the disease in the generation preceding. The distinction between hereditary chorea and an hereditary tendency to chorea must be borne in mind. In the first there is a specific disease, with characteristic symptoms; in the other, several cases of chorea may occur in a family, or one of the parents may have had chorea in childhood. It occurs between the ages of thirty-five and forty years, although it may occur earlier or later. It rarely begins, however, after forty-five years of age, and there are but few instances in which it has occurred previous to thirty. Gray[7] detailed the case of a child which he considered one of congenital Huntington's chorea, although there had been no other members of the family similarly affected. Dr. S. C. Stevens reported, in a paper read before the South Carolina Medical Association, two brothers, one of eleven and the other twelve years of age, both of whom were suffering from chorea which had existed in one from birth, and in the other the disease was noted during the first year. They belonged to a family in which the hereditary form of chorea had existed for several generations.

Two cases of hereditary chorea occurring in twins are reported by James W. Russell.[8] The father and his mother suffered from the same malady; one brother was attacked at the age of twenty-six, and the other a little earlier.

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[1] American Medical Times, December 19, 1863.
[2] Medical and Surgical Reporter, April 15, 1872.
[3] New York Medical Journal, April, 1885.
[4] Medical Record, March 12, 1892.
[5] Revue de Médecine, August, 1888.
[6] Chorea Chronique, Paris, 1889.
[7] Transactions of the American Neurological Association, 1892.
[8] Birmingham Medical Review, January, 1894, p. 31.

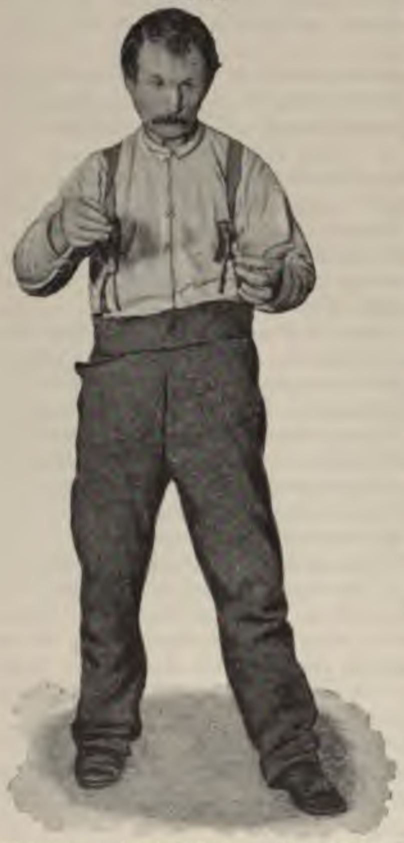
The following table is from Huet's memoir, and gives the age of onset in 67 cases:

TABLE III.

10 years	1
10 to 15 years	1
15 to 20 "	2
20 to 25 "	1
25 to 30 "	5
30 to 35 "	16
35 to 40 "	13
40 to 45 "	11
45 to 50 "	7
50 to 55 "	8
60 to 65 "	1
65 to 70 "	1

As a rule, if one member of a choreic family escapes the disease, his descendants do not have it. There are, occasionally, exceptions to this rule, but in most cases the rule holds good.

FIG. 60.



Adult chorea, showing the dramatic position involuntarily assumed. (DERCUM.)

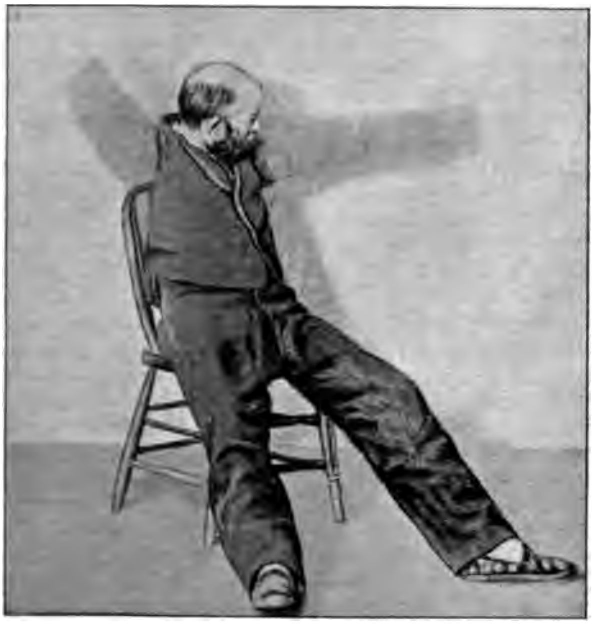
As many cases occur in males as in females. In families who suffer from this strange affection the sexes are equally affected. The disease sometimes begins during pregnancy. In two families, which I have reported, there were eleven males and fifteen females who suffered from chorea. The women in families in which this malady exists are usually prolific, and large families seem to be the rule.

The movements in hereditary chorea begin usually in the upper extremities, and are slight. They are similar in their features to those of Sydenham's chorea; but as the disease progresses the movements become more general, and involve the upper and lower extremities equally; the trunk and facial muscles are also involved, and speech and deglutition become greatly interfered with. When the disease is fully developed, the movements present quite different features from those of ordinary chorea. They are more extreme, more rhythmical, and more inco-ordinate. A wider range of movements come into play, and whole groups of muscles are brought into action.

The gait is characteristic. The patient takes a few long steps naturally, then makes a long step with one leg, bringing up the other quickly to it, and makes one or two hops, giving the appearance of dancing; he then takes several more natural steps, which are again interrupted by the dancing steps. Another feature which has been observed in these cases is a tendency which the patient has to remain standing in one position for a length of time.

One of King's patients was accustomed to stand for many hours, especially when trying to masticate food, with her body thrown back on a level with her hips, and her arms extended backward by the sides of the body, the palms looking forward.

FIG. 61.



Adult chorea, showing excessive and rapid movements. The arms are blurred in the figure in consequence. (DERCUM.)

Speech is very much interfered with, but it is seldom that any involuntary ejaculations are made. The words are indistinctly and haltingly pronounced, but there is no scanning. Deglutition is also difficult in most cases. As the disease progresses the movements become more violent, more continuous, and less under the control of the patient's will. In the early stages the movements may be arrested by voluntary effort, and some writers have regarded this ability to control the movements as a point of distinction between this malady and Sydenham's chorea; but in the later stages not only does voluntary effort fail to control the movements, but when a patient is told to try and keep still the contortions become more violent. Strange to say, that in spite of the unceasing and extreme character of the movements, the patient never complains of fatigue, and prefers to remain out of bed. In many cases there appears to be a remarkable insensibility to pain. Patients bruise and injure themselves, and do not suffer.

Mental disorder is found in all cases, and usually in the terminal stages. There are cases, however, in which mental disease occurs first, and a number of instances of this form of chorea have been met with in insane asylums. Diller[1] recorded 39 cases of hereditary chorea, 33 of which were found in asylums.

From these facts I have come to the conclusion that there are two forms of hereditary chorea; one in which the muscular disorder begins first, and after a number of years mental deterioration begins; and another type in which insanity precedes for some years the chorea.

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[1] American Journal of the Medical Sciences, 1892.

The usual form of insanity which occurs is melancholia, with sometimes a suicidal tendency. There are delusions of various forms; at times of persecution, and at other times delusions of grandeur. The patients are suspicious and unsociable. Occasionally, they may be violent. Rheumatism has been present in a number of cases which have been recorded; and in several there has been disease of the heart. The knee-jerks are exaggerated, and ankle clonus is sometimes present. There is apparently but little change in the sexual functions, several women having been recorded as having borne healthy children after having become choreic.

Pathology. The pathology is undoubtedly like that of other developmental diseases, like Friedreich's ataxia, and the disease is due to a hereditary or congenital defect in the motor tracts of the brain and cord. This is shown by the fact that a number of cases are known to have begun in early childhood. The disease is distinctly different from Sydenham's chorea in its pathology as well its other features. A number of autopsies have been made within the past few years, with more or less uniform results. There has generally been found evidence of disease in the motor region of the brain and inflammation of the meninges.

An interesting point in the pathology of hereditary chorea which points to the possibility of its being an infectious disease is the fact that in Long Island, where the disease has existed for years, tetanus is also extremely common.

Lesions in the cord are frequently present. In an autopsy which was made in a case of mine[1] there was found congestion of the meninges, with subarachnoid œdema and adhesions between the dura and the skull. Owing to an accident, the brain was not examined microscopically. In the spinal cord, however, there was found an increase in the connective tissue in the white matter and thickening of the walls of the bloodvessels. The region of the central canal was occupied by a mass of nuclear tissue much more abundant than in normal cords. Greppin[2] reports an autopsy in a patient in whose family chorea, insanity, brain, and spinal disease had occurred. He became choreic at the age of fifty, and died in a condition of dementia. The post-mortem examination showed pachymeningitis and leptomeningitis. The convolutions of the brain were flattened, and slight atheroma of the vessels of the base was found. Everywhere were found through the gray and white matter accumulations of cellular elements, with "not much developed membrane and nucleus, and composed of many granular nuclei, so that here and there ganglion cells and nerve tubes were completely obliterated by them. In many places the cells were conglomerated, and, having lost their nuclei, made a deformed mass. These masses were most numerous in the white matter of the frontal, parietal, and temporal convolutions. Osler[3] found, in one autopsy which he made, meningitis and atrophy of the convolutions and some bloodvessel changes. Phelps[4] calls attention to the analogy between hereditary chorea and paresis. Both diseases begin in the adult life; in both the mental symptoms are of the same character; both are essentially chronic; and the post-mortem findings in both are similar.

The diagnosis of hereditary chorea depends upon the history of the disease, but may be confounded with Friedreich's ataxia. In the latter disease patients are attacked in early life, and the movements are athetoid rather than choreic. In disseminated sclerosis the movements cease while the patient is at rest, and occur only on voluntary effort. In paralysis agitans the tremor is fine and rhythmical, and there is no disturbance of speech or deglutition.

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[1] Medical Record, March 12, 1892.

[2] Schmidt's Jahrbücher, June 15, 1893.

[3] Practice of Medicine, page 914.

[4] Journal of Nervous and Mental Diseases, October, 1892.

Prognosis. The disease is incurable and terminates fatally by exhaustion or some intercurrent disease. It may last for fifteen or twenty years, during which time there is a progressive increase in all of the symptoms. The disease is seldom arrested.

Treatment. There is no treatment for this affection which affords positive relief. Arsenic has been recommended and has been given in large doses, but without benefit. Hyoscine in full doses sometimes gives temporary relief, but the influence of the drug is soon exhausted. Sulphonal has afforded temporary amelioration of the symptoms, but with no lasting good. In one case that I have seen the patient was made much more comfortable by being kept in bed. In all cases it is important to keep the patient clean by frequent bathing, and to give food which can be easily masticated. Excitement and disturbances of all kinds make the patient worse.

SENILE CHOREA.

Irregular choreic movements are not infrequently met with in the aged, and sometimes fully developed and typical chorea occurs in persons after the age of sixty. A number of cases have been reported of late years, and I have seen several myself. Of the cases which have been under my care one was eighty-two years of age, another eighty-six, and another eighty. The movements in senile chorea are seldom violent, and are frequently capable of being controlled by voluntary effort. The legs are less affected than the arms. Sometimes the movements are extreme and involve the face and muscles of speech to such an extent that the patient cannot articulate intelligibly. Speech is seldom affected, and the facial muscles are not involved. It is by no means an incurable disease, and in two cases which I have seen the movements ceased entirely after treatment. Charcot[1] thought that in old persons suffering from chorea there was almost invariably a condition of dementia, but he probably refers to a different class of cases from those in which the chorea is the result of mental deterioration. In some of the cases which I have seen there was a history of rheumatism, and valvular disease of the heart was present.

Senile chorea may be mistaken for paralysis agitans and senile trembling. In senile trembling the movements are generally confined to the head, and consist of a continuous rhythmical tremor. In paralysis agitans there is loss of power in the parts involved, the tremor is regular and gesticulatory, and there is a history which shows that at first the tremor was under the influence of the will, and afterward became more violent and continuous; moreover, the peculiar facial expressions of paralysis agitans help in clearing up the diagnosis.

The treatment of this form of chorea is like those for other forms of chronic chorea, and consists in keeping the patient as free from excitement as possible, and enforcing quiet. Arsenic has a distinctly curative influence in the treatment of these cases, and some are benefited by the use of moderate doses of strychnine.

In senile chorea there is probably some degenerative changes in the motor area of the cortex. In one of my patients, a man of eighty years, there had been for several years distinct choreic movements of the left arm, which did not increase in severity, but rather diminished in their extent. He became suddenly hemiplegic on the left side, and died after a few days' illness. This case would seem to indicate that there had been disease in the cortex which had at first been merely sufficient to cause the irregular movements, but that finally the degeneration became so great that there was loss of function in the motor region. Changes have been frequently found in the brain of patients dying from senile chorea, but there have been no constant lesions discovered. Berkeley made a post-mortem examination in a case of seven years' duration, and found many lesions in the cortex, but they were probably secondary in character.

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[1] Medical Times and Gazette, March 9, 1878.

CHOREA MAJOR.

Chorea major of the French is simply a form of hysteria. The name has also been applied to pandemic chorea which occurred in the Middle Ages, and which has likewise occurred in Kentucky and elsewhere. The movements of hysterical chorea are usually violent and continuous, and are never controlled by voluntary effort; on the contrary, they appear to be exaggerated when the patient is told to try to arrest the movements. Other stigmata of hysteria are also present, and the diagnosis is therefore not difficult. In a case of hysterical chorea which I saw some years ago in a young married woman, the patient's movements were continuous and so violent that she was very much emaciated, and sleep could not be obtained except under the influence of large doses of chloral, and there was distinct mental disturbance. She recovered completely under moral influences and the withdrawal of the drugs to which she had become habituated.

HABIT CHOREA.

This affection has been called by Gowers "habit spasm," as he does not consider it a true form of chorea, and the term "convulsive tic" has also been given it by the French. The latter name, however, is unfortunate, as it has also been used for painless facial spasm. This affection was described by Weir Mitchell.[1] He found that it was most frequent in girls from seven to fourteen years of age. In many cases there is a simple grimace, or a motion made in some part of the body. The affection lasts for a few months and disappears. In other cases the habit movement first formed does not last, but another takes its place, and thus a variety of other movements may be produced which become more troublesome and more persistent. In many cases there is some failure in the general health of the child, but in others the odd grimaces or movements seem to have developed, as it were, by accident, and the child becomes unable to control them. The movements are frequently confined to the face, and consist in winking of the eyes or wrinkling of the forehead. Sometimes the movements are confined to the muscles about the mouth, and twitching of the corners of the mouth, or the protrusion of the tongue occurs, and is suddenly repeated. In other cases the movement consists in a shrugging of the shoulders, and in one or two cases that I have seen the muscles of the ear which, as we know, are usually not capable of being moved voluntarily, are thrown into frequent contractions. In a patient twenty-eight years of age, who came under my observation some years ago, there was a frequent shrugging of the neck to one side, and the chin was drawn upward with an occasional shoulder movement. This man ecially those forms in which the movements are confined to the face. De :weinitz[1] has related a number of cases of habit chorea which were due to action errors, and which were entirely relieved by properly adjusted es. Diseases of the nose of an obstructive nature are also liable to duce this affection. In boys masturbation is said to be a cause of severe l intractable cases of habit chorea. In many cases imitation can be ed as a direct cause. It is quite common to see more than one child in a ily with some form of choreic twitching, and it is not uncommon to find the affection in a parent or an older member of the family has been uired by one of the children.

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[1] Lectures on Nervous Diseases, Philadelphia, 1881.

he character of the movements vary in different persons in extent and ee, and may occur at intervals of a minute or two, or there may be a few in the course of a day, or the choreic spasm may be almost conous.

hen confined to the face the affection often occurs in the form of blinkof the eyes, or as a series of spasmodic contractions of the orbicularis ebrarum, simulating blepharospasm; in some cases there are twitchings he mouth involving the zygomatic muscles, and in others a broad smile peated at intervals without any occasion for mirth. Gowers refers to a gyman who was in the habit of making meaningless smiles, and was unto control the habit until he was treated for the affection; and I have a one or two cases myself in which this involuntary smiling was repeated ntervals, much to the embarrassment of persons speaking to the patient, o seemed to be laughing at some serious remark.

Wrinkling of the brow is a frequent form of the malady, and alternate conctions of the occipito-frontalis muscles give rise to active twitching of the ene scalp. In a lady whom I have seen there was spasm involving the occipitontalis muscles. The effect upon her bonnet, in which some long feathers re placed, was very striking. In other cases movements of the head are t with, either in the form of nodding or shaking, and these movements ay either be continuous when the head is unsupported, or may occur at g intervals. I have seen one patient who was so much annoyed by the dding of her head that, being unable to control it otherwise, she acquired e habit of biting her cheek whenever the tendency to nod was perceived her.

can be checked by voluntary effort. In many cases the disease is duration, but in others the habit once formed continues throughout l resists every form of treatment.

In nearly all cases it is of first importance to look after the general of the patient. The child, who should be taken from school, should t to take sufficient outdoor exercise, cold baths, frictions, massage, a liberal administration of iron are almost always of benefit. If the fails to yield to these means, arsenic should be given in ascending do as it is administered in the treatment of Sydenham's chorea. Mitcl found that arsenic given hypodermically is successful in cases whic resisted the same remedy when given by the stomach. Scolding and never help in the cure of a case, but frequently the promise of reward to will aid greatly in bringing about recovery. It is quite common witl promise the child some favor or reward when he is able to control th ments for a certain number of minutes, and some new favor is grant the increase in the length of time that the movement can be controlle

In all cases in which the orbicularis palpebrarum and occipito-fi muscles are affected the eyes should be examined for defects, and the and nose should also be investigated. In fact, every possible source of irritation should be traced and relieved.

"Button-makers' chorea" has been described as occurring in girls w employed in button mills, as the result of too close application to their The movements of the fingers continue when they are not at work, foll the same motions as those employed when making buttons. This n regarded as a form of habit chorea.

ELECTRICAL CHOREA.

Electrical chorea, or Dubini's disease, is met with in Lombardy and of Italy. The term has also been applied to varieties of hysterical and to cases of ordinary chorea in which the movements were violen shock-like. Dubini's disease, however, is a totally different affection, not a true chorea. It resembles chorea, however, in the spasmodic ments which occur, but, in adition, there is a progressive palsy and mu wasting. The muscular movements are sudden and shock-like, resen the movements which are produced by a sudden current of electricity; the name electric chorea. The disease ends fatally in many cases.

The etiology of the affection is obscure. It has been ascribed to ma influences, on account of the fact that it is prevalent in low, marshy dis

The peculiar shock-like contractions in Dubini's disease begin usua the arm, and spread thence to the leg of the same side; and later the site side becomes affected. In a few months the members which wer affected become feeble, and muscular atrophy, with loss of faradic i bility, is present. The paralysis extends by degrees and soon be general. Occasionally epileptiform convulsions are met with durin course of the disease; in acute cases there is elevation of temperature.

Autopsies which have been made in fatal cases have revealed no con lesions in the central nervous system. It has been suggested that it is a of infectious myelitis, due to malaria, but no proof of this has been of Treatment has not been of any avail.

SALTATORIC SPASM.

SYNONYMS. Latah; Miryachit; Tic Convulsif; Palmus; the Jumpers; Gilles de la Tourette's Disease.

Under these various names is described a peculiar affection which most writers classify with chorea, but which is not a form of that disease. For convenience, however, it may be treated of here. The malady was first described by Bamberger in 1859. It occurs more frequently in males than in females, and it may exist at any age—from ten to seventy years. In the cases described by Bamberger there is frequently a history of hysteria and epilepsy or some other disturbance of the nervous system; and in other instances there has been some depressing influence upon the system at large.

There are sometimes premonitory symptoms, such as tremor or stiffness of the legs; but in other cases the attack is sudden. When the patient attempts to stand there are violent contractions of the flexors and extensors of the muscles of the entire leg, so that a jumping and springing movement is produced. These jumps are frequently so violent and often repeated that the patient is thrown upon the floor. The movements occur only upon attempts to stand, and while the patient is either lying or sitting there are no involuntary contractions of the legs. The disease may be of only short duration, or it may last for many years. All of the subjects of it are neurotic, and there is, no doubt, a large underlying hysterical element.

This affection has been met with in epidemics in different parts of the world; at least, I believe that the affections described by different writers as occurring in Russia, Canada, Java, and other places, characterized by jumping and imitative movements, are identical with the saltatoric spasm of Bamberger. Beard[1] describes a malady which occurs in certain parts of Maine and Canada. The subjects of this affection are known as "jumpers" or "jumping Frenchmen." This writer found that, in addition to the jumping movements which were made involuntarily by these patients, they had a tendency to do whatever they were told. For example, Beard ordered one man who was sitting on a chair, with a knife in his hand, to throw it. The knife was at once thrown so quickly and violently that it struck a house opposite; at the same time he repeated the order to throw the knife with a loud cry. In the subjects of this disease there was also echolalia and coprolalia; that is, they would imitate any words given to them and made use of indecent language. Beard repeated the first lines of the Æneid to one of these illiterate jumpers, and he immediately repeated the sounds of the words as they came to him in a quick, sharp voice, and at the same time he jumped or shrugged his shoulders and made other violent muscular movements.

Hammond[2] gave an account of a similar affection which occurred in Siberia, and was known there as "miryachit."[3] O'Brien[4] also has given an account of this disease as met with among the Malays, and called by them "latah." He states that on one occasion he was talking with a respectable Malay woman, whom he did not suspect to be a subject of the disease. Suddenly a friend who was with him took off his coat, and the woman immediately began to undress herself and entirely disrobe herself, in spite of his remonstrances, although she was violently angry at the outrage to her sex, and called him an abandoned hog and other abusive names all the time she was undressing. This writer also says that the cook of a steamer, "a latah," one day was carrying his child in his arms; one of the crew stood before him with a log of wood in his arms, which he nursed in the same way as "the latah" was nursing his baby. The sailor amused himself by throwing the log of wood on an awning and allowing it to roll down, catching it in his arms. The cook repeated every motion with his child, and, when the sailor allowed the log of wood to fall upon the deck, he let the baby fall and killed him on the spot.

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[1] Journal of Nervous and Mental Diseases, vol. vii., p. 487.

[2] New York Medical Journal, February 16, 1884

[3] Neuroses from a demographic point of view. Journal of Nervous and Mental Diseases, June, 1891.

[4] Journal of the Straits Branch of the Royal Asiatic Society, June, 1883. O'Brien, who was not a medical man, defines latah as "including all persons of a peculiar nervous organization, who from their mental constitution seem absolutely subservient to another's will." I am indebted to Mr. Talcott Williams for the opportunity of seeing the above article.

In some of these cases there is a tendency to persistent and continuous ideas, and even a slight delusional insanity, with other forms of mental disturbance.

According to Rosse, the proper spelling of this name is "emeryaki."

Rosse[1] gives an interesting letter from Lieutenant Schuetze, United States Navy, who saw much of this disease during a prolonged stay on the Lena Delta in connection with the Jeanette search expedition. The symptoms of the disease, as portrayed by Lieutenant Schuetze, are singularly like those met with in the "jumpers" of Maine and Canada. He remarks that it is common to meet with persons afflicted with this disease in the streets and market-places of Yakutsk and other towns, and his belief is that the exciting causes of the disease are the extreme cold, often 87° below zero, the lonely life, and excessive vodka and tea-drinking.

The first case which he observed was a Russian exile, who held the position in Yakutsk of assistant to the Chief of Police. At an evening card party he was standing back of the chair of one of the players, a district judge, watching the game. At the suggestion of another guest, Lieutenant Schuetze threw a small pellet of bread at the patient, striking him lightly on his bald head, his back being turned away. "He at once threw up his hands, gave vent to a yell or shriek—a sort of a loud chatter—boxed the ears of the judge, and disturbed the game generally; at the same time he trembled violently. Of course, the judge indignantly inquired for the cause of the attack, and the victim replied by indignantly demanding why he, the judge, touched him on the back of the head, as he knew what effect it would have."

Lieutenant Schuetze had a servant, a Cossack, who suffered from this affection, and on one occasion he called the servant by name when he was leaning out of the window. Not hearing him, his master touched him on the shoulder, and he at once began to chatter and was on the point of jumping out of the window.

In other cases, simply pointing the finger at a person who was a victim of the disease would cause wild excitement and gesticulatory movements, as well as imitation of anything that was said, the sufferer continually begging to be let alone. Another patient would imitate anything that was done before her, and Lieutenant Schuetze was told that in such cases among the natives it would be dangerous to draw a knife across your throat as if you were cutting it, as they would repeat the operation on themselves in a serious manner. In all of the instances described by the writer as soon as the exciting cause was withdrawn the subject would, after an interval of two or three minutes, resume his ordinary manner.

Gilles de la Tourette, who quotes O'Brien's observations, described nine cases of this affection, all of which began in early life, and heredity existed in more than half of these.

The movements which occur in these cases may begin in the face or upper extremities, and soon after the lower extremities are affected. The movements are sudden and are not continuous, but are abrupt and shock-like, resembling electric chorea. The patient's body is usually bent forward, as in jumping, and he leaps upward. These jumping movements occur as the result of a command to jump, or from some sudden excitement, like striking the patient on the shoulder or clapping the hands. The movements generally represent an exaggeration of natural gestures, such as raising the brow, elevating the shoulders, or making a movement of deference. They are often so violent that the patient is brought into contact with hard objects near him, and he frequently injures himself. Voluntary efforts do not induce the bizarre movements. The patient can control his muscular movements so as to feed himself properly or to use his hands and limbs in various ways. In this respect the malady differs markedly from Huntington's chorea or chronic non-hereditary chorea.

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[1] Loc. cit.

Prognosis. The prognosis is unfavorable. Most of the cases described lasted throughout life. In a few the attack is temporary, and these cases are probably hysterical; but in other cases, especially those in which there is a hereditary taint, the affection lasts throughout life, although there may be remissions of months or years. Gray[1] says that he has seen several cases in which there was absolute intermission of the symptoms for many months, so that he was justified in believing that a cure had been effected.

Diagnosis. The affection resembles chronic adult chorea and electric chorea, but differs from these affections in the imitative tendencies, and in the fact that the movements are produced by a shock or a mandate from another person. In the treatment of these cases bromide of potassium, arsenic, and anti-spasmodics have been recommended.

POST-PARALYTIC OR POST-HEMIPLEGIC CHOREA. (Post-hemiplegic Mobile Spasm. Gowers.)

Many years ago Weir Mitchell called attention to certain choreiform movements which sometimes occurred in partially paralyzed limbs after an attack of hemiplegia. Charcot shortly afterward recognized the affection, and detailed the symptoms with his usual clearness. This condition never occurs in completely paralyzed limbs, but commonly makes its appearance at the period when the paralysis is disappearing. The choreiform movements become more established as power returns to the limbs, or they may appear suddenly. The movements are most marked in the fingers and toes, and become less as the shoulder and hip-joints are reached.

Clonic choreiform movements occur much more frequently in the arm than in the leg, and when they exist in both the former is always most affected. The muscles of the face are occasionally affected by the mobile spasms, causing distortion of the features on the paralyzed side when the patient laughs or cries. The movements in the upper extremities most commonly met with are inco-ordinate gyrations of the fingers and thumbs, flexion and extension of the wrist and elbow, and shrugging and other movements of the shoulder-joints. The interossei are particularly affected, and, consequently, the movements of the hand most generally observed consist of varying degrees of flexion and extension at the metacarpo-phalangeal articulations. The movements are disorderly and irregular, and may or may not continue during rest. They invariably cease during sleep. The movements are more athetoid than choreic in character, but are quicker than the movements of athetosis, and almost always occur principally on voluntary effort. Associated with the choreiform movements is always a considerable degree of inco-ordination in the manner in which muscular acts are performed. Post-paralytic chorea may be met with at any age, but is more frequent in the hemiplegias of children than in those of adults. (Fig. 62.)

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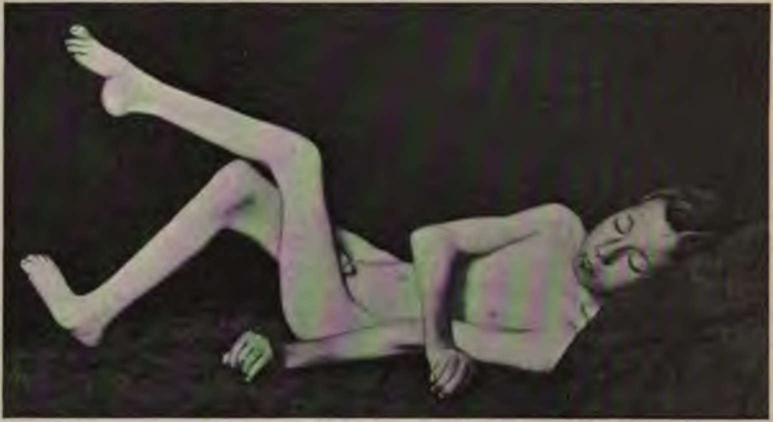
[1] Nervous and Mental Diseases, page 418.

FIG. 62.



Post-hemiplegic chorea. (Spastic diplegia.) Philadelphia Hospital.

Pre-hemiplegic chorea is occasionally met with preceding an attack of hemiplegia. The patient complains of numbness and feebleness of the extremities of one side, and the arm of the affected side is attacked with irregular choreic movements. These symptoms may continue for some days, and possibly months, as in the case of senile chorea above quoted, when complete hemiplegia, which is frequently associated with hemianæsthesia, is established.

ATHETOSIS.

This disease was first described by W. A. Hammond,[1] and named by him athetosis—'Αθετος, *without fixed position.* Considerable confusion exists as to the character of the affection, from the fact that it has been frequently confounded with post-hemiplegic chorea, which it closely resembles. It is an open question as to whether it is best to regard athetosis as a separate disease, for the symptoms which characterize it belong to many lesions of the brain, more especially those of the motor tract and caudate nucleus. As originally described by Hammond, athetosis was not associated with or preceded by paralysis of the affected parts; indeed, in the first cases described by him, he expressly notes the fact that the muscular power of the members affected with athetoid movements is not at all impaired. In the great majority of cases, however, there has been hemiplegia preceding the athetoid movements; and it is, therefore, best to divide the affection into a primary and secondary form.

Primary athetosis occurs without premonitory symptoms. In many instances no direct cause can be traced. In Hammond's classical case the patient was strongly addicted to alcohol; in other cases intemperance, exposure to severe cold, injuries, and fright have been assigned as causes.

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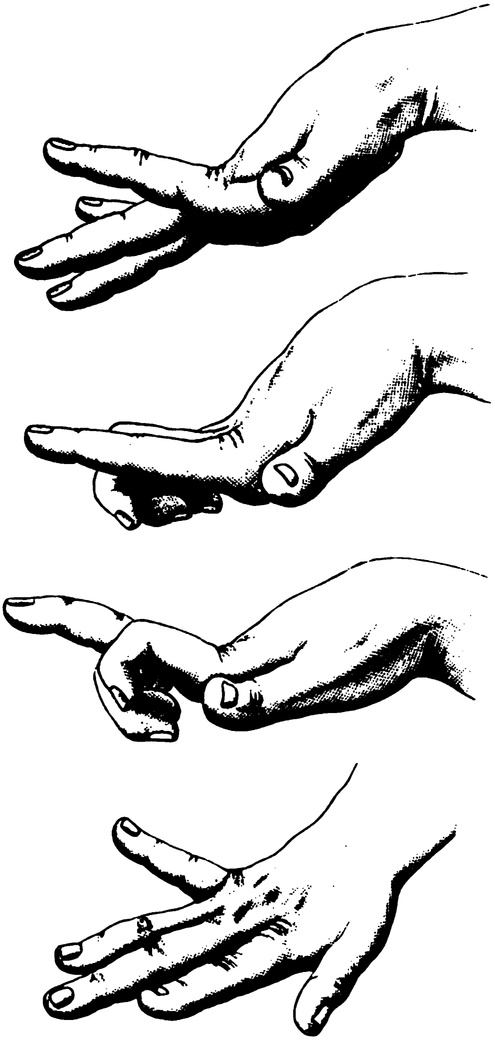
[1] Diseases of the Nervous System, 1871.

In the secondary form hemiplegia or diplegia precedes the irregular movements, and there is always some gross cerebral lesion, such as polioencephalitis (Strümpell), porencephalus, softening, or other lesion.

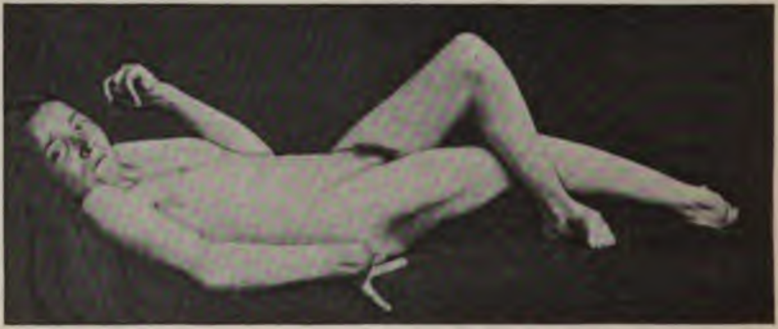
FIG. 63.



Examples of the position of the fingers in the movements of athetosis. (STRÜMPELL.)

Symptoms. The movements of athetosis are usually confined to the fingers and toes, but in most cases, especially in secondary athetosis, the whole arm is involved. The leg is seldom affected, but the feet and toes are occasionally the seat of the athetoid movements. The peculiar feature of the movements is that they take place while the part is at rest; that they are continuous, and that they are slow, differing in all these particulars from post-hemiplegic chorea. There is every variety and degree of movement met with. They begin usually in the fingers and hand, and, as the interossei are most frequently involved, the movements consist of flexion of the metacarpo-phalangeal articulations and extension of the fingers, with abduction and adduction. The spasm involves first one finger, then another, and sometimes all are in spasm at the same time. A favorite position of the hand is that in which all of the fingers are widely separated and the thumb is in extreme extension; or the forefinger alone may be extended and the others flexed. The accompanying drawings from Strümpell show more graphically than I can describe them some of the various positions in which the fingers are placed (Fig. 63). The whole arm may be contorted into many bizarre postures. Often the upper arm is abducted, while the elbow is flexed; occasionally the forearm is extended, and is in a position of extreme supination. In a young woman recently under my care the arm was rotated, the forearm pronated, and the whole arm carried behind the back, with the palm turned outward, this movement being followed by flexion of the forearm and supination of the wrist. In this case the head of the radius was almost dislocated by the extreme supination, and the patient usually sat with the hand of the affected side under the thigh, so as to control the movements.

FIG. 64.



Athetoid movements in a case of spastic diplegia. Philadelphia Hospital.

Subluxation of the distal phalangeal joints often occur as the result of long-continued overaction of the interossei, so that the head of the second phalanx is very prominent on the palmar surface of the finger. The continual muscular activity causes hypertrophy of the limb. In the patient just referred to, the left arm, which was the one affected, was much larger than the other, and the muscles stood out like a blacksmith's. Fatigue in the affected side is never complained of.

In athetosis the movements are always involuntary, slow, wavy, and extensive, and are generally rhythmical, occurring often in regular order. The movements proceed at all times; but they increase when the patient is aware of being observed, or when he attempts a voluntary movement. They are diminished when the patient's attention is distracted. They can occasionally be controlled by strong will power, but even then for only a short time. In one of Hammond's cases violent compression of the wrist controlled the movements. As a rule, the movements do not cease during sleep.

Bilateral athetosis is common, and is almost invariably the sequel of spastic diplegia. The illustration shows the typical athetoid movements in a case of spastic diplegia (Fig. 64). The patient was nineteen years of age at the time the photograph was taken, and the trouble was first noticed when she was five years of age.

The muscles of the face are occasionally affected with the athetoid spasm, and the tongue is at times involved to such an extent as to make speech inarticulate. Athetosis has been met with in cases of locomotor ataxia, and in these instances it is always bilateral.

Diagnosis. Athetosis is to be distinguished from chorea and post-hemiplegic spasm by the character of the movements and the parts involved. In chorea the movements are quick, jerky, irregular and misdirected; in athetosis the movements are slow, rhythmical and consecutive. In the majority of cases of athetosis, paralysis or imbecility exists. As already remarked, athetosis and post-hemiplegic chorea are confounded by many observers. The main points of distinction are that in post-paralytic chorea the movements occur on voluntary effort, are inco-ordinate and spastic, and are quicker than those of athetosis. In post-paralytic chorea they usually cease during sleep. The latter is usually accompanied by hemianæsthesia. Gowers says that this exists in fifty per cent. of the cases.

Pathology. The lesions producing athetosis depend on the nature of the case, and vary in extent and location in different cases. In Hammond's original case, in which it will be recalled that there was no paralysis, it was found after death that there was a degeneration consisting of fibrous connective tissue, which extended from the posterior third of the thalamus and posterior third of the internal capsule to the lenticular nucleus.[1] In this case the motor tract was not implicated. In many cases it will be found that the thalamus and posterior portion of the internal capsule are involved; but in other cases, in which there is an associated paralysis, the lesions must be disseminated, and include the cortex and motor tract. Cases have been recorded in which absolutely no lesion was visible in the brain to the naked eye.

In twelve autopsies collected by Kuhler and Pick the parts mainly involved were the thalamus, internal capsule, and lenticular nucleus. In one of Oulmont's cases[2] an area of softening was found in the lenticular nucleus and corpus striatum of one side. Lauenstein reported a case of primary athetosis in which the movements were confined to the fingers of the left hand, the athetoid movements having been observed but eleven days before death. At the autopsy a fresh lesion was discovered at the anterior end of the right thalamus. In a patient of my own, in which there was a tumor of the left thalamus,[3] in the early part of the case the movements of the right hand were inco-ordinate, and later paresis developed. At the autopsy the left thalamus was found to be occupied by a tumor as large as a hen's egg, which involved the corpus striatum and internal capsule to a slight extent, but the caudate nucleus was not affected. It seems, therefore, that athetoid movements are liable to occur as the result of an irritative process in any part of the pyramidal tract in its course where it ascends between the thalamus and internal capsule.

Athetosis is more likely to follow hemiplegia from cerebral softening due to the occlusion of a vessel than to apoplexy, and it is also much more frequent after infantile hemiplegias. Gowers[4] remarks that the significance of these facts are: First, that in softening slight damage to cerebral tissue is more extensive than actual destruction of tissue, and that the spont spasms are probably due to overaction of the gray matter which is in of altered function and nutrition. The significance of the secon namely, the frequency with which athetoid movements follow infantil plegia, is the greater facility with which growing and developing ner recover, and their greater susceptibility to disorder of nutrition whe development is interfered with.

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[1] G. M. Hammond: Trans. Amer. Neurological Association. 1891.
[2] Études Cliniques sur l'Athétose. Paris, 1878.
[3] University Medical Magazine, October, 1893.
[4] Diseases of the Nervous System, 2d edition, p. 88.

Prognosis The outlook in all cases of athetosis and post-paralytic is unfavorable. Occasionally a case of athetosis is met with which is tional, and this may recover.

Treatment There is practically no remedy for the conditions asso with athetosis. The use of galvanism and massage has proved of som efit in certain cases, but no permanent relief is afforded by these or remedies. In functional cases arsenic has been found of value, and in cases there has been undoubted benefit from the use of alteratives, su iodide of potassium and mercurials.

MYOCLONUS MULTIPLEX.

SYNONYMS. Paramyoclonus Multiplex; Convulsive Tremor; Myo

This rare disease is closely related to convulsive spasms and to chore is characterized by quick clonic spasmodic contractions of the muscles trunk and extremities. The spasms are bilateral, involving the symme muscles, and occur only at intervals. The affection was originally po out by Friedreich,[1] and since his paper several other writers have rec cases.

Many different forms of spasm have been described under the na myoclonus multiplex, but a large number of these are instances of hyst chorea or saltatoric spasm. In the typical form of the disease the pati rather abruptly attacked, the muscles of the trunk and hips are seized violent and rapid contractions, which may throw him down if standin hurl him from a chair or bed in which he may be sitting. The mu most involved are those of the trunk and upper part of the limbs, espec the deltoids, biceps, triceps, supinators, quadriceps extensor femoris, flexors of the leg, and the calf muscles. Sometimes the facial and muscles are affected, but those of the feet and hands are seldom implic The diaphragm is sometimes affected, producing hiccough, or a violent e ratory sound may be made, in the production of which the larynx also t part, resembling that made by a man when cutting wood. The spasm muscular contractions are bilateral, differing in this respect from ordi chorea. They begin in most instances in the shoulders and arms, and ext to the trunk. The tendon and skin reflexes are increased, but there ar disturbances of sensation, either anæsthesia or hyperæsthesia. These i mental disorder, but the subjects of this affection are usually neurasth and may be hysterical. The causes which have been met with are fri shocks, overmuscular exertion, and mental strain. The muscular sp occur at intervals, sometimes of only a few minutes, and sometimes an e week will elapse between paroxysms. The muscular movements are vic and rapid, varying from 5 to 180 per minute, but are commonly abou per minute. In a case related by Starr[2] the contractions were counte him, and reached 90 per minute. In this patient the disorder resulted a strain in lifting a heavy box, and the muscles first affected were those of the abdomen and back. There were short and quick alternate contractions of the dorsal muscles and recti abdominis. As these movements became more violent the body and head were thrown backward and forward until the patient became dizzy, and he would almost be pitched out of the chair in which he was sitting.

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[1] Virchow's Archiv., Bd. lxxxvi. p. 421.
[2] Familiar Forms of Nervous Diseases, p. 244.

I have seen one case of this affection, who was a patient of my colleague, Dr. James Hendry Lloyd, in the Philadelphia Hospital. In this case the muscles of the trunk and upper part of the limbs were affected with violent clonic spasms which were almost continuous. During the attacks the patient would be almost thrown from his bed. He was bathed in perspiration, and sharp expiratory sounds were made by the spasms of the diaphragm. His speech was interfered with, and the words were uttered in a short and explosive manner. The patient's mental condition was not markedly impaired. The patient died from exhaustion after five or six years. At the autopsy no naked-eye lesions were discovered in the brain or cord.

In the majority of cases tapping the tendons or irritating the skin will bring on an attack. In Lloyd's case an attack was brought on when he tried to speak, and any emotion exaggerated the movements excessively. In some cases voluntary effort has arrested the spasm.

This affection has been recorded by many as merely a form of chorea, and others regard it as hysterical. Gowers thinks that it is most closely allied to senile chorea. It differs from this disease, however, in that it is usually bilateral, and the movements are far more violent and are paroxysmal. Age has little to do with the development of the malady; but it is far more frequent in males than in females. But two cases in females have been recorded, and this is an additional fact against the hysterical origin of the disease. It is a variety of chorea, functional in character, and is more nearly allied to chronic adult chorea than any other form.

The pathology is unknown. In a case which died of phthisis and was examined by Professor Schultze[1] no lesion was revealed; and in Lloyd's case, which I have just quoted, the result of the autopsy was negative.

Prognosis. The prognosis is usually favorable. In many cases the disease lasts but a few months, but in some instances it is fatal.

Treatment. Galvanism and static electricity have been found most useful in the treatment of this affection; the latter has been especially recommended. When galvanism is used strong currents to the spine should be applied, and the anode should be placed over painful points on the spine, should they exist. Antispasmodics and nervines are occasionally useful, but attention to the general health and the administration of iron and arsenic is always likely to prove useful. In severe cases it may be necessary to administer morphine hypodermically.

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[1] Starr: Op. cit.

CHAPTER VIII.

LOCAL SPASMS. OCCUPATION SPASMS.

BY C. W. BURR, M.D.

FACIAL SPASM. MIMIC SPASM.

Etiology. In many cases no cause can be traced. It is mainly met with in adults, and is far more frequent in women than in men. Anæmia, general ill-health, grief, and worry predispose to it. It is alleged to arise sometimes from peripheral irritation acting reflexly; for example, a decayed and painful tooth. In many cases, however, removal of the supposed reflex cause will not cure the affection. It occasionally is associated with pregnancy. Cold is sometimes causative. It may result from the extension of the spasm in torticollis. Besides these cases that we are compelled to call idiopathic, it may be caused by organic disease of the nervous system. The lesion then is situated in the motor cortex, or in or near the nucleus of origin of the nerve. Thus Berkeley reports a case of cortical tumor, Moos a tumor pressing on the nerve at the base of the brain, and Gowers an aneurism of the vertebral artery.

Symptoms. The onset of the disease is usually gradual, and at first only one or two muscles—the orbicularis palpebrarum most frequently—are affected. Thence the spasm spreads until the patient has lost all control of the muscles of one side of the face. Not infrequently there is slight twitching on the opposite side, but serious involvement of both sides is rare. The spasm is both clonic and tonic—either frequent momentary contractions like those from an electric shock, or irregularly recurring single contractions. More frequently there are short paroxysms of combined tonic and clonic spasm. Motion and emotion, speaking, eating, or excitement of any kind will often precipitate an attack. The paroxysms vary in frequency from two or three in a day up to thirty or forty in an hour. The muscles most affected are the orbicularis palpebrarum, the levator superioris alæque nasi, the zygomatici, and the corrugator supercilii. The frontalis and the platysma are rarely, and the muscles of the ear very rarely attacked. The stylohyoid, the digastric, and the velum palati are almost never affected. The muscles of mastication are, of course, affected only if the motor portion of the trigeminus is involved by the disease. The spasm decreases greatly or ceases during sleep. In true facial spasm there is no pain save perhaps the fatigue ache of muscular overaction. Usually there is no paralysis, but it must be remembered that spasm may develop in a case of long-standing Bell's palsy. There may be an increase in the electrical irritability of the nerve, but no other change. Gowers notes the loss of the sense of taste on the front of the tongue, and in one of his cases of bilateral spasm there was a temporary increase in the secretion of the saliva. Occasionally the spasm extends far beyond the region of the facial nerve, involving successively the neck, shoulder, and arm muscles. There may or may not be tender points in the course

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We know nothing of the morbid anatomy and pathology of the disease beyond the few facts given under etiology—namely, that a chronic irritative lesion in the facial cortical centre, in the nucleus, in the pons, or of the nerve itself, may cause the disease.

Diagnosis is usually easy. The most important point is the differentiation from the secondary spasm occurring after facial paralysis. In this condition there is always a persistent contracture and weakness along with the occasional spasm. If, in chorea, the face is much involved and the extremities but little, mistakes may arise. In habit chorea the character of the movements is different. The diagnosis of the seat of lesion is difficult. The more limited the spasm and the more confined to muscles accustomed physiologically to act together, the greater the probability that the lesion is cortical. Increasing weakness and the addition of other symptoms point to a progressive organic cause.

Prognosis. The duration of the disease is long, often till death. Occasional remissions and intermissions are not rare. In cases that have lasted more than a few months the chance of complete and permanent recovery is slight. The danger to life is only of importance when the cause is organic and progressive, not from the spasm itself.

Treatment. In recent cases hot applications to the face and the production of free diaphoresis should be employed. In chronic cases many medicines have been used, none of which are of any great value or to be greatly depended upon in any given case. The general health should be looked after, and all possible sources of peripheral irritation removed. Sedatives, for example, gelsemium and conium, are much used, and sometimes in ascending doses do good. Morphine alone, or in combination with atropine, given hypodermatically, often stops the spasm entirely, but the very grave risk run of establishing the morphine habit must never be lost sight of. Faradism is always harmful. A weak, uninterrupted galvanic current, the anode placed in front of the ear and the cathode on the muscles, exerts sometimes a good influence. Mitchell recommends freezing the cheek daily with a rhigolene spray. Operative measures usually cause the spasm to cease for a time, only to return after a longer or shorter time. In a collection of twenty cases made by Keen, in only two did the spasm remain absent more than six months. For details of operation see chapter on Surgery. On the whole, treatment is thoroughly unsatisfactory.

Blepharospasm is a tonic or clonic paroxysmal spasm of the orbicularis palpebrarum, completely closing the lids. It is not infrequent in painful diseases of the eye, and may be due to refractive errors. The clonic form is called nictitating spasm, and is frequent in habit chorea and hysteria. A paroxysm of tonic spasm may be caused by voluntary firm closure of the eyelids, eye strain, or a bright light. In children a curious condition occurs, first noted by von Graefe, in which for weeks at a time there is a persistent lid cramp, and when the eyes are finally opened there may be temporary blindness, without visible lesion or permanent blindness, with gross ophthalmoscopic changes. In the common form the spasm can often be relieved by pressure over certain points, especially the supraorbital foramen. The position of the points is variable, and they should be sought for throughout the distribution of the trifacial nerve.

MASTICATORY SPASM. (Romberg.)

Masticatory spasm is an affection of the muscles supplied by the fifth nerve, the temporals, masseters, and pterygoids. It may be tonic (trismus) or clonic. In the former the teeth are firmly pressed against each other, so that the jaws cannot be separated, and the muscles of mastication are strongly contracted and as hard as wood. Attempts at forcible movement of the lower jaw cause pain, which may also result from the intensity of the spasm itself. The condition is almost always bilateral, but may be confined to one side. It is the beginning symptom in tetanus. As a manifestation of hysteria it is quite common. It may also be caused by peripheral irritation from injury as shown by Romberg, and from irritation of the fifth nerve caused by decayed teeth. Cases due to organic disease have been reported. Marot and Wernicke have seen it occur in tumor of the pons, and Gowers had a case which he thinks was due to disease of the basilar artery. It may occur during an attack of facial neuralgia. Very rarely only single muscles are affected, as in a case of Leube's, in which the jaw was held laterally displaced for several days—probably on account of spasm of the pterygoids on one side.

The most common example of clonic spasm is that which occurs during a chill. It is a not infrequent beginning symptom of a general convulsion. Hirt speaks of an old gentleman who for several hours daily would go through the movements of chewing, sometimes even unintentionally biting off the end of his cigar. Romberg relates the case of an old lady whose teeth chattered constantly, ceasing only during sleep and mastication. In the later stages of paralysis agitans tremor of the lower jaw may be sufficiently extreme to be called spasm.

In another form of the condition there are single and sudden contractions of the muscles, severe enough, it may be, to bite the tongue or cheek. It is not infrequent in hysteria, and may occur in anyone with an enfeebled nervous constitution.

The diagnosis of masticatory spasm is easy. The only condition with which it can be confounded is anchylosis of the jaw. The history of the case, and the fact that spasm relaxes under ether are sufficient to differentiate it.

The prognosis is good, except in the rare cases in which it is due to organic disease.

NODDING SPASM.

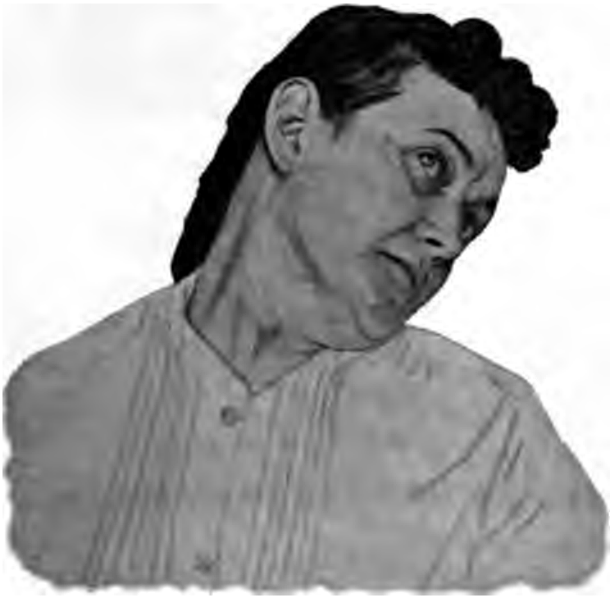
Not very rarely in improperly fed and rickety children there occurs a condition of clonic spasm of the sterno-cleido mastoids and adjacent muscles, causing nodding and rotatory movements of the head. Nystagmus, and more rarely strabismus, may be present. The condition in older children may be merely a trick, a form of habit spasm. See p. 258. If, as sometimes happens, instead of there being constant movements there are occasionally one or two movements, accompanied by momentary unconsciousness, we have to do with a much more serious matter—namely, an attack of *petit mal.* See Chapter XI.

SPASMODIC WRY-NECK (Spinal Accessory Spasm; Tic Rotatoire. Nickkrampf).

Under this head are included all types of spasm occurring in the muscles of the neck, whether they be supplied by the spinal accessory nerve or not.

Etiology. There is never, or almost never, direct inheritance, but epilepsy and other functional nervous diseases in the parents create a predisposition toward it. It is a disease of middle adult life, though rare and usually aberrant cases have been reported in infants. It is slightly more frequent in women than in men. Exposure to cold seems sometimes to be an exciting cause. Not infrequently patients give a history of exposure, pain and stiffness in the neck, and later spasm. The opinion that palsy of the muscles of the opposite side produces spasm from unopposed muscular action has little grounds for belief. A case of Annandale's, due apparently to excessive use of the muscles, ought probably to be placed rather among the occupation spasms. It was a young girl, a weaver, whose work required her to turn her head frequently from side to side. When any attempt was made to turn the head away from the left clonic spasm developed. Simon records a curious case in which malarial poison seemed to be the cause. The attacks were periodic, and were cured by quinine.

FIG. 65.



Spasmodic torticollis.

Symptoms. In rare cases the onset is very acute. Ordinarily it is slow, the spasm coming on only occasionally, affecting but one or a few muscles, and occurring only under excitement. As time passes the spasm becomes more severe, involves more muscles and endures longer, until finally it is constant. Though spasm is usually the first symptom, it may be preceded by dull, aching, or even sharp pain in the muscles or in particular points in the occiput, behind the ear, the cervical spine, or even in the arm.

The spasm may be tonic or clonic, or, as is most frequent, mixed. The position of the head and the direction of movement depend upon the muscle or muscles affected. Usually several are involved. If only one muscle be affected, it is usually the sterno-cleido mastoid. In Gowers's series of thirty cases it happened thus in seven. In this condition the chin is turned toward the opposite side and tilted up, while the ear is brought toward the clavicle. Tonic spasm fixes the head in this position. If both sterno-cleido-mastoids be affected the head is drawn first to one and then to the other side, or, if the spasm be tonic and equal on both sides, strongly forward and bent upon the chest. Spasm of the trapezius draws the head back and toward the diseased side, at the same time elevating the shoulder and bringing the scapula nearer to the spine. In cases of posterior bilateral spasm (retrocollic spasm) the head is drawn backward, usually moderately only, but sometimes so strongly as to make the face look directly upward.

The intensity of the spasm varies from time to time, and is more or less increased by emotional excitement. It can often be controlled by the will for a few moments, but soon the imperative need to move becomes so great it cannot be resisted and the spasm returns—at first more violently than before.

Not very infrequently, as time passes, the spasm extends to the muscles of the arm. In such cases there is almost always involvement of both sides of the neck. In one case of Gowers's only the deltoid was affected. Again, the spasm may begin in the arm and extend to the neck muscles. There may also be involvement of the face, twitching of the eyelids or of the muscles of mastication. If the spasm be forcibly stopped it may appear temporarily in other muscles.

There is always more or less discomfort and often dull, aching pain, which, though not acute, may cause great suffering. The overacting muscles always hypertrophy. Electrical irritability is either normal or increased.

Morbid Anatomy. In many cases nothing has been found post-mortem. In some, tumors of the brain, meningitis, cervical caries, and tumors of the medulla have been present. The seat of lesion must be either in the cortex or the pons. It cannot be a disease of the muscles themselves. Further than this we cannot go.

Prognosis as to life is good, unless it be secondary to some organic disease. As to recovery the outlook is not so good. While the tendency is to increase until several muscles are affected, it may remain slight and confined to one muscle. There may be intermissions, even without treatment, lasting a shorter or a longer time. Of course, the more severe the spasm, and the greater the number of muscles affected, the less the chance of recovery.

Treatment. Morphine, chloral, cannabis indica, conium, gelsemium, the bromides all do good for the time at least, though their discontinuance is apt to be followed by a return or increase of the spasm. Morphine is the most useful, but the danger of forming the habit is in no disease greater, and in few so great. Gelsemium, in the form of the fluid extract, is of distinct value in many cases. Electricity, though much lauded, seldom has any permanent influence. Instruments intended to forcibly check the spasm cannot be worn for any length of time. Many times the tendons of the affected muscles have been cut, but with no good result. Nor could good result, since section of a tendon cannot prevent the contraction of a muscle. If the spasm be confined to the distribution of the spinal accessory nerve, stretching it will arrest the spasm for a time at least, and cutting it cause arrest until reunion takes place, which, unfortunately, is too apt to occur, even though quite a large section be removed. This operation is, however, sometimes followed by complete recovery. Keen has shown that, even if other muscles be affected, the spasm in them is greatly reduced, or made to disappear, by subsequent section of the posterior branches of the two or three upper cervical nerves. (See chapter on Surgery.)

ŒSOPHAGEAL SPASM.

Spasmodic dysphagia is not uncommon in hysteria; it occasionally follows dyspeptic symptoms, long-continued vomiting, or irritation from hot or irritating foods. It is alleged to sometimes arise reflexly from uterine disorders. As an independent affection it is rarely observed. It is characterized by periodic or permanent painless difficulty, or even inability in swallowing. The food, after reaching a certain point, is regurgitated. On passing a probang it is stopped at a certain point. The diagnosis is from organic stricture. Under ether, the spasm relaxes and the sound passes without difficulty. Mistakes may, however, be made. In one case of supposed œsophageal spasm known to the writer, on post-mortem examination myelomalacia of the gullet was found, and death was due to rupture, not, however, caused by examination.

RECTAL SPASM.

Occasionally in association with vaginismus, spasmodic dysmenorrhœa, or from pressure of a misplaced uterus, there is spasm of the rectum and sphincter, producing the flattened stools and other symptoms found in organic stricture.

Urethral spasm may be caused by irritant substances in the urine, as, for example, cantharides, by strictly surgical causes, or it may be hysterical. It is characterized by sudden inability to micturate or a great lessening in the size of the stream, with great pain and straining, often accompanied by a feeling of weight in the perineum.

SPASM OF THE TONGUE. (Aphthongia.)

Berger, Dochmann, Hirt, and others, have reported a very curious condition in which, paroxysmally, the tongue is protruded and retracted violently, rolled roughly around in the mouth, and pressed so firmly against the teeth that it may be injured. In some instances there occur short rhythmical twitchings of the whole organ. In a case of Berger's there was a preceding attack in which the tongue felt thick and swollen. In Hirt's case the muscles of mastication were involved. As an accompaniment of hysteria the condition is not rare.

PHANTOM TUMOR.

Most frequently this condition is found in the abdomen, and is probably produced by relaxation of the rectus and spasmodic contraction of the diaphragm. Ordinarily there is local bulging of the abdominal wall near the middle line and below the umbilicus, simulating a solid mass. There may, however, be enlargement of the entire abdomen. The intestines, inflated with gas, are pushed forward. The local swelling simulates a true abdominal tumor not only in appearance and feeling, but also by sometimes giving a dull note on percussion. The spasm, and with it the tumor, disappears under ether. The condition is always a manifestation of hysteria. Usually there is great abdominal pain. In some cases it appears and disappears several times in the day, and may be brought back by the slightest handling of the belly. It is chiefly of importance because the mimicry of organic disease has sometimes been so close as to lead to operation.

Much more rare are phantom tumors occurring in other parts of the body. Weir Mitchell describes a hard, dense " tumor " in or on the left great pectoral muscle above the breast. It was an oval, flattened swelling with quite abrupt edges. Hard rubbing gradually dispersed it, only to form again in a few hours. In another case there was a similar " tumor " situated on the calf. There is probably a close relation between certain phantom tumors occurring over muscular masses and angio-neurotic œdema.

TRIGEMINAL COUGH.

Schadewald described under this title an affection characterized by paroxysmal cough, occurring in persons whose respiratory apparatus was in normal condition, and due to trigeminal irritation in the nose, pharynx, or external auditory meatus. Schadewald regards it as a reflex neurosis. Hack believes that the erectile tissue of the nose is responsible.

OCCUPATION SPASMS.

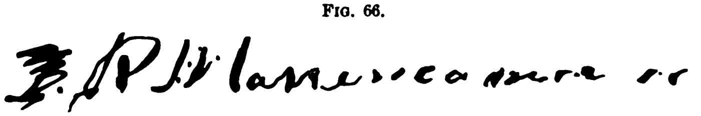
In certain occupations requiring the constant repetition of particular movements, usually complicated and fine, but sometimes simple and coarse, there often arises a condition in which the person, while well able to perform all other movements, cannot make the special movements required by his work. This condition is called an occupation neurosis or occupation spasm. Neither designation is exactly true, for the first assumes that there is no organic causative lesion, while probably such lesion exists, but is too delicate to be shown by our present means of examination, and the second misleads, since in many cases there is as much paralysis as spasm. The most important example of the disease and one that may be taken as a type of all the others is writers' cramp. Therefore we shall study it first and most carefully.

WRITERS' CRAMP (Scriveners' palsy, graphospasmus, cheirospasmus, mogigraphia) was first described by Sir Charles Bell in 1830.

Etiology. The disease is most apt to occur in the neurotic. Hence, though it is not, properly speaking, ever inherited, the tendency to it may be, and a pseudo-inheritance result. Thus Gowers speaks of a lady whose father also suffered, and Gallard saw it occur in mother, son, and daughter. Vance had a patient who, on going to visit a distant brother, was surprised to find him a co-sufferer. Its occurrence in groups is in some cases at least certainly due to unconscious mimicry. Everyone who has much writing to do has at times twinges of pain and much aching, and in certain dispositions it needs but little fire to make a great smoke. Indirect inheritance through epileptic or insane parents is a quite strong factor. Prolonged or sudden grief and sheer stress of life is the greatest predisposing cause. For example, a man may for years write many hours daily without discomfort, but let him become involved in financial or domestic difficulty, and the first signs of breakdown may be writers' cramp. A lady played upon the piano many hours daily for years without any trouble, but her husband failing in business she became a music teacher, and almost immediately piano-players' cramp developed. She has never recovered. Venereal and alcoholic excess predispose to the disease. Local injury in the hand or arm may precipitate an attack, as in an army officer mentioned by Gowers, who, having sprained his thumb, was compelled to write a great deal before it had recovered, and soon characteristic pain and cramp appeared. Sleeves too tight around the wrist, and even the pressure of a large sleeve-button have been blamed. Runge reports a case in which periostitis of the humerus was the cause. Vance saw it in slight cases of hemiplegia, and in one case of not severe brachial monoplegia, it was the only symptom left. In the last there was absolutely no palsy and no interference with any other movements, no matter how fine, except those of writing, any attempt at which would cause spasm. There was no agraphia. Rarely a distinct neuritis of the median, radial, or ulnar nerve, or of the brachial plexus has seemed to be the cause. But it is probable that in most cases the nerve tenderness, which is common, is neuralgic; is a part of the affection and not its cause. It is far more common in men than in women. It occurs most frequently between the twentieth and the fiftieth year. Curiously, it is never found in children learning to write, nor do the other trade spasms occur in beginners, but only in those who have worked some time. The great cause is an improper method of writing. The way the pen is held is of much less importance than the muscles brought into action in the movements of writing. According to Gowers the worst method is that in which the little finger is used as a fixed support. The use of the wrist as a fixed point is better, but still bad. In both these methods small muscles have all the work thrown on them, and consequently fatigue comes on sooner. Undoubtedly the only proper way of writing is by using the upper arm and shoulder muscles, allowing the forearm, wrist, and little finger to rest upon the table to take off the weight of the limb, but making them move along from left to right as the writing progresses. The advantage of a free hand is shown by the fact that stenographers who are bound to adopt it to attain speed seldom if ever suffer. Steel pens have been blamed, but scarcely justly, since the disease was known before their invention. Finally, it must not be forgotten that the disease may occur in those who write but little, being then a local manifestation of a general neurasthenia.

Symptoms. Moritz Benedict described three types—the paralytic, the spasmodic, and the tremulous. They are rarely absolutely separate, two being usually present, with one predominating. Spasm, accompanied by pain is the most frequent. In rare cases the onset is sudden. Ordinarily it is slow. Usually at the beginning there is fatigue, pain in the fingers, and dull aching in the metacarpal and wrist joints. There is often formication and numbness, but no true hyperæsthesia or anæsthesia. The nerve trunks may be slightly tender. At first all discomfort passes away soon after the patient ceases to write, and does not return until he has written again for a longer or shorter time. Later the sense of fatigue becomes permanent, extends up the arm, and may reach as far as the scapula. Soon, and in some cases without preceding pain, motor troubles, spasmodic or paralytic, or both, appear. The patient notices he cannot guide the pen, it digs into the paper, the strokes are irregular and go too far up or too low down. The pen is grasped too tightly and the patient cannot let go. The writing "resembles that done in a jolting carriage." (See Fig. 66.) The cramp is usually tonic, not clonic.

FIG. 66.



Handwriting in writers' cramp.

There may be flexor spasms of the thumb, forcing it into the palm. Again there may be flexor or extensor spasm of the index finger, or of it and the third finger. If there is extensor spasm of the thumb and index finger the penholder will fall from the hand. If the flexor and extensor carpiulnaris be affected the pen is forced strongly against and digs into the paper. There may be spasm of the pronators and supinators of the forearm, in which event the pen goes hither and thither over the paper, and the markings resemble rather chicken-tracks than writing. Rarely there may be an accompanying spasm of the shoulder muscles. Clonic spasm occurring only on beginning to write and affecting the thumb and index finger occurs infrequently. It should be possible to tell from the handwriting the muscles affected in the disease and whether by spasm or palsy.

Tremor almost never occurs alone. It is usually accompanied by tonic spasm. At the beginning it occurs only when writing and gives the handwriting a "shaky" form. Later it may continue while the patient is at rest or be caused by any movement, and then is apt to invade the left hand also. Ordinarily the tremor is confined to the thumb and first finger. A purely paralytic form of the disease rarely if ever occurs. There is always some spasm, but weakness may predominate. There is fatigue pain in the affected muscles and some difficulty in the voluntary contraction of them.

It is rare for coarse movements to be affected, and fine movements, if they differ in character from those made in writing, may be uninfluenced. Some one other complicated movement, may be involved as in a patient of Gowers's who could do anything except shave. There may be a certain amount of weakness in particular muscles as shown by Piore, but usually the grasp of the hand is good. Slight local muscular wasting sometimes occurs.

There may be no change in the electrical reactions of either nerves or muscles. In long-standing cases there may be increase or decrease to both the faradic and galvanic currents. Eulenburg found qualitative changes in the nerve trunks.

There is apt to be a general condition of neurasthenia or mild hysteria. Various paræsthesiæ are complained of. Painful points in the course of the nerves of the arm are common, and the nerves may be swollen. Not infrequently there are tender spots in the spine, especially in the cervical region. There may be a transitory marble pallor of the fingers from vasomotor constriction. Stammering is an occasional complication. If the patient persists in writing, the condition gradually becomes worse, until finally the act is impossible. At the beginning it is often possible to write well with a pencil. Patients often use the left hand in writing. In such cases the chances are about equal that it will escape.

Pathology. There are no known anatomical changes in the disease, but it is highly probable that if we could use the same methods as Hodge used in his experimental study on the effects of fatigue we could find distinct, though fine, lesions in the motor brain cortex, or in it and the cells of the cervical enlargement of the cord. Three theories have been offered to explain the disease. First, that it is local weakness of certain muscles, permitting overaction of their opponents; second, that it is reflex; and, third, that it is central—a want of proper balance in the co-action of the various motor centres concerned in the action of writing. The last is the most satisfactory.

Diagnosis of the fully developed disease is easy. The beginning stage may be mistaken for neuritis, a slowly on-coming hemiplegia, or even paralysis agitans, but only if the possibility of the existence of writers' cramp be overlooked.

Prognosis in a bad case, or one of long standing, is never very good. It is better in those cases in which outside elements—ill health and worry—have a large causative influence than in those due solely to excessive writing. It is better in cases in which there is a large pain element than in those which are purely motor. Finally, the worst case may recover, while one apparently much less severe may persist.

Treatment. The best treatment is avoidance of the disease by adopting a proper method of writing. The patient must cease absolutely to write with his right hand. No mechanical appliance will do more than stave off the increase of symptoms for a time. There is no reason why he should not use the left hand in writing, since, if it is attacked he is no worse off than before. Galvanism sometimes does good, and should be used, but faradism may be distinctly harmful to the too irritable muscles. Massage will often relieve the pain, and has a good effect on the disease itself. Local hand-gymnastics, the fingers being made to perform movements as much unlike writing as possible, are excellent. Internal medication is of no benefit, except as regards the general health.

OTHER OCCUPATION SPASMS.

There is a long catalogue of occupations which may produce conditions exactly parallel in etiology, pathology, prognosis, and treatment, to writers' cramp, the only difference being the character of the movements affected.

The following are the most important:

SEAMSTRESSES' CRAMP. Any occupation requiring constant sewing may lead to a condition of rigid flexion of the fingers used. Similar spasmodic conditions occur in the lower extremities in ballet dancers and operators on the sewing machine.

Spasm of the tongue has been met with in players on the clarionet.

Burns gives a long list of other occupations—drummers, money counters, masons, compositors, engravers, cigarette makers, enamellers, artificial-flower makers, painters, letter-sorters, turners, and knitters. Eichhorst speaks of the case of a girl whose daily work was to fasten 4000 needles in papers.

SMITHS' CRAMP, or hammer palsy, is a good example of the disease as caused by coarse and simple movements. The spasm may cause the hammer to fall from the hand, or there may be a loss of control, causing unexpected movements of the arm. In one case, the hand holding the chisel would be unexpectedly supinated. I saw, quite recently, in Dr. Morris J. Lewis's clinic, a car driver whose work required that he should press the brake handle with the palm of the right hand continuously. Some months previously he noticed slight trouble in grasping the brake with the fingers, extension of the arm occurring with every attempt. Finally, the spasm and pain became so great that he was unfit for work. There was also occasional clonic flexor spasm.

TELEGRAPHERS' CRAMP. This occurs only in operators who use the Morse machine, in which there is repeated flexion and extension of the finger pressing and releasing a key in order to make dots and dashes. The constant repetition of this movement many thousands of times daily produces a spasm of the finger, which prevents it from being raised quickly enough from the key, and hence dots become dashes. Pain may accompany the spasm.

PIANO-PLAYERS' CRAMP occurs more frequently in women than in men. It may be either spasmodic or neuralgic. It usually begins in one finger of the right hand, and is characterized by spasmodic extension of the finger, which prevents the key being struck at the right time, or the spasm holds the finger fixed upon the key. There may be pain alone, without spasm, so severe as to compel the player to stop playing, and occurring in the hand only or extending up the entire arm. Players on the organ, the zither, or the harp may be similarly affected. In violin players the spasm may attack either the hand holding the bow or the one controlling the strings.

CHAPTER IX.

FUNCTIONAL TREMORS.

By LANDON CARTER GRAY, M.D.

Definition. Under the term functional tremor are embraced those various forms of tremulousness which are not of the type of disseminated sclerosis or paralysis agitans.

Clinical History. At the outset of this subject it should be understood that a distinction must be made between tremor and the fine muscular movements which are seen in chorea and the so-called[1] convulsive tics. In the latter the movements are either fibrillary or extend over a whole muscle or set of muscles, and they consist of abrupt or more or less abrupt contractions, succeeded by abrupt or more or less abrupt relaxations, and the intervals between each muscular contraction are distinctly perceptible or prolonged. In the former, however, the movements are chiefly, indeed almost entirely, fibrillary, and the muscular contractions are minute in range, succeed each other rapidly, and are rhythmical in character. They may be divided into four great classes, accordingly as they are fine, coarse, intermittent, or continuous. Based upon the dogmatic teachings of Charcot, the belief was entertained for many years that the tremor of paralysis agitans was always continuous, and that of disseminated sclerosis always of the so-called intentional type, *i. e.*, only present when a voluntary movement was made (see also p. 27); and, proceeding upon the same line of thought, it has been supposed that the same distinction would be found in the tremors that were not due to paralysis agitans or disseminated sclerosis. Of late years, however, Charcot's dictum has been regarded with more and more caution by neurologists, until I think I am safe in stating that nowadays it is generally believed that the tremor of disseminated sclerosis is mainly, if not entirely, of the intentional type in the earlier stages; that that of paralysis agitans is mainly of the continuous form, although there are many exceptions to this rule; whilst in the so-called functional tremors no absolute rule has as yet been established. Tremor has thus fared the fate of other symptoms of disease in having been shown to be unreliable as pathognomonic, as we have slowly plodded our way to recognition of the wider truth that disease evidences itself by a combination of symptoms and not by any one alone. Tremor may affect all the external muscles of the body or a group of them, either in one limb or in a segment of one limb. They may therefore be found throughout the head and neck, in the eye muscles (nystagmus), the facial muscles, the tongue, the lips, or the extremities. Tremor of the internal muscles, such as the diaphragm, or the more deeply seated of the trunk muscles, has been very scantily studied, and we know scarcely anything about it. When tremor is of the intentional type it will be evidenced by making some voluntary movement, as by having the patient approximate a glass of water to the lips, or write the name, or do some other voluntary act, although it is a curious fact that this intention tremor may be present in one voluntary act and absent in another. When the tremor is continuous, it will be present, as the term implies, when the patient is at rest, so far as making a voluntary act is concerned, although it should not be forgotten that these continuous tremors will be exaggerated by emotion or by mental or physical effort. The rate of tremors has been determined by the sphygmograph, the best of which is said by Peterson to be that of Edwards, although the European observers have made use of Marey's, and in Peterson's article will be found an excellent *résumé* of the sphygmographic studies that have been made. According to Peterson, the tremor of paralysis agitans varies from 3.7 to 5.6 per second, this variation being often observed in the same individual. In well-developed multiple sclerosis the latter observer found a rate of from 4.6 to 6.3 per second, although in the earlier stages it may run as high as 7.9 to 8.1 per second. Gowers, however, has found the rate in paralysis agitans to run between 4.8 and 7 per second. The following tables will show the variations in the different tremors by different observers:

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[1] For these convulsive tics I have suggested the name of Palmus.

Observer.	*Disease.*	*Rate per second.*
Peterson	Basedow's disease	8.7 to 12
	Hysterical tremor	7 6 " 7.8
	Alcoholism	8.5 " 11.2
	Delirium tremens	5.6 " 6.8
	Neurasthenia	7.4 or more
	Multiple sclerosis	4.6 to 6.3
	Lateral sclerosis	6
	Paralysis agitans	8.5 " 5.6
	Exophthalmic goitre	8.7 " 12
Wolfenden and Williams	Basedow's disease	10.8 to 11.5
	Hysteria	... ...
	Divers' paralysis	... ...
	Paralysis agitans	5 to 11
	Disseminated sclerosis	5 " 8
	Lateral sclerosis	5 " 5
	Exophthalmic goitre	10.8 " 11.5
	Multiple sclerosis	5 8
Dana	Neurasthenia	10
	Hereditary tremor	8 to 9
	Quinine	10
	Alcoholism	6.2
	Exophthalmic goitre	8 to 9
	Epilepsy	10
	Paralysis agitans	3 " 6.5
	Hysteria	5
	Senile tremor	4 " 5.5
	Multiple sclerosis	... ...
	Lateral sclerosis	6
	Post-hemiplegic sclerosis	10 to 12
	Myelitis	5 " 6
Dutil	Alcoholism	8 to 9
	Exophthalmic goitre	8 " 9.5
	Paralysis agitans	5 " 6
	General paresis	8 " 9.5
	Hysteria	5.5 " 7.5
	Senile tremor	4 " 5.5
	Multiple sclerosis	4 " 5.5
	Lateral sclerosis	... ...
Charcot, Dutil, and Marie	Exophthalmic goitre	8 to 9.5
Gowers	General paresis	10 " 12
	Paralysis agitans	4 " 8.7
	Multiple sclerosis	6 " 7
Féré	Epilepsy	7
Horsley	Lateral sclerosis	8 to 10
Charcot	Multiple sclerosis	7 to 10

The data for this table have been gathered by different sphygmographic instruments, about the reliability of which there has been considerable difference of opinion, so that the American observers have discarded entirely the Marey sphygmograph which was employed by the Europeans. The subject is therefore in need of investigation by some one standard instrument. As far as they go, however, these observations would seem to show that the rate of tremor of paralysis agitans is the lowest of all, although even this dictum is contradicted by Gowers; next in frequency is that of multiple sclerosis, although even in this Charcot found a rate from 7 to 10 per second; whilst the other tremors, although varying in some individual cases, as a rule range higher. Regarding the table still more critically it will be observed that the range of rate in the same disease varies according to the different observers, so as to leave us in very considerable doubt. Thus, in paralysis agitans, the tremor varied from that observed by Charcot, 3 per second, to that observed by Gowers, 7; in multiple sclerosis from 4 (Dutil) to 10 (Charcot); in alcoholism from 6.2 (Dana) to 9 (Charcot and Dutil); and in exophthalmic goitre from 8 (Charcot, Dutil, Marie) to 11.5 (Wolfenden and Williams). We cannot therefore base any diagnostic certainty upon the rate of tremor, and the sphygmographic tracings have only as yet been scientific curiosities.

The classification of tremors may be made as follows:

Paralysis agitans;
Disseminated sclerosis;
Tremor from lesion of the brain, spinal cord, or peripheral nerves;
Toxic tremors;
Tremors from neurosis;
Emotional tremor;
Febrile tremor;
Senile tremor;
Hereditary tremor;
Convulsive tremor.

Besides these there are certain muscular movements which, with a strange lack of precision, are often included under the head of tremor, such as the intermittent clonus of epilepsy, the fibrillary contractions of muscular atrophy, the purring tremor or purring thrill, the shaky movements of subsultus tendinum, and vibratile tremor or fremitus, so familiar to auscultators when an aneurism, a bronchial rale, a pulmonary cavity, an endocarditis, or some other disturbance of the thorax or abdomen is present.

The tremor of paralysis agitans and disseminated sclerosis are elsewhere treated, and need not detain us here. (See Chapters X. and XXII.)

Almost any lesion of the brain, spinal cord, or peripheral nerves may be accompanied by a tremor, and this may be present only in the parts affected by the nervous lesion, or may extend beyond them in varying degrees. Through a lamentable confusion of thought, under this heading have often been included, especially by the French writers, hemichorea and hemiathetosis, even by so competent a writer as Demange, as late as 1885; and this is the more inexplicable because the so-called hemichorea is really a hemiathetosis and consists of gradual worm-like muscular movements with long intervals of rest, resembling in no wise the minute, quickly-succeeding, partial fibrillary activity to which the name of tremor should really be given.

The toxic tremors are those caused by arsenic, mercury, lead, copper, and alcohol. Charcot, shortly before his death, began to teach very positively that mercurial tremor was always hysterical, and he based his conclusions upon the sudden recoveries and the frequent presence of his so-called hysterical stigmata, namely, concentric limitation of the field of vision, an emotional condition, and seemingly erratic impairments of sensation. Could we bring ourselves to believe that this were true of the tremor induced by mercury, the correlated conclusion would be that the tremor of alcoholism, lead, copper, neurasthenia, and febrile diseases were also hysterical. But when we remember that the so-called hysterical stigmata have been observed in very many diseases of the nervous system other than those of hysteria, as has been shown by the careful observations of Oppenheim and Thompson, it will be seen how unsupported by proof this conclusion of Charcot's is, whilst sudden recoveries under proper treatment occur in a thousand non-hysterical diseases.

The tremors that are observed in the course of the neuroses are not of special clinical value, except historically. What is generally called choreic tremor is really, as I have already said, not a tremor at all, but consists of fine fibrillary movements with distinct intervals, or in the more violent cases of muscular movements involving muscles or groups of muscles. In some very rare instances, however, a chorea will be accompanied by a genuine tremor. In epilepsy tremor is not usual, and when it is present it is observed only as the epileptic attack is passing off. Hysterical tremor is usually of great diagnostic importance, and it will often throw light upon a case that is otherwise obscure. I have, in many instances, from this symptom alone, made a tentative diagnosis of hysteria, which the further progress of the case has confirmed. In convulsive tic, or palmus, as I have called it, I have seen tremor in those cases which were accompanied by slight mental symptoms, but the disease is so recently a subject of observation that I am not able to state the frequency of this particular symptom.

Emotional tremor is too well known to need more than a mere mention, and the same is true of the febrile tremors.

There is a growing inclination at the present day among neurologists to consider senile tremor as a variety of paralysis agitans, and I am inclined to agree with this opinion, the more especially as the pathological findings which are dealt with at length in the chapter upon paralysis agitans itself lend credence to this view. At the same time there are some doubts in my mind as to whether there may not be a clinical distinction between senile tremor and paralysis agitans, especially in the early stages, and in the predominance in the former of tremor of the jaws and head.

There is a form of tremor which is hereditary, beginning in young adults, and frequently giving rise to a suspicion of disseminate sclerosis, and in later years often of that and also of paralysis agitans. It is easily distinguishable from both of these affections, however, by the fact that it has none of the classical symptoms of either of these two diseases, such as the nystagmus, characteristic speech, attitude, position of the hands, etc.

Dr. William A. Hammond, in his text-book on diseases of the nervous system, calls attention to a disease characterized by a paroxysmal tremor of short duration, which had previously been described by Tollmouche and Parkinson. The attacks are usually of great violence, accompanied by loss of consciousness and febrile rise, and succeeded by a feeling of great fatigue, but no sleep or stupor. The prognosis is said to be generally favorable. I have never seen a case of this kind, and cannot do more, therefore, than speak of it from the experience of others.

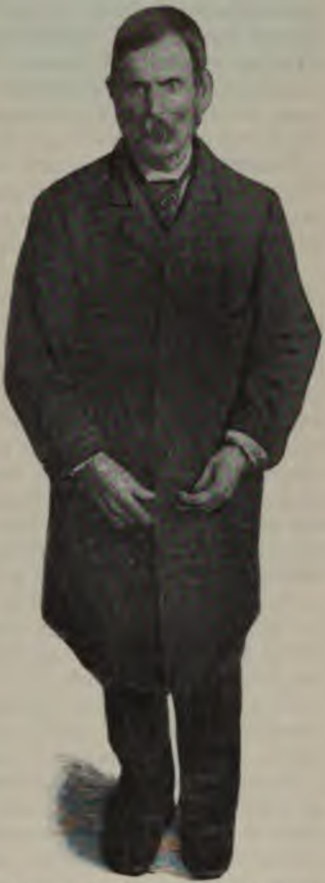
Pathology. The pathology of tremor, in the true sense of the term, is quite unknown. It is true that in the two great standard types of disease, paralysis agitans and disseminated sclerosis, in which tremor is the salient symptom, the chief pathological lesion would seem to be an alteration in the connective tissue of the brain and cord. That this cannot be the real cause of the tremor, however, has been demonstrated by those several cases of spinal cord lesion which are alluded to in the chapter upon paralysis agitans, and in which the connective tissue of other portions of the cords was found to be altered, without a sign of tremor. It would seem to be most reasonable to assume that tremor can only be produced by direct or indirect implication of the motor tract. These motor strands have a long textural journey to pursue. They start above in the clusters of motor cells in the motor convolutions of the cerebrum, and at this station they are connected by the so-called commissural fibres with many other areas of the brain. From these cells they go on down through the centrum ovale to converge into the internal capsule, thence passing to the crura, to the pons, to the medulla oblongata, where they decussate, some passing down through the lateral pyramidal columns of the cord, others through the anterior columns of the cord or the columns of Tuerck, both of these sets of fibres making their way to the cells of the anterior columns through fibres that have been demarcated of late years by the researches of Golgi, Ramon y Cajal, Lenhossek, and others; and from these cells of the anterior horns passing off into the anterior roots of the motor structures of the trunk, head, and periphery. It is easy to conceive that any direct irritation of this motor tract—as by the sclerotic areas of disseminated sclerosis in the cerebrum, the pigmentation and atrophy of the cells of the anterior cornua, the endarteritis of the spinal vessels, and the increase of connective tissue in the lateral and anterior pyramidal tracts of paralysis agitans—may well impair conduction between the cortical cells above and the peripheral structures beneath, so that the nerve current flows through imperfect media. But we do not know wherein consists the molecular disturbance of this motor tract producing tremor, and those other disturbances of the same motor tract causing epilepsy, chorea, and convulsive tic; and until we do know this, and until we are perfectly cognizant of the nature of these varying molecular conditions, we will not have a distinct idea of the genesis of tremor, or how it is produced by emotion, heredity, the neuroses and toxic agents. So far we have fallen into the mistake of attaching too much importance to those pathological findings which are evidently the result of the primary lesions inducing tremor, when in reality we know nothing of these primary causes themselves.

Diagnosis. A diagnosis of the nature of a tremor can usually be made with ease. A tremor is but a variety of involuntary muscular contraction. It is usually of four grades. First, violent muscular movements of large range, such as are seen in the so-called convulsive tremor; second, the violent convulsive movements of a large range alternating with fine muscular movements, constituting a true tremor, this combination being seen in those cases in which one or more limbs are affected, with recognizable organic disease of the cerebrum, as is evidenced by other symptoms of paralysis, contracture and anæsthesia; third, fine tremor, which is seen only when voluntary movements are made, and which is known as the *intention* tremor; fourth, the fine movements which are continuous, observed when the individual is at rest or making some movement. Of these four varieties the convulsive tremor is only mentioned here for the sake of completeness, as it may be regarded as a variety of epilepsy without loss of consciousness, or at all events being of a different type from the true tremor. The second variety is, as has been said, always indicative of some gross organic lesion in the cerebrum. Too much diagnostic importance should not be attached to the tremor being present on voluntary movement or continuous, for, as has already been said, whilst it is true that the tremor of disseminated sclerosis is usually of the intention type in the early stages, and that of paralysis agitans is generally continuous, there are yet many exceptions to these two rules, and the other tremors are sometimes continuous, sometimes intentional and sometimes continuous or passive at one stage of the disease, or *vice versa*.

When tremor is due to disseminated sclerosis the diagnosis can be made by the fact that the patient is usually a child or young adult, that there is nystagmus, and that there is none of the characteristic gait, attitude, position of the hands or flushing of the face of paralysis agitans, or the deliberate speech of that disease.

The diagnosis of paralysis agitans can be made by the fact that the patient is generally at or beyond middle age, and that there is a deliberate, slow, wise speech, that the body is bent forward and held stiffly, which, with the slow, shuffling gait and the bread-crumbling position of the hands (Figure 67), constitutes the peculiar attitude; that there is usually a dilatation of the facial capillaries, and often a tendency to go backward (retropulsion), to go sidewise (lateropulsion), or to walk faster and faster (festination). In these cases of paralysis agitans, too, it will sometimes be found that the patient will be unable to maintain the centre of gravity long, which may, in the slighter cases, be shown by the festination, by the tendency to fall forward after walking a while, or to fall backward in attempting to stand erect.

FIG. 67.



Photograph of a case of paralysis agitans, showing the attitnde, the position of the hands, and the *facies.*

Hysterical tremor can be diagnosed by the emotional or traumatic onset, the paraplegia, with anæsthesia; the monoplegia, with zones of anæsthesia, or complete; either the entire absence of muscular atrophy or the presence of hemianæsthesia, involving the sense of sight, smell, taste, the upper and lower limbs, or the concentric limitation of the field of vision (which may coexist with hemianæsthesia). Great stress has been laid in this diagnosis upon the ovarian tenderness, but this I have found to be too frequent in sensitive women to be of much diagnostic value. It must never be forgotten, however, that hysteria may exist without any of these so-called stigmata, and then the diagnosis may become a matter ot much difficulty, as it can only be made through careful exclusion by one who is at home in the wide realm of nervous and mental disease. Indeed, were I to judge by many personal experiences of my own in this matter, I should say that the bulk of hysterical cases in America were not accompanied by these so-called hysterical stigmata. It would seem probable that either too much stress has been laid upon these by the French writers, or that the Latin race is more prone to these symptoms than is that mixture of races which has gradually become fixed upon the soil of North America. Another, though occasional diagnostic factor of great moment which has not hitherto been alluded to by writers, is the bed-ridden condition; by which I mean to say that a patient who has been bed-ridden through a number of years, without appreciable cause that can be determined by a competent physician, is, in the vast number of cases, hysterical.

The diagnosis of the toxic tremors can be made only by means of exclusion and the knowledge or suspicion of the toxic agent having been used. Of late years so many precautions have been adopted in protecting artisans from poisoning by lead that the toxic tremor is not frequent, and very few cosmetics are now sold containing it. The history of colic, the blue line upon the gums, occasionally the history of some form of paralysis, especially that of the extensors of the wrist, or mental torpidity, would make the diagnosis easy. The tremor of mercury can readily be diagnosed if salivation has been present; but in many cases this is absent, and an opinion can then only be formed by a rigid history. The tremor of arsenic can usually only be determined positively by the history, although in two cases I have seen it accompanied by paralysis of the upper and lower limbs. The diagnosis of tremor having been caused by copper is dependent entirely upon the history. That tremor is due to tobacco may be surmised when the characteristic heart is observed, although usually the diagnosis of this variety will also be based upon the history. The tremor of the neuroses can readily be determined by the accompaniment of the characteristic symptoms of the neurosis itself.

Prognosis. The prognosis of tremor is good in the cases which are due to the neuroses and toxæmia, and unfavorable in those which are the accompaniments of organic, spinal, or cerebral disease. The hereditary form is often incurable, although frequently susceptible of great amelioration.

Treatment. The treatment of tremor should be addressed to the removal of the cause, if it is possible, and to the treatment of the tremor itself. Lead, arsenic, copper, mercury should be removed from the system in the well-known ways, which need not be detailed here. The tremor itself can be benefited by hyoscyamine, hyoscine, the bromides, oxide of zinc, galvanism, faradism, rest, and tonics. Hyoscyamine, in doses of $\frac{1}{100}$ grain once or twice daily, is an excellent remedy; but the objection to it is that it will dry the mouth and dilate the pupils in an unpleasant degree in many people if long continued. Hyoscine, the hydrobromate or hydrochlorate, is a much more pleasant remedy, and can be given three or four times a day without unpleasant effect; but it is not always so efficacious as the hyoscyamine. It is often supposed that these two drugs are identical in their effects, because of the chemical similarity. This is a great mistake, however, as hyoscine is much more efficacious in emotional or insane excitement than hyoscyamine, and has none of the depressing effects of the latter. The bromides are most useful in the treatment of tremor, when conjoined with hyoscine and hyoscyamine; the dose should be from 10 to 15 grains two or three times a day. The oxide of zinc, in doses of 1 to 3 grains two or three times a day, will often have an excellent effect, especially in the mercurial tremors, but it cannot be relied upon. Galvanism is always useful, and in proper cases frequently curative. It should be applied to the brain and the spinal cord; to the former a dose of 1 to 2 milliampères in sittings of two to three minutes; to the latter in doses of 3 to 5 milliampères in sittings of three to ten minutes. But no one should apply galvanism in these cases who is not properly equipped with a galvanic apparatus, accurate rheostat and milliampère meter, and a proper outfit of electrodes. The old habit of turning on so many cells of a battery without the slightest knowledge of the amount of current that is passing is akin to the old method of dispensing medicine in unmeasured boluses. Faradism is a much simpler form of electricity to apply, and may be used simply by holding the electrodes in the hands and letting a very gentle current pass for five or ten minutes, or, if the lower limbs are affected, applying the large electrode to the sole of one foot and holding the other electrode in the palm of the hand, and thus taking each foot and arm into the circuit in turn. Faradism is usually not efficacious by itself, although there are many exceptions to this rule, and the best results will be obtained by using it in alternation with galvanism. Rest is always useful in cases of tremor, and it should be proportioned to the needs of the case. If it is a weakened muscle that is tremulous the exercise of this muscle should be diminished, and if it is a weakened condition of the whole organism that is at fault the expenditure of energy by the whole organism should be correspondingly diminished. The particular application of this rule will vary with the tact of the physician and the circumstances of the patient; but in every case it is safe to say that in choosing between too much or too little rest the preference should be given to the former. In all cases of tremor the general health should be brought up to the highest pitch possible; indeed, in some slighter cases of neurasthenia I have seen tremor disappear in a few weeks when this alone was done; and in this raising of the standard of the general health tonics are, of course, necessary. Of these the best is quinine or calisaya bark, and iron should be conjoined with these in cases of anæmia.

CHAPTER X.

PARALYSIS AGITANS.

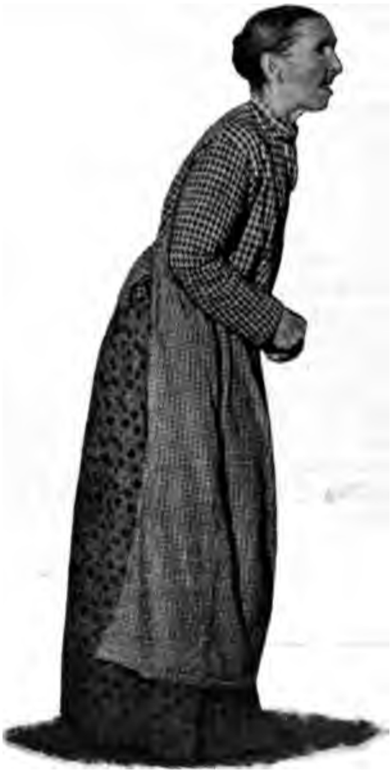
BY LANDON CARTER GRAY, M.D.

SYNONYMS. Shaking palsy; Parkinson's disease; Schüttelämung.

Parkinson, of London, first described paralysis agitans in 1817. Alth all the well-known authors have made this disease a subject of considera nothing essential has been added to Parkinson's description of it, which sl him to have been a man of rare intellectual capacity.

Symptoms. The onset is usually gradual, although it may in s cases be sudden. It occasionally begins in the lips, though most quently in one of the extremities. One case of my own, beginning in lips, continued for fifty years. A gradually extending tremor embraces muscles of the head and neck, the extremities, and sometimes the to and facial muscles. In some cases this is unilateral, but it generally ext bilaterally. In pronounced cases, or in those suffering a temporary exa bation, the tremor is so coarse that it almost resembles a mild localized r cular convulsion, although in ordinary cases it is simply coarse, without t additional movements characterizing a convulsion. A peculiarity of speech consists in its being deliberate, like that of a wise man caref choosing his words, and the attitude assumes a character indicative of particular disease. The head and neck are bent slightly forward, and patient holds the head and shoulders stiffly, and steps with a short, shuff gait. The facies is also peculiar, and very difficult of description, being singular blank immobility. The thumbs and fingers assume at this st what is known as the bread-crumbling position—*i. e.*, the thumb and fin are approximated and move over each other, as if in the act of crumbl bread. So-called propulsion takes place—that is, a tendency to go forwar and when once the patient is started in a forward direction his walk beco accelerated, and he finds it almost impossible to stop. This quite of occurs, but in rare cases the tendency is to retropulsion—going backwa In other cases a still rarer movement is observed—that of a sidewise incli tion, so-called lateropulsion. A slightly red color of the face is shown in marked cases of paralysis agitans, seemingly indicating good health, l in reality due to vasomotor paresis. Great stress is laid by Charcot on t fact that the tremor is usually only manifested when voluntary muscu movements are made, he claiming this as a valuable diagnostic sign; b too much importance should not be attached to its presence or absen as in cases that have not reached the typical stage it is often absent. some cases of paralysis agitans involuntary movements are observed. C siderable study has been devoted to the sphygmographic tracings of t tremor in the attempt to derive from it data by which to distinguish it fro multiple sclerosis. (See Chapter on Functional Tremors.) Charcot fou that the line traced by the tremulous extremity in paralysis agitans one of slight tremulous, continuous waves when no voluntary movemen are made, but longer and somewhat more irregular during voluntary effort. In multiple sclerosis, however, almost a straight line can be drawn when no voluntary movements are made, while in the case of effort the line is in large, coarse curves, much more so than at any time in paralysis agitans. Contracture and rigidity of the muscles are almost always to be observed, and exceptionally increase of the tendon reflexes. Among symptoms occasionally found are increased perspiration, tachycardia, a feeling of increased heat over the body or in certain areas, or of cold, numbness, prickling, or pain. Most patients afflicted with this disease are extremely restless, finding great difficulty in keeping quiet, and even insisting upon being allowed to walk about when they are almost helpless. The mental faculties are usually intact, and I have even known cases of fifty years' duration in which no impairment whatever could be detected in the mind. This was notably shown a few years ago in the case of a great national leader, who though helpless in body, continued vigorous in mind until the last. However, in some cases there is marked stupidity, and in others peevishness or slight mental dulness. The duration of the disease is entirely indefinite, and, a perfect cure being never effected, it continues during the remainder of life. It is a question whether it tends to shorten the patient's life, as I have known cases extending over thirty, forty, and in two cases even fifty years, thus bringing the patient up to sixty, sixty-five and seventy, a fair age for even a healthy person to live. Exacerbations and remissions often occur, sometimes so intense as to cause the patient to expect death. At such times the patient often becomes perfectly helpless, and at times comatose.

FIG. 68.



Side view of a case of paralysis agitans showing forward inclination of trunk. Tendency to propulsion. (DERCUM.)

Etiology. Age may be considered as a factor of importance, as most cases occur between the ages of forty and sixty, and sometimes up to seventy. Men are somewhat more frequently affected than women.

In various cases, however, the cause may be:

Cold and damp;
Emotions;
Heredity;
Trauma;
Strong peripheral irritation;
Hemiplegia;
Gout;
Typhus.

I have occasionally obtained a history of cold and damp as prominent factors, but this has not often occurred, although European writers claim that they are frequently the exciting causes of paralysis agitans.

Worry and anxiety are sometimes distinct causes, and fright also plays a strong part in the cases of many. A patient of mine, who suffered from paralysis agitans for fifty years, gave a history of having been crossing a river on a ferry-boat, and, while so doing, saw a child leap from its mother's arms into the water. It was swept down the river by the current for a considerable distance, being buoyed up by its clothing, and was finally rescued by a longshoreman. On returning to the cabin after witnessing this scene, my patient found, much to her surprise, that her upper lip was "going," as she expressed it, "like a rabbit's," and in this way the malady began.

I have found that heredity is a frequent cause, although it generally requires close and patient questioning to elicit the facts, as it may not have manifested itself in the immediate parents of the patient, although different generations of the family may have been irregularly affected. Emigrants from the north of Ireland and their immediate descendants, as well as many from among the population of Scotland, have been the most frequent cases in our New York clinics.

Trauma, gout, and typhus may be often found to be the exciting cause.

Cases of paralysis agitans have been reported by Westphal which have followed a wound, a laceration, or burns of an extremity.

Hemiplegia may sometimes precede paralysis agitans, as I have observed in five cases, although such cases should not be confounded with those in which one limb or one side of the body becomes tremulous.

Prognosis. The prognosis is generally good so far as the probabilities of life are concerned, for the disease seldom has a fatal or sudden termination, and it is a doubtful question as to whether it really shortens life. In most cases the prognosis is good as to improvement under proper treatment, although writers differ very much as to this point. However, cases in which the tremor is confined to one side of the body, or to one extremity, are apt to be very obstinate, resisting all treatment, and in these cases the affected limbs often become contractured and rigidly paralyzed.

Pathology. Until within a very short time it had been generally supposed that paralysis agitans was a functional disease of the nervous system, in the sense that no demonstrable pathological lesions were present. It is true that as far back as 1872 Joffroy had claimed that in three cases of the malady he had found the central canal of the cord almost obliterated by pigmentation, but this was also observed in many instances in which no clinical symptoms of paralysis agitans were present; and it was shown, too, that the slight indurations of the membranes, hitherto vaunted as pathognomonic lesions, were of frequent occurrence in the aged. But the observations of Borgherini, Ketscher, Koller, Schultze, Saas, Dana, and Redlich have brought this question to the fore again in a new light, for they have demonstrated that in the spinal cord and medulla oblongata of these cases there are islets of sclerosis which very evidently have their origin in the vessels. The condition is therefore designated as perivascular sclerosis. The vessels affected are those of small calibre. Their lumen is narrowed, there is a slight proliferation of the intima, the layers of the media are thickened, and are somewhat nuclear, hyaline, and concentrically layered. To the outside of these, generally in close proximity, sometimes with a small space intervening, there is a region of thick, horny tissue, which colors readily in ammoniacal carmine, and answers topographically to the layers of the adventitia or the adventitial lymph spaces. Miliary aneurisms or hemorrhages from this cause have been observed. From this point there is a thickening of the neuroglia extending into the surrounding nervous parenchyma, in places surrounding groups of nerve fibres, in others only individual nerve strands. Toward the periphery of the cord these appearances diminish. If there are many vessels close together, the otherwise separate small islets of sclerosis may become confluent, and there is formed a large sclerotic mass of tissue. The nerve fibres are not affected where the sclerosis is of small degree and especial groups of nerves are surrounded; but where the sclerosis is more marked, the nerve fibres seem to be compressed, so that the medullary layer and the axis cylinder have become diminished in size or have disappeared entirely. The large vessels may also be affected, even those outside the spinal cord, as the posterior fissural artery, and the interfunicular artery especially, in the coats of the media. These sclerotic changes are seen most clearly in the posterior columns of the cord, and especially in the middle and ventral portions, close to the posterior commissure. But the lateral columns are also affected, although the lesions are not limited to them; the direct cerebellar column, the Gowers column, and the lateral tract proper, bordering upon the gray matter, being implicated in a diffuse manner. The anterior columns are but very slightly involved, and often not at all. The vessels of the gray matter are often thickened, but there is no implication of the surrounding tissue. The lumbar cord and the cervical enlargement are the sites of the greatest implication, but the lesions are relatively far less marked in the upper cervical region and in the lower dorsal, and still less in the upper and middle dorsal. In some cases, also, there has been an evident implication of the cortical region of the cord, especially in the posterior portions of the lateral columns. Occasionally in this latter region there are streaks of tissue histologically similar to the cortical layer of the cord extended into the lateral columns, but disappearing around the nerve roots; in other instances there was a slight diffuse sclerosis, with increase of the [illegible] and of the neuroglia, apparently not of vascular origin, and in one case of Redlich's there was found a diffuse sclerosis of the perivascular character [illegible] has been described. In almost all cases the ganglion cells of the anterior horns were markedly pigmented, sometimes so much so that the whole of the cell was filled with yellow granules, hiding the nucleus and leaving only a small portion of the protoplasm, although this excess was [illegible] as the form and processes of the cell usually remain. A high degree of pigmentation is also present in the cells of Clarke's columns. The central canal is usually obliterated by proliferated ependymal cells. Amyloid bodies were found in large numbers, although nowadays there is good reason to believe that these formations are not to be regarded as pathological, inasmuch as Redlich has shown that they are present in the normal brain and often in large numbers. The pia of the cord was generally thickened to a slight degree, and excessively pigmented. The anterior and posterior roots were normal, although their vessels, whilst not thickened, have [illegible] been found widened. The nerve fibres of Lissauer's and Clarke's columns have been generally found intact. In one case of Dubief's the axis cylinders had a varicose shape, and he found the main site of the lesions in the cervical and upper dorsal cord. In one of Borgherini's cases aneurismal enlargements and hemorrhages were observed, and he also found the posterior columns affected, but to a slight degree relatively, whilst he differed from [illegible] the authors in observing marked alterations in the gray substance. Similar alterations have been found in the medulla oblongata by Ketscher and Dana. In some cases similar lesions of the vessels have been [illegible], but not the sclerosis. Borgherini, Saas, Ketscher, and [illegible] described alterations in the peripheral nerves and muscles, proliferation of the connective tissue and a consequent atrop [illegible] and muscular tissue, with thickening of the vessels.

The question has arisen as to whether these alterations that are be characteristic of paralysis agitans are not really due to senility. About twenty cases have been examined so far, and in all of these the appearances have been fairly constant, and in most of them the severity of the pathological lesions have borne a fair ratio to the clinical phenomena. But it is a well-known fact that obliteration of the central canal of the cord by proliferation of the ependymal cells, pigmentation of the ganglion cells, increase of amyloid bodies, and slight thickening of the membranes, are frequently found in old age in cases that have presented no signs of paralysis agitans, as has been shown by the observations of Leyden, Demange, and Redlich. The opinions of the neurological pathologists have differed concerning the meaning of the alterations in the vessels, the perivascular sclerosis, and the increase of connective tissue running in from the peripheral tissue of the cord. Most of the authors are inclined to regard these as purely senile. Ketscher, in particular, made a number of observations on old people, and found similar alterations of the vessels to those which have been described in paralysis agitans; but the intensity of the lesions was less, and the outer horny layer which has been described in the latter affection is generally wanting, as was also the perivascular sclerosis, whilst it is also to be noted that paralysis agitans, although it generally occurs in individuals toward fifty, may yet occur in young adults. Demange has described what he calls a progressive tabetic constricture of vascular origin of the lateral and posterior columns, clinically characterized by marked contractures of the limbs, later of the arms, without decided paralysis or sensory irritation, or objective sensory defect, increase of the tendon reflexes at first, with loss of them at a later period. These cases are, according to this author, those of chronic myelitis, caused by an endo- and periarteritis, and standing in a general relationship to general atheroma. Copin has described alterations in the aged of a somewhat similar nature, attended with muscular enfeeblement, but without the contracture described by Demange. Gowers's senile paraplegia probably belongs to the same class of cases. Buzzard has described the case of a man of thirty-nine who had had syphilis, then symptoms of diplopia, inability to walk or stand without assistance, although the legs could be freely moved in bed, loss of patellar reflex, one-sided optic nerve atrophy, marked sensory disturbances in the lower limbs, and immobility of the pupils, in whom there was found a widespread peri- and endoarthritis, with connected sclerosis in the cervical and lumbar cord, especially in the posterior columns, and to a much slighter degree in the lateral columns. Redlich has given the histories of several cases of old individuals in whom there was alteration of the vessels, with perivascular sclerosis in the posterior columns, particularly in the ventral portions of them, both the lumbar and dorsal cord being especially affected, the cervical cord to a less degree, and in some a slight ascending degeneration of the columns of Goll being present, the lateral columns being but slightly implicated. From all this it is evident that the character of the lesion in paralysis agitans is not so pathognomonic as is its localization, for there is no reason why the same lesions present in the cases of Demange and Gowers should not produce the symptoms of paralysis agitans if they were located differently. The two salient symptoms of paralysis agitans are the tremor and the contracture. Blocq suggests that the latter may be due to an affection of the muscles, but it has not been proven, and we must therefore assume that both the tremor and the contracture are due to the spinal lesion. With our existing knowledge it would be easy to suppose that the anatomical location of the lesion in the lateral or anterior pyramidal strands would be sufficient to account for both tremor and contracture; but, unfortunately, most of the autopsies have shown that the posterior columns are chiefly affected. It would seem, therefore, as if under certain circumstances the implication of certain fibres in these posterior columns was to be held accountable for the clinical symptoms. Why it is, however, that in the cases of Demange, Gowers, and Buzzard the implication of these posterior columns did not produce the symptoms of paralysis agitans can only be explained by the supposition that in these cases a different set of fibres was affected. In the cases of Demange and Redlich there was only contracture, and no tremor, so that by exclusion it would seem probable that the tremor was not due to implication of the posterior column, and was probably caused by affection of the lateral columns.

Diagnosis. The diagnosis of paralysis agitans is in most cases an easy one. The symptoms are so characteristic as to be readily recognized, even in meeting a patient in a car or on a ferry-boat. They consist, as has been said, of a coarse tremor, peculiar facies, tendency to ruddy hue of the skin, the slow deliberating, hesitating speech, the peculiar attitude, the bread-crumbling action of the fingers, and the age between forty and sixty. Cases in which these typical symptoms do not appear are difficult of diagnosis. In some patients tremor does not manifest itself, and the other symptoms must then be relied upon for diagnosis. Again, some patients present the tremor, but not the characteristic facies, color, attitude, or speech, and if this happens in a patient nearly thirty a diagnosis may not be possible without sufficient time to closely observe the progress of the disease. The character of the tremor was formerly thought to be of great importance, but such is no longer the case. I believe that in the majority of cases the tremor is present both when the patient is performing voluntary movements or is at rest; but in the early stages of the disease there are many cases which manifest the tremor only upon voluntary movements, or in which at such times it is much exaggerated. Much stress has been laid upon the sphygmographic tracings; but these are open to objection, because in many cases the tremor is continuous and fine, whilst in others it is very coarse and not continuous. Paralysis agitans should be differentiated from the following diseases:

Disseminated sclerosis;
The tremor of mercury, lead and copper;
Alcoholism;
Chorea.

Disseminated sclerosis presents a tremor only during voluntary movements —so-called intention tremor; the patient is a child or young adult; there is also interrupted, scanning speech, and nystagmus; symptoms of focal disturbance of the brain may appear, and the characteristic ruddy complexion, facies, slow and deliberate speech, bread-crumbling position of the fingers, and attitude of paralysis agitans are absent.

In lead-poisoning other symptoms besides tremor will be manifested, such as the wrist-drop and the "lead-line" upon the gums, conjoined with the history of the use of lead; while the tremor of mercury and copper, as well as that of lead, is generally fine. The history of copper or mercurial poisoning may usually be obtained.

Alcoholism often presents a fine tremor which may be of the intention type, especially after a debauch, but the diagnosis will generally be made clear by the history and appearance of the patient. In chronic alcoholism there may be a tremor, but this need only be observed in conjunction with the well-known symptoms and the history of the case.

Sydenham's chorea should never be mistaken for paralysis agitans, as in the former the movements are of the quickly beginning and ending type, and fibrillary in character, or in the athetoid variety they are more gradual in beginning and ending, while the patient is almost always a child. In some cases of chronic chorea, where proper treatment has been lacking, or frequent relapses have taken place, the tremor may become so spasmodic and marked as to mislead an inexperienced observer, but the diagnosis will be cleared up by the history and the character of the symptoms already described. Some writers have unfortunately referred to the jerky, fibrillary movements of chorea as choreic tremor.

Treatment. Definite and energetic treatment should be employed in cases of paralysis agitans. Drugs, however much vaunted for the purpose, are generally useless except for temporary relief. The expenditure of energy should be restricted. As much as possible, all fatigue should be avoided, and the patient should either be put to bed entirely for a time, or kept there until noon each day, retiring very early at night. As the patient improves he may be allowed to rise at the usual time, but nothing of a fatiguing nature should be undertaken, and it is much better to rest too much than too little. The physician should give explicit directions as to just how many hours the patient should rest, fixing the hours of rising and retiring, and the exact amount of exercise to be permitted. This is of the highest importance, as general principles of rest are of no use at all to the inexperience of the layman. Great attention should be paid to the diet, which must be of the most nourishing and abundant character. Three good meals should be taken each day, and in addition to this it is advisable for the patient to take a quart of milk during the twenty-four hours; and it may be even useful to give beef tea, using a pound of beef made into beef tea, or else a preparation of beef extract or of the peptonoids. Stimulation by alcohol should always be resorted to, and it may be either in the form of two or three ounces of whiskey in the twenty-four hours, or of wine or ale, an equivalent quantity of alcohol being obtained in the latter. Nutrition is also greatly assisted by malt extracts, of which only fresh and reliable preparations should be prescribed. The solid malt extracts are better than the liquid, as the precise quantity of malt can be better obtained in them, but in case the liquid form be more agreeable to the patient, it may be used. Tonics are always necessary, and the best of these is the sulphate of quinine, which should be given three times a day, in doses of 2 or 3 grains, an hour or two after meals, in order that no disturbance of digestion may be occasioned by it, and the best form is the tablet triturate. It should not be administered in solution with an acid, as this is apt to act unfavorably upon digestion. Besides these measures of rest, alcoholic stimulant, diet and tonics, the tremor requires remedies to be applied with a direct view to its control. The best agent for this purpose is hyoscyamine, the crystallized form, as unpleasant constitutional effects are apt to be produced by the uncrystallized form. One $\frac{1}{100}$ grain should be employed once or twice a day, and continued for weeks or months, as may be found necessary. Ten grains of the bromide of potash may be combined with the hyoscyamine, if the latter does not sufficiently control the tremor. This should also be given once or twice a day, usually for several months continuously. Hyoscine also seems to be quite useful, either in the hydrochlorate or the hydrobromate, although my own experience of it has not been sufficient to enable me to give a positive opinion as to whether it will supplant the hyoscyamine, which would be desirable, as it possesses none of the objectionable properties of the latter in causing dryness of the mouth and dilating the pupils, and it can be given much more frequently. The treatment thus outlined, consisting of rest, diastase, alcoholic stimulation, and muscular sedatives like those mentioned, comprises all that is needed in paralysis agitans. I believe that observers have been often misled by the ruddy color of the face, have thus refrained from administering stimulants, and have given the sedatives to such an extent that patients have been unduly depressed; but I have found that nourishing and stimulative treatment alone will accomplish more than the exclusive use of the sedatives. Galvanism is a nervous stimulant of a high degree of usefulness, and should almost always be employed. It should be resorted to at least three times a week, and one large electrode should be applied to the nape of the neck, while a smaller one is placed at the lower dorsal spine. Hot water should be used to moisten both the electrode and the skin. From three to five minutes should be occupied at a sitting, and a current of three to five milliampères should be used, and this may be increased to a sitting of five, fifteen, or twenty minutes, and to a current of ten to fifteen milliampères. I can positively assert that with this treatment most cases of paralysis agitans can be modified so that the patient's life may be made comparatively pleasant. If eight or twelve weeks of this treatment bring relief, the hyoscyamine and bromide may be gradually withdrawn, the galvanism discontinued, and the rest, the diet, the diastase, and the alcoholic stimulant will be found to be all that is necessary. The same means can be employed to control recurring exacerbations, which usually take place in the course of a few weeks or months. I tell my patients frankly that a complete cure cannot be expected, and that these relapses must therefore be looked for and combated by treatment, so that no alarm need be felt at their recurrence. In those cases which occur after a hemiplegia affecting only one limb or one side, there is, unfortunately, not much hope of improvement. But the cases of paralysis agitans which occur after hemiplegia should not be confounded with those of sudden onset in one limb, for the latter are usually only the local manifestation of a general paralysis agitans which will yield to the therapeutic measures that have been laid down.

CHAPTER XI.

EPILEPSY.

By LANDON CARTER GRAY, M.D.

SYNONYMS. Latin, Morbus sacer seu comitialis; French, l'Epilepsie; German, Fallsucht; Spanish, Mal caduco.

Definition. Epilepsy is derived from επιλαμβανω meaning "the falling sickness," as a loss of equilibrium was the first phenomenon that attracted attention. But since that time the term has been applied to a far wider range of phenomena, which are characterized by a sudden loss of consciousness, with convulsions affecting muscles in varying degree, or loss of consciousness without muscular implication, or muscular convulsions without loss of consciousness; and it is also given to conditions of alternating consciousness, as well as to certain mental phenomena that are supposed by some authors to be the equivalents of the convulsive or comatose symptoms.

HISTORY. The disease has been known as far back as the time of Hippocrates, but it has been confounded with hysteria and other nervous affections, as in the case of Mahomet, for instance, who was probably afflicted with what is known now as hysteria major, whilst the oft-cited case of Napoleon the Great does not answer at all to the description of what we nowadays know as epilepsy; indeed, our exact knowledge of the subject has been gained largely within the last twenty years, synchronously with the information we have obtained concerning the anatomy and the functions of the nervous system.

Symptoms. The principal clinical forms assumed by epilepsy, or rather epileptiform affections, are the following:

Grand mal; major epilepsy;
Petil mal; minor epilepsy;
Convulsive movements without loss of consciousness,
Vertigo;
Double consciousness;
Hystero-epilepsy;
Procursive epilepsy.
Laryngeal epilepsy.

When we speak of typical cases we mean those that are the most numerous. In this sense the typical attack of epilepsy consists of a sharp cry, an instantaneous loss of consciousness in which the patients falls heavily, the rapid supervention of tonic and then of clonic convulsions, both of the latter being of short muscular range and automatic in character rather than purposive, these phenomena lasting for several seconds or minutes, when the patient becomes quiet, awakens into a dazed consciousness, and then gradually sinks into a deep sleep lasting one hour or several. This is known as the grand mal. But the deviations from this type are innumerable. The most frequent is the so-called petit mal. This consists of a loss of consciousness so slight that it seems often like mere absent-mindedness. The eyes may turn upward and have a staring appearance, and there may be some slight fibrillary movements of the facial muscles, and I believe, as is claimed by Hughlings-Jackson, that these slight muscular movements always occur in these cases; but they may readily be overlooked, a fact which is of great medico-legal importance as bearing upon the nature of the serious maniacal attacks which may follow, and during which assaults or murder may be committed. In other cases there may be only convulsive movements of the muscles, as in one patient of mine who could never undertake to carve at his dinner-table because of the tendency that his arms had to fly apart and whirl carving knife and fork in eccentric and dangerous orbits. A sensation of vertigo very frequently occurs in epileptic individuals in the intervals between the *grand* and *petit mal* attacks; but, so far as may experience goes, it never occurs without more pronounced evidences of epilepsy, and can therefore be readily distinguished from true attacks of vertigo, which are so common from other and harmless causes.

The conditions of double consciousness occur after the attacks of *grand* or *petit mal*, and there is no limit to their variety. I believe that many of the cases that figure in the newspapers of people wandering away from home and coming to themselves afterward in far-a-way places may be ascribed to this cause. One singular instance of this was a boy who came to my clinic. He would leave home after an attack of *grand mal*, disappear into the slums of the city, engage in some menial occupation far below his usual social station, fill it without arousing the slightest suspicion in those about him, and suddenly return to his normal condition of consciousness after several weeks, when he would return home very much discouraged and amazed. Of these varieties the most frequent is the *grand mal*, and next the *petit mal*. In many of these attacks there are certain sensations known as *auræ*, and these vary extremely in kind, depending probably on the localization of the lesion in the nervous system. The color of the face in these attacks is very variable, sometimes pale, sometimes suffused, sometimes cyanotic, and sometimes there is no alteration at all. The statements of on-lookers regarding this fact are apt to be unreliable, as the latter are usually too much disturbed to be accurate observers, whilst the physician himself seldom sees an attack, even if he be the resident of a hospital, for, while I have probably treated thousands of cases, I could easily count on my fingers all those that I have observed during the attack itself.

Hystero-epilepsy is the name given to an odd combination of hysteria and epilepsy, and the descriptions are largely due to the writings of the physicians of that great neurological hotbed, the Salpêtrière of Paris. But the cases that have been seen in England and this country are of a far less sensational nature. True hystero-epilepsy rarely begins with a cry or change of facial color, but generally with the tonic convulsions of true epilepsy. These are followed by a so-called "clownishness," which is characterized by all sorts of absurd movements, opisthotonos, distended abdomen, cramps, etc. Then succeed a series of passionate attitudes, with delirium. It has been in these cases especially that ovarian compression has been so much vaunted, but I have not found it of much use. Hystero-epilepsy differs, however, radically from true epilepsy in the admixture of predominance or purposive or seemingly purposive movements, instead of a tonic muscular movement of shock-like character and of limited muscular range, while opisthotonos, a symptom rarely or never seen in true epilepsy, is occasionally observed. (See also page 97.)

Procursive epilepsy has been recently described by Marie, and consists of running movements forward, but otherwise presenting the usual phenomena. It may alternate with ordinary epilepsy, precede it, or merge into it.

The so-called laryngeal vertigo is unquestionably a variety of epilepsy, and the term vertigo ought to be discarded, because it should be reserved for those subjective sensations of dizziness without loss of equilibrium which are not usually accompanied or followed by any impairment of consciousness. As yet this form has only been described in adults. It consists of a slight loss of consciousness, occasionally with light tonic or clonic movements, and the exciting cause is some laryngeal, tracheal, or bronchial affection, such as laryngeal tumors, asthma, etc. Although the cases so far reported have ceased after the cure or removal of the seeming cause, the histories have not as yet extended over sufficient time to warrant us in stating that they may not recur.

In some cases of epilepsy what is known as the *status epilepticus* may be observed, consisting of frequently recurring attacks of convulsions with intervening unconsciousness, both lasting for hours or days. This may be observed in almost any patient from whom the bromide has been suddenly withdrawn after he has been accustomed for a long time to large doses of it.

In some epileptics the disappearance of the epilepsy may lead to the supervention of certain so-called equivalents, one of which is a peculiar dream-like state, another the so-called larvated epilepsy of Falret, consisting of an outbreak of hysterical insanity, usually of the maniacal type, or, as I have occasionally seen, an attack of violent migraine, great irritability of temper or enuresis. Falret claims that there is never any *petit mal* preceding these attacks, but Hughlings-Jackson has called attention to the fact, which I have already alluded to, that petit mal may be very readily overlooked, because of its short duration and the slightness of the muscular twitchings of the face.

Much confusion has been injected into the subject by the failure to recognize that epilepsy is only a symptom, just as is fever or cough, and that in every case the question of diagnosis is resolved into the further one of what the epilepsy is a symptom of. From this point of view we may, therefore, divide all epilepsies into those that are due to recognizable organic disease and those that are idiopathic.

Idiopathic epilepsy can, of course, only be diagnosed when the different organic lesions have been excluded, but it has certain features which are of importance from a therapeutic and diagnostic standpoint, and prominent among these are the nocturnal recurrence of some attacks, the quasi-periodicity of others, the association with migraine, and the temporary response, usually in a favorable way, to slight changes in the environment or the treatment. Nocturnal epilepsy should always be suspected in an individual who is hereditarily predisposed or who wakes in the morning exhausted and pale. For some thirteen years I have been calling attention to the quasi-periodicity of many epilepsies, manifested sometimes by a recurrence of the attacks at certain periods, as in females at menstruation, or in others at certain times of the month or at certain intervals, the latter sometimes lasting months or even years. For thirteen years, too, I have been lecturing upon the conjunction of epilepsy with migraine, and I had not known until the publication of Féré's book, in 1890, that Tissot, Parry, and Liveing had previously observed the same fact. In these cases the migraine alternates with the epilepsy, the former disappearing upon the supervention of the latter, and *vice versa*. This does not imply that all cases of migraine are subject to epilepsy, but only that there is a very close relationship between the two diseases; and I may furthermore state my belief that almost all cases of migraine, if carefully examined, will be found at some period to have had a loss of consciousness, with or without convulsive movements. It is not generally known that epileptics are readily influenced by slight changes in the environment and in the treatment, a fact which explains why epileptics almost always do well upon any change of treatment, whether this be medical or surgical—whether it consist of cutting off the prepuce, removing the clitoris, extirpating ovaries, doing operations upon the male or female genitalia, using the hot iron or moxa, cutting the eye muscles, or even, as in one case of my own, in etherizing the patient and cutting a piece of skin out of the buttock. Moreover, these epileptic attacks are very prone to recur in variable spells, every day or every alternate day for weeks or perhaps months, then disappearing for a variable length of time. Most epileptics, as I have pointed out, have large and variable pupils. Marie and Musso have contradicted me upon this point, but the very variability of the pupils renders their measurements inconclusive. Most epileptics have peculiar facies, which it is impossible to describe or photograph, but which may be readily detected by the trained eye. This facial characteristic is, of course, much more marked in those who have been for a long time under the bromide treatment, and to it will be superadded a peculiar restlessness, pallor, acne, coated tongue, and the peculiar breath. The temper of most epileptics is extremely irritable, and especially is this the case in children whose malady has caused their parents or relatives to spoil them.

Etiology. The following factors bear certain relationships to epilepsy:

Age;
Sex;
Heredity;
Migraine;
Organic brain, spinal, or peripheral lesions;
Traumata;
Lesions or impairment of functions of non-nervous organs;
Hysteria;
Malnutrition.

By far the largest proportion of cases occur under nineteen years of age, and most frequently from ten to nineteen. After this the greatest number are observed from ten to twenty-nine; and after the latter age there is a rapid decrease in frequency, until the cases become very infrequent after fifty, still more so after sixty years of age. I have been for years in the habit of showing by the histories of my clinical patients that many cases of idiopathic epilepsy will begin with a fit in early infancy, after which no convulsions may occur for a year or for years, when the attacks will again appear, and at this or some later period the case will become well marked.

Females are rather more subject to epilepsy than males; and heredity occurs in about one-third of the cases. It is undoubtedly true that epilepsy may be caused by diseases of the heart and of the kidneys, by impairment of functions of the gastro-intestinal organs, and of the lungs; but this is rare, and the so-called genital epilepsy is a myth.

The association of hysteria and epilepsy is by no means frequent.

Profound malnutrition may be a cause of epilepsy, for I have had several cases in which the attacks have disappeared for ten, eleven, five, seven, or nine years after the restoration of the general health.

Pathology. Epilepsy may be divided pathologically into two great classes: first, that which may be referred to organic disease, and second, idiopathic epilepsy.

Organic epilepsy may be caused by syphilis of the nervous system; diseases of the peripheral nerves; meningitis, either cerebro-spinal, tubercular, or suppurative; tumors of the brain; abscesses of the brain; ear disease; sclerosis of the brain and cord; the cerebral palsies of childhood (due to such lesions as porencephalitis, hemorrhage from the cerebral arteries, embolism or thrombosis of the cerebral arteries or veins.)

Very little is known of the pathology of the idiopathic form. Neurologists have now almost entirely discarded the old theory of vasomotor spasm producing cerebral anæmia or hyperæmia, for no logical proof of it has ever been produced. Convulsions may be induced by great hemorrhage or by ligature of the carotid artery; but the profound ischæmia thus resulting has never been proven to be present in the attacks of idopathic epilepsy; whilst, on the contrary, there is much to show that no marked vascular alterations take place in the cerebrum of epileptic human beings and animals. Vulpian has found that no hyperæmia or ischæmia appears during an attack of epilepsy artificially produced in guinea-pigs. No warrant exists for the conclusion drawn by some authors from the facial circulation, it being by no means an index to the cerebral circulation, as it often happens that in a profound hyperæmia of the cerebral meninges the face may be deadly pale. This also applies to deductions that might be drawn from the condition of the circulation of the retina. A sclerotic condition of the brain will be found in some cases of idiopathic epilepsy, and in some the pia mater will be the seat of an old meningitis, whilst no lesions are discoverable in many cases. Much discussion of the probability of sclerosis of the cornu ammonis being a cause of epilepsy has taken place; but this is by no means a constant feature, and it is impossible to demonstrate whether it is a cause or an effect. Great stress has been laid upon a peculiar variety of sclerosis by Barthez and Rilliet, to which they have given the name of tuberous or hypertrophic sclerosis, but the same objections which have been brought to sclerosis of the cornu ammonis also apply to this. In five epileptic brains examined by Chaslin, at the request of Féré, neurogliar sclerosis was found in four. The convolutions were small, hard, smooth, or slightly roughened, macroscopically shrivelled. No abnormalities or adhesions of the pia mater were observed. A great irregularity in distribution over the surface of the cerebrum prevailed, large normal portions sometimes extending to the medulla oblongata or the cornu ammonis. The olivary bodies were only thus affected in one case. A microscopical examination of the fundamental lesion shows that the cerebral tissue, especially the gray cortex, had been invaded by a number of rough fibrillæ of uncertain length. The author says: "In the normal state the first layer of the gray cortex contains certain so-called spider cells, whose prolongations are scarcely visible. In this condition, on the contrary, this first layer is formed by a bundle of fibrils arranged nearly parallel to the cerebrum, and it can be distinctly seen to originate from numerous cells with hypertrophied prolongations. In the preparation which I am viewing at this moment there is a place where this transformation invades all the layers, but leaves intact numerous nerve cells and vessels. It can be seen, moreover, that in a certain space these fibrillæ form in the depth of the cortex a network of nodal points in which lie the cells of the neuroglia. Finally, and I would call attention particularly to this fact, this first layer is studded in places by large compact bundles, which are evidently formed from these fibrillæ. I would observe, in passing, that the vessels which remain do not present a trace of inflammation, there being simply, in certain points, a hyaline transformation of the capillary wall." There is a question in my mind whether Chaslin has not regarded as connective tissue what Cajal has recently demonstrated to be protoplasmic prolongations of the nerve cells of the molecular layer of the cortex. Chaslin adopts certain of Ranvier's views, and maintains that a distinction should be made between the connective tissue of mesodermic origin and the neuroglia, which is of epithelial or ectodermic origin; the latter embracing Müller's fibres of the retina, the fibres and cells of the neuroglia in the spinal cord, and the slightly differentiated prolongations of the spider cells in the brain. He describes, for the first time, this peculiar neurogliar sclerosis, which is, he claims, entirely distinct from a sclerosis of the connective or mesodermic tissue. The peculiar fibrillæ are emanations from the neuroglia cells, indirect proof of the same assertion being found in the non-adherence of the pia mater and the vessels. These fibrillæ resist the successive action of a 40 per cent. solution of potassa for ten minutes, as well as washing with water and concentrated acetic acid, whilst the picrocarmine color remains after washing, and formic glycerin is an efficient preservative. Treated in this manner, the connective tissue swells and decolorizes, so that the neuroglia will be intact in a section of the spinal cord, whilst the pia mater will also be decolorized and swollen. Moreover, after the action of alcohol diluted to one-third, all other forms of the connective tissue are decolorized, but these fibrillæ remain colored by the carmine when subjected to the acetic acid. This pathological distinction of Chaslin's merits attention on account of its definiteness. Bevan Lewis has observed certain striking changes in the cells of the second layer of the cortex, and in some cases this degeneration has been seen to affect all the layers, including the spider cells.

This indefinite knowledge of the pathology of epilepsy is no more exact than that of the *modus operandi*. The older writers held that the medulla oblongata was the portion of the nervous system most concerned in the symptomatology. Nothnagel claimed that there was a certain area in the floor of the fourth ventricle which he called the convulsive centre, and that irritation of this caused the epileptic convulsions. He regarded this as the vasomotor centre of the medulla oblongata. Wernicke holds that the location of this area corresponds with the lateral motor field of the tegmentum in the pons. Complete section of this region did not produce death by bleeding nor convulsions, and a simple section will suffice as an irritant. Violent and generalized convulsions are caused by the near approach of the section to the corpora quadrigemina, and a simple deep section will cause convulsion of the lower extremities. This area of Nothnagel's is the point of termination of the so-called long nerve strands in which run the fibres of the upper and lower extremities, as has been demonstrated by Osjannikow, so that it simply adds another fact to our knowledge of the various focal lesions causing epilepsy. Van der Kolk had an airy theory to the effect that the nuclei of origin of the hypoglossal nerve in the floor of the fourth ventricle were indurated in proportion to the amount of tongue-biting which was manifested in the convulsions, but this is scarcely worth mentioning, much less soberly discussing. Fritsch and Hitzig have by their experiments accumulated facts tending to demonstrate that indirect excitation of the cortex or of the nerve strands leading to the peripheral structures is causative of epilepsy in a great number of cases, and, in the light of these discoveries, Nothnagel's observation has only demonstrated a similar subsidiary centre. It has been shown again and again that convulsive attacks of clonic or tonic nature can be produced by mechanical or electrical irritation, and that the same effects can be caused by similar irritation of the nerve strands underlying the cortex. Duret has experimented in this direction also, and has produced the most widespread convulsions by irritation of the cerebral membranes, particularly the dura mater. It should not be forgotten, however, that many extra-cranial lesions may be productive of epilepsy, such as diseases of the spinal cord, alterations in the blood, such as are to be found in malnutrition and albuminuria, disorders of the peripheral nervous system, as well as of many of the non-nervous viscera, and by various febrile conditions and toxic agents, so that it would be presumptuous to assert that the cortex of the brain is alone affected in all these varying conditions. The truth probably is that the epileptic manifestations are due to a peculiar molecular condition of the motor tract running from the motor convolutions to the peripheral structures and muscles. But as to the exact nature of this molecular condition we are in complete ignorance, as we are also ignorant of the conditions producing any one of the functional nervous diseases, and it is highly probable that such lack of knowledge will prevail until in the far-off future such precise appliances will be ours that we will be enabled to see the molecular play in the living brain and spinal cord, or until our methods of preparation and staining are so perfected that we can detect such minute cellular alterations as have thus far escaped our observation. Many metaphysical theories have been offered of these cellular changes, but they are too puerile and ridiculous to merit any attention. Whatever this altered molecular conditions may be, there is no doubt that it expresses itself in epilepsy through the motor tract running from the motor convolutions to the muscles of the periphery. Muscles can only be convulsed by direct excitation of the muscles itself, or of the motor tract leading up to the convolutions, or of the convolutions themselves. (See in this connection also Chapters XIV and XVI.) But some varieties of epilepsy are due to an excitation of the nervous system in some part that extends beyond this motor tract, as is manifested by such symptoms as word-deafness, hemianopsia, aphasia, or the forward movements of propulsive epilepsy, indicating a lesion in the corresponding centres of the cortex, or cerebellum, or the nerve strands underlying them; and the correctness of this view has been demonstrated by autopsies; while in the convulsion arising from lesions of non-nervous organs, from toxic agents, and from diathetic conditions, in direct implication of this motor tract, must be the cause. Idiopathic epilepsy, like migraine, neuralgia, chorea, and hysteria, is therefore a neurosis, and it is a matter of grave doubt whether the cellular and sclerotic changes discovered are not effects rather than causes.

Diagnosis. The fact should ever be borne in mind that epilepsy is a symptom, and the object should be to discover of what it is a symptom. Our chief efforts should be directed to ascertaining whether it is caused by any organic disease, such as lesions of the brain, spinal cord, or peripheral nerves; whether it is related to nephritis, malnutrition, heart disease, marked gastro-intestinal disturbances, or whether it accompanies the cerebral palsies of childhood.

After the exclusion of all these organic lesions a diagnosis of idiopathic epilepsy may be made. This should not be difficult. The convulsions are tonic or clonic, with loss of consciousness, or are of slight force, with loss or simply impairment of consciousness, and need only be differentiated from simulation and hysteria. Hysterics do not fall and bruise themselves, or bite the tongue, as epileptics do, for true hysterical attacks have not the same loss of consciousness. Moreover, the movements of hysteria are of a volitional character to a large extent, and also of wider range, whilst in epilepsy the muscular movements consist only of simple rigidity, with slight tremor, extending only over a short period of time, or of limited flexions and extensions. However, it should be borne in mind that in hystero-epilepsy, true hysteria and true epilepsy may be conjoined.

Epilepsy is rarely simulated in this country, except in some prisons, where a transfer to the hospital or asylum may be effected by a patient afflicted with this disease. It is said to be very frequent in Europe among those wishing to escape military service. Of all the attempts made by various authors to discover some symptoms of epilepsy that should be absolutely pathognomonic, none have been successful. Marc thought that the convulsions of true epilepsy would not be increased by holding asafœtida under the patient's nose, but this has been controverted. Voisin placed great dependence upon the condition of the pulse before, during, and after an attack; but similar variations have been observed in other conditions than epilepsy, while Féré has shown that they are absent in some epileptics, and vary considerably in others. There exists among English thieves a class known as "dummy chuckers," whose business it is to feign convulsions in a crowd, attracting the attention and sympathy of the bystanders, whilst a confederate rifles their pockets. One of these men was for some time an inmate of our American prisons, and I have frequently had him simulate a convulsion for the benefit of my class, and it is certainly true that this counterfeit convulsion is extremely like the genuine, and difficult to detect, except by repeated and close observation, when the tendency to overdo the tonicity or clonicity of the movements and the bleeding of the tongue (which is generally cut with a knife beforehand) may assist in the detection.

Prognosis. It is still an undetermined matter as to what constitutes a cure of epilepsy. Some cases may continue free from attack for a period of ten, fifteen, or even twenty years, and an infantile convulsion may not be succeeded by further attacks for many years. I have in my own practice known remissions of several years—sometimes six or seven. The life of a patient may sometimes be made more endurable, and even pleasant and useful, by modifying the violence and lessening the frequency of the attacks. Some cases, however, are beyond the means afforded by the best skill of the medical profession. Most cases of *grand mal* may be improved, but a cure can only be effected in a few of them. The only way in which it can be ascertained what degree of improvement can be made is by the experimentation of a month or so of treatment. The patient's relatives should always be informed that the treatment must be faithfully continued for years to even obtain a relief. *Petit mal* is very rarely affected by treatment, and my experience has been that only in those induced by malnutrition and formation of ptomaines from acute indigestion does improvement occur. Epilepsy of peripheral origin may be expected to improve under treatment, although the cases are seldom entirely freed from a convulsive tendency. Those in whom migraine is associated may usually be favorably affected by treatment.

Treatment. As epilepsy is of many varieties, so its treatment must vary according to the different manifestations of the disease. An operation should be done upon cases caused by intra-cranial lesions if our present knowledge of localization can be availed of as a guide. But before operation is attempted the nature of the lesion should be fully determined. It would be folly to operate in cases of single or double hemiplegia and in the paraplegias of the so-called palsies of childhood, as they are due to losses of cerebral substance known as porencephalitis, to hemorrhage, arterial or venous thrombosis, or sclerosis, which usually proceeds from an anterior arterial trouble. An operation should always be resorted to in cases of well-defined brain tumor; also in abscess, if it can be localized and is in an accessible area. The scalp should be carefully shaved in cases of cerebral trauma, and a minute search made for the cicatrix or depression, and trephining should be performed if either is found, for most excellent results have been obtained in many cases by removal of slight adhesions of the membranes. Under proper antiseptic precautions trephining is a harmless procedure, and may bring to light conditions which otherwise might remain hidden. (See also Chapter XXXIII.)

My friend, Dr. E. G. Mason, has kindly collected for me forty-four cases of epilepsy that have been operated upon.

No.	Reported by, or operator.	Date of trauma.	Interval between trauma and appearance of fits.	Character of fits.	Interval between and number of fits before operation.	Interval between appearance of fits and date of operation.	Date of operation.
1	C. A. Dundore	November 8, 1888	3 months	Marked on right side of body.	8 fits before operation; intervals not given.	1 month, 13 days	1st oper. March 23, 1889; 2d oper. July 20, 1889.
2	J. T. Wilson	Not given; wound of coronal suture ½ an inch above temporal bone.	....	....	......	......	9 months after trauma; dates not given.
3	J. T. Wilson	Not given; fracture left frontal bone near junction with parietal.	5 weeks	....	At irregular intervals; number not given.	15 months	Not given 1st oper.; 2d oper. 4 months after first.
4	F. Salzer	1884: left side of head.	Immediately?	Right hand and arm, muscles of face and eyes.	1 or 2 daily until 7th d'y after injury, when ceas'd, and had none for 3 years, then at irregular intervals.	4 years	1888
5	Ch. Féré	Nov. 28, 1870, struck on frontal bone by fragment of shell.	6 months	....	Fits at irregular intervals, very much increas'd when drinking absinthe.	17 years	Oct. 29, 1887
6	Robert Jones	Date not given; trauma over left Rolandic fissure.	1 week +	....	Not given.	More than 2 years	Not given
7	Guy Hinsdale	February 17, 1888, injury to left testicle.	60 days	General	At irregular and frequent intervals.	8 months	Dec. 29, 1888
8	Guy Hinsdale	Not given; bean in left nostril.	Not given	General	At irregular intervals.	Not given	Not given
9	Whipple	June, 1891, over right Rolandic fissure ¾ inch from sagittal suture.	4 days	....	Five or six weekly.	6 mos +	Jan. 8, 1892
10	John Snyder	No trauma; uterus small, anteflexed, and very small cervical canal.	....	General	Irregular intervals.	......	......
11	Stephen Crane	No trauma.	....	General	Irregular intervals.	......	Aug. 1, 1892
12	F. T. Hewston	1885; injury to left occiput.	10 months	....	As many as 4 daily.	5 years+	March, 1891
13	A J. Miller	No trauma; palmar contractions and tight prepuce.	....	Right hand and arm flexed; right leg extended.	At irregular intervals.	5 years	Dec. 6, 1891

Kind of operation.	Lesion found.	Interval between and number of fits after operation.	Result as stated by reporter.	Length of time under observation after operation.	Reference.	Remarks.
Trephine. Trephine.	....	13 fits until second operation; 4 after second oper.	......	5 mos.+ after 2d operation.	Col. & Clin. Rep, 1889. vol. x. p. 281	Time of observation after operation insufficient.
Trephine.	Thick cicatrix in dura which was not disturbed.	No fits for 3 months, when died during a severe seizure.	......	3 months	Med. & Surg. Rep., 1888, vol. xlix. p. 737	Autopsy showed cicatrix and bulging of dura, abscess, spicule of bone, softened brain tissue. Temporary recovery (3 months), then death.
Trephine. Trephine.	Loose piece of internal table. Tense cicatrix of dura removed.	First fit 21 days after, then at irregular intervals for 4 months, when had 2d oper.; 5 or 6 mild fits during next 3 months, then they ceased.	......	5 years	Ibid., p. 738	Cure after second operation.
Trephine.	Thickening and adhesion of dura	Fits 8 days after operation, continuing at irregular intervals.	......	....	Wien. Clin. Woch., 1889, vol. ii. p. 91	No improvement.
Trephine.	Rough projection of bone removed.	Not given.	......	Not given	......	In all probability discharged on 14th day after operation, "and could now be considered quite cured." Time of observation after operation insufficient.
Trephine.	¾ in. spicule of bone removed.	Not given.	"Patient made an excellent recovery."	Not given	Liverpool Med. & Chir. Journ., 1889, vol. ix. p. 451	Time of observation after operation insufficient.
Removal of left testicle.	Atrophy of glandular structure of testicle.	No fits.	Cured.	3 months	Am. Journ. Med. Sci., 1889, vol. xlvii p. 587	Time of observation after operation insufficient.
Removal of bean.	....	Not given.	Cured.	....	Ibid.	Time of observation after operation not given.
Trephine.	Bone thick; dura adherent.	None.	Recovery.	1 month	Lancet, 1892, vol. i. p. 868	Time of observation after operation insufficient.
Cervical canal enlarged and straightened.	....	....	"Epilepsy cut short as if by magic."	No definite date; came under treatment May, 1891; published July, 1892	Physician and Surg., 1892, vol. xiv. p. 305	Time of observation after operation insufficient.
Dilatation of cervix uteri.	....	8 fits.	Well.	5 months	Maryland Med. Journ., 1893, vol. xxviii. p. 529	Time of observation after operation insufficient.
Trephine.	....	No fits.	Recovery.	10 months	Trans. Roy. Acad. Med., Ire., 1891-2. vol. x. p. 166	Improvement, though time of observation was insufficient to determine amount of improvement.
Palmar facia forcibly stretched; prepuce "slit up."	....	1 fit.	Patient returned some mos. afterward and reported himself quite well; cured.	....	Edinb. Med. Journ., 1893, vol. xxxix. p 20	This case was given bromides in addition; improvement. Time of observation insufficient.

No.	Reported by, or operator.	Date of trauma.	Interval between trauma and appearance of fits.	Character of fits.	Interval between and number of fits before operation.	Interval between appearance of fits and date of operation	Date of operation.
14	C. F. Barber; operation by Hammond	1877; depressed fracture of right frontal bone.	Immediately	....	Vary from 1 monthly to 1 weekly.	8 years	......
15	C. F. Barber; operation by F. W. Rockwell	No trauma; epilepsy since childhood.	....	Fits general; aura in left arm.	1 to 20 in 24 hours.	Not given; some years	......
16	Thomas Wells through C. F. Barker	When 6 years old depression in post. sup. corner of right parietal.	10 years	General	Vary from 1 daily to 1 in two weeks.	Not given	Aug. 24, 1885
17	H. C. Dalton	1876, kick by mule in left frontal region.	1 month	General	Irregular intervals.	12 years	1888
18	M. A. Starr; operation by McBurney	April, 1891, fracture of left parietal bone.	3 weeks	Numbness and tingling of right hand, shoulder, body, and leg; no loss of consciousness.	Frequent intervals.	20 months	Nov. 9, 1892
19	Starr and McBurney	1882, fracture over left coronal suture; 1890, a second fall on head.	1½ years after second fall.	Spasm of right hand; no loss of consciousness	As many as 6 daily.	1½ years	Jan. 30, 1892, Apr. 1892, 2d oper. Jan 7, 1893
20	Starr and McBurney	April, 1888, fracture about middle of coronal suture, right side.	3 years	Left arm and hand, turning head to left, then became general.	As many as 2 daily; longest interval was 9 weeks.	1 year	March, 1892
21	Starr and Weir.	April, 1888, fell on left side of head near vertex.	Not given	Spasm right foot and leg, turn to right, lose consciousness and fall in a general fit.	As many as 6 daily.	3 years	Jan. 17, 1890
22	Starr and McBurney	1882, fell on head.	Immediately.	Twitching of eyes and head, most on right side; general convulsions.	Irregular intervals.	Petit mal until two years before, after which grand mal	Dec. 2, 1892
23	Starr and Briddon	August, 1889, fracture of right temple.	About 1 year	At first general, but later muscles of face right side, right side of neck, right arm and hand.	Irregular intervals.	2 years	Dec. 19, 1892
24	Starr and McBurney	1887, fall on vertex, right side anterior to Rolandic fissure.	"Soon" afterward	General, with green visual aura.	Not given.	5 years	June 10, 1892
25	Starr and McBurney	April, 1890, struck on left side of head with sandbag.	3 mos. +	Tingling movem'nt in right hand and face; face drawn to right, mouth opened and closed; speech lost; on two occasions lost consciousness.	Irregular intervals.	11 months	June, 1891
26	Starr and Poore	Not given; fall on right side of head.	3 months	Spasms of left arm.	Irregular intervals; frequently as 7 daily.	Not given	Oct. 1889

Kind of operation.	Lesion found.	Interval between and number of fits after operation.	Result as stated by reporter.	Length of time under observation after operation.	Reference.	Remarks.
Trephine.	....	1 weekly, varying to 1 in two or three weeks.	......	Not given	Brooklyn Med. Journ., 1893, vol. vii. p. 640	No improvement.
Trephine over centre for left arm.	Nothing abnormal	Fits reappeared in one week	......	....	Ibid.	No improvement.
Trephine.	....	None.	......	About one year.	Ibid.	Was under bromides. Time of observat'n after operation insufficient.
Trephine.	Unusual amount of fluid escaped.	2 fits day after operation, 1 on third day.	Successful.	3 years	Med. Fortnightly, 1892 vol. i. p. 75	Cure.
Trephine.	Splinter of bone in motor area; cyst of pia.	2 slight attacks subsequently.	Recovery.	4 months	Brain Surg., Starr, p. 29	Time of observation after operation insufficient.
Trephine over right arm centre; small pus cavity at site of operation evacuated; trephine.	Dura and pia adherent; 2 cysts; brain appeared somewhat atrophied. Scar tissue; cyst.	Fits reappeared in 3 months, again reappeared in 4 months; 2 fits subsequently.	Recovery. Recovery.	Less than 3 months after last operation.	Ibid., p. 31	Time of observation insufficient.
Trephine over arm centre on right side.	Ext. table fractured; small splinter of bone indenting dura; dura thickened; pia and brain yellow and œdematous.	About one month after operation there was recurrence.	Recovery; recurrence of fits.	Less than a year; fits frequent and severe as before operation.	Ibid., p. 35	Time of observation insufficient.
Trephine over upper third of motor area for right side.	Small white specks resembling miliary tubercles over pia and area for leg.	....	Death followed operation.	....	Ibid., p. 37	Death.
Trephine over area for right arm.	Subcortical cyst.	2 attacks of petit mal subsequently.	Recovery.	4 mos.—	Ibid., p. 38	Time of observation after operation insufficient.
Trephine over motor area for face and Broca's convolution.	Thickened dura; brain-substance replaced by connective tissue; pia œdematous	2 in the two subsequent months.	Recovery; recurrence of fits.	4 mos.—	Ibid., p. 40	No real improvement; time also insufficient.
Trephine over seat of fracture.	Depression of ext. table; dura, pia, and brain normal.	5 in subsequent four months; at last visit were as frequent and as severe as before operation.	No result.	4 mos. +	Ibid., p. 43	No improvement; time also insufficient.
Trephine over arm centre extending toward face and motor speech centres.	Nothing abnormal.	2 in subsequent six months.	Improvement; relapse; death.	6 mos. +	Ibid., p. 44	Personal observation ceased Dec. 1891, but it is stated that fits became more frequent, and death occurred November, 1892.
Evacuation of cyst; did not trephine.	Triangular defect in bone covered with thick connective tissue beneath which a cyst.	No spasms for one year, when they returned as before.	Recurrence of fits.	Not given	Ibid., p. 46	Author says in all probability cyst has filled again, another operation contemplated. Improvement; recurrence.

No	Reported by, or operator.	Date of trauma.	Interval between trauma and appearance of fits.	Character of fits.	Interval between and number of fits before operation.	Interval between appearance of fits and date of operation.	Date of operation.
27	Starr and Hartley.	1878, fall: extensive fracture of left parietal and frontal bones.	7 years	General, always began with turning of head to right; had an immediate right hemiplegia and motor aphasia which lasted one year after accident.	Irregular intervals; as many as 5 daily, but under bromides usually 1 in 3 weeks.	7 years	Oct. 18, 1892
28	Starr and McBurney	January, 1890, fall on left parietal region; no fracture.	"Soon" after	Always at night; closing of fingers and thumb of right hand, twitching; arm flexed and trembled; hand brought to face by shoulder movement; twitching right side of face; head slightly turned to right.	Frequent intervals.	2 years	Feb. 25, 1892
29	Starr and McBurney	Fell several years before; trephined one year previously; nothing founds; fits continued.	"Subsequently"	General, beginning in right hand.	Not given.	Not given	Not given
30	Starr and McBurney	1867, fall; scar over left parietal bone.	14 years	General, epigastric aura.	2 or 3 w'kly; at times as many as 5 daily.	9 years	March 8, 1890
31	Frank and Church	Sarcoma of middle and lower thirds of left motor area.	....	Spasm right index finger, wrist, hand and arm; general convulsion, loss of consciousness; pain right hand; increasing paralysis; leg weak; some aphasia.	Irregular intervals.	1 year	May 21, 1889
32	Keen	Nov. 1886, fall on right side of head; depressed fracture over middle ⅓ of right motor area.	6 months	Left fingers anæsthetic after injury; sudden vertigo followed by temporary paralysis of left hand.	Irregular intervals.	1 year	April 18, 1888
33	Keen	Not given; fall on left side of head; depressed fracture over arm centre of left side.	2 fits in 11 years, then became frequent.	Flexion of right hand, spasm of arm, general convulsion.	Frequent intervals.	......	Oct. 29, 1890
34	Keen	Not given; depressed fracture over lower parietal convolutions on right side.	2 years	General.	......	14 years	Nov. 21, 1890
35	Diller	Fall at 6 months; convulsions and left hemiplegias.	3½ years	Beginning in left arm, then face, then leg; unconsciousness; hemiplegia worse in arm; sensation diminished in arm	.....	7½ years	Jan. 9, 1891
36	Lloyd and Deaver	1869, struck on head.	5 years	Numbness and spasm in left hand and arm extending to face (left side); paresis of left hand and face followed; consciousness occasionally lost in an attack.	Frequent intervals.	14 years	June, 1888

Kind of operation.	Lesion found.	Interval between and number of fits after operation.	Result as stated by reporter.	Length of time under observation after operation.	Reference.	Remarks.
Trephine.	Depression opposite lower ⅓ of Rolandic fissure; skull thickened; dura adherent; pia thickened and clouded; brain rough and red; large number of capillaries; pia closely adher.	1 fit two months subsequently.	Recovery; recurrence of fits.	5 months	Ibid., p. 47	Improvement; recurrence. Time of observation after operation insufficient.
Trephine over middle ½ of Rolandic fissure.	Dura adherent to bone; small whitish plaque of connective tissue on pia; brain normal.	One month; in about 6 mos. fits changed to a somnambulistic character	Recovery; return of attacks.	1 year+	Ibid., p. 49	Dec. 20, 1892, put on bromides, and in March, 1893, cessation of observation, "entirely free from attacks." Slight improvement. Return of fits; ceased under bromides.
Trephine over seat of fracture; frontal bone anterior to motor region.	Fracture of external table; brain, pia, and dura normal.	Not given.	No result.	Not given	Ibid., p 52	No improvement.
Trephine over site of scar.	Fracture of external table; dura, pia, and brain appeared normal.	23 days; as frequent as before operation.	No result.	1 yr. and 0 mos.	Ibid., p. 52	No improvement.
Trephine over left motor area, middle and lower thirds; brain excised 1½ in. in circumference, ½ in. deep.	Thick cicatricial mass on cortex; sarcoma,	Return of fits in three months.	......	....	Am. Journ. Sci., July, 1890	Improvement. Return of fits in three months.
Trephine over depressed fracture.	Spicule of bone projecting into brain; cyst; chronic meningo-encephalitis.	None for four months after operation.	Temporary paralysis of hand; no fits for four months.	4 months	Ibid., Oct. 1888	Time of observation after operation insufficient.
Trephine; bone and membranes about depression removed; hard centre excised.	Projecting pieces of bone; brain disorganized and depressed.	None for eight months.	Paralysis and anæsthesia of hand gradually passing away.	8 months	Ibid., Sept. 1891	Time of observation after operation insufficient.
Adhesion between brain and scalp divided.	Bone deficient; brain adherent to scalp.	2 fits in subsequent two weeks, then ceased.	......	6 months	Ibid.	Time of observation after operation insufficient.
Trephine over motor area for arm, right side	Fissure in bone, cyst containing 3 oz. clear fluid under cortex.	No fits after operation.	Recovery.	43 days; patient died at expiration of this period.	Pittsb. Med. Rev., Nov. 1892	Cyst was drained; when drain was removed it filled again. Drain replaced for 40 days, when discharge became purulent, and patient died on 43d day.
Trephine over junction of middle and lower thirds of motor area; hand centre excised.	Brain normal.	Convulsions for three weeks, when they ceased.	Paralysis and anæsthesia in left hand permanent.	3 months	Am. Journ. Sci., Nov. 1888	Time of observation after operation insufficient.

No.	Reported by, or operator.	Date of trauma.	Interval between trauma and appearance of fits.	Character of fits.	Interval between and number of fits before operation.	Interval between appearance of fits and date of operation.	Date of operation.
37	Knapp and Post	1882, struck over right temple.	1 year —	Head turned to left, then convulsions of left side of face and neck; left arm; sometimes general convulsions follow.	4 or 5 daily.	8 years	May 1, 1891
38	Knapp and Post	1885, struck on head; depressed fracture over left second frontal convolution.	Severe and constant headache since the trauma	General; commence by turning of head and eyes to the right.	......	......	Nov. 1890
39	E. D. Fisher and J. W. Bryant	1878, slight depression of skull over hand centre of left side.	2 years	General	......	12 years	May, 1892
40	E. D. Fisher and Geo. Woolsey	Indefinite history of trauma.	....	Commence with sensory aura in fingers of left hand, and passing toward face, after which loss of consciousness and general convulsions.	......	......	May, 1892
41	E. D. Fisher and J. E. Kelly	1887, injury to head; depression of parietal bone behind motor area; side not given.	1 year	Slight at first, later complete seizures.	7 attacks; 1 monthly: slight ones daily.	5 years	Mar. 1892
42	E. D. Fisher and J. E. Kelly	Not given.	Not given	Almost continuous and usually limited to left side.	Not given.	Not given	June, 1892
43	E. D. Fisher and J. E. Kelly	1878, injury to head from fall; depression over left frontal bone at margin of hair.	Not given	Very frequent; general.	Not given.	Not given	May, 1892
44	Morrison	No trauma.	....	At first slight, later severe; beginning with face and head turning to right.	......	6 years	Aug. 1891

SUMMARY.

Cured	2
No improvement	10
Death due to operation	3
Improvement, return of fits: malignant brain disease	1
Cases in which there was a cessation of fits one year after operation, but in which bromides were used	2
Temporary improvement; time of observation, however, not given	3
Cases in which time of observation after operation is insufficient to make them of value in statistical tables	23
Grand total	44

Kind of operation.	Lesion found.	Interval between and number of fits after operation.	Result as stated by reporter.	Length of time under observation after operation.	Reference.	Remarks.
Trephine over 2d frontal convolution, site of scar.	Dura normal; pia œdematous and opaque; Brain markedly bluish.	....	Recurrence of fits as before.	6 months	Boston Med. and Surg. Journ., Jan. 7, 1891	No improvement.
Trephine.	Bone thickened and adherent to dura, dura adherent to brain; dura and portion of brain substance excised.	....	Continuance of convulsions.	4 months	Ibid.	No improvement.
Trephine over seat of depression.	No fracture or adhesions.	....	Attacks first increased in frequency, then decreased, and finally became as before operation.	....	Brain Surg., Starr, p. 62	No improvement. Patient intemperate; insane.
Trephine over centre for left arm; tumor opened and then united.	Nothing abnormal.	....	Attacks as frequent as before, but no longer commenced on left side.	....	Ibid., p. 63	This patient showed signs of dementia. Mental condition did not improve after operation. No improvement.
Trephine.	Inner table pressing on dura; no internal fracture; dura not opened.	....	Up to date slight attacks of dizziness; no seizures; Date not given.	Not given	Ibid., p. 63	Temporary improvement. Time of observat'n after operation not given.
Trephine over right motor area.	Nothing abnormal; part of hand centre excised.	....	Death six hours after operation.	....	Ibid., p. 64	Death.
Trephine over site of depression.	Nothing abnormal.	....	Mental condition improved; attacks less frequent, but later became as before operation.	Not given	Ibid., p. 64	Patient demented and addicted to masturbation. Temporary improvement.
Trephine over junction of temporal ridge and coronal suture.	Dura and brain normal.	Fits returned three weeks after operation.	No result.	....	Ibid., p. 57	No improvement.

NOTE.—In those cases classed as "cured," observation was kept up for more than three years. All cases which were not under observation for at least one year, and in which there was neither death nor non-improvement, have been classified as "cases in which time of observation after operation is insufficient to make them of value in statistical tables." In most of them, however, there was an improvement, usually temporary.

From this summary it will appear that there were only two cases out of the forty-four to which the word "cured" can accurately be applied, although, of course, this conclusion would shock many of the narrators whose time of observation of the patient after the operation has not been sufficient, in my opinion, to render any conclusion warrantable.

In peripheral irritation a valuable adjunct to medication is the removal of the irritant, even though only temporary improvement may be secured. Phimosis or an adherent prepuce should be rectified, and an irritable clitoris should either be treated by soothing applications or by a careful application of a 60 per cent. solution of nitrate of silver. Vaginitis should be carefully treated, if it exists. The removal of the insufficiency of ocular muscles is, in my opinion, quite useless, but marked errors of refraction should be remedied. If so-called laryngeal vertigo or epilepsy exists, the laryngeal tumor giving rise to it should be removed. As the difficulty of eradicating the epileptic tendency is very great, surgical measures should always be accompanied by medication.

Children afflicted with epilepsy are very apt to be spoiled, and, as a result of injudicious petting and indulgence, they often become almost savage in their dispositions. They should be treated with the utmost firmness and care, either by a trained nurse or in many ways which the good sense of the physician or parents may suggest. In cases of this kind moral treatment is often wonderful in its effect for good.

If migraine exists in conjunction with epilepsy it should be carefully treated. (See Chapter XXXII.)

If a periodicity is manifested, careful attention should be paid to the treatment at such periods. The patient should be kept especially quiet, in many cases it being advisable to put them to bed at the time of the expected attack, and either add to the medication or increase the quantity already being administered. A dose at bedtime may be all that is necessary in nocturnal cases. The attacks may be diverted from their usual time of recurrence in some of these cases, so that an attack may occur in the daytime or some other time of the month. In such cases attention should be paid to the periods, and the treatment kept up continuously.

Without doubt the most valuable means of treatment at our disposal are the bromides. They should be given in large doses, unless the idiosyncrasy of the patient prevents. As a rule, epileptic patients bear the bromides well, but in some instances this is not the case, as in some exceptional circumstances the disease seems to be aggravated by the use of bromides; in some an ounce may be administered without ill effect, and in others a dose of 10 grains will produce a collapse. In consideration of all these facts, my plan of treatment is as follows: The bromide is administered in doses of 10 grains each, three times a day, unless marked periodicity is manifested, in which case it is given in proportionately large doses. My usual prescription is as follows:

℞.—Potass. brom.	℥ss.
Aquæ	℥iv.—M.

S.—Teaspoonful three times daily, after meals, in half-tumbler of water.

After about a week of treatment by the smaller dose I increase the dose to 15 grains three times a day, unless bromism has taken place, or improvement is manifest. In case the patient bears the bromide well, but does not improve, I still further increase the dose to 30 or 40 grains daily. If bromism is manifested, but no improvement in the epilepsy, I conjoin 5 grains of the bromide of sodium with the bromide of potassium, as in the following prescription:

℞.—Potass. brom. . . . . ʒss.
Sodæ brom. . . . . ʒij.
Aquæ . . . . ℥iv.—M.
S.—Teaspoonful three times daily in half-tumbler of water.

The two bromides in combination will often have an increased effect upon the disease without increasing the constitutional effect of the drug. If the patient is satisfactorily brought under control of the bromide, this dosage should be kept up. In case of cease of improvement, or relapses of the attacks, I have recourse to a laxative, a brisk cathartic, a change of scene, or combination of belladonna, borax, or hyoscyamine, with the bromide treatment already given. I employ these latter means for a short time only, discontinuing their use gradually. I use the following prescriptions:

℞.—Potass. brom. } . . . . āā ʒss.
Sodæ borat. }
Aquæ . . . . ℥iv.—M.
S.—Teaspoonful t. i. d., after meals, in half-tumbler of water.

Or.

℞.—Potass. brom. }
Sodæ borat. } . . . . āā ʒss.
Bellad. ex. fld. }
Aquæ . . . . ℥iv.—M.
S.—Teaspoonful t. i. d., after meals, in half-tumbler of water.

Merck's preparation of hyoscamine should be used, in tablet triturate (gr. [illegible] night and morning, alone). Some authors consider the loss of uvular reflex of great importance, and the ability to tickle the throat of a patient without producing nausea is accepted as a symptom that sufficient bromide has been given. I place but little stress upon this, however, as I have known [illegible] grow worse when this reflex had been abolished; improvement has [illegible] in others when it was still present, and in others still the bromide [illegible] borne in increasing doses, after the cessation of the reflex. As the [illegible] of ammonium has not proven useful in my hands except as an adju[illegible] I have confined my application of the bromides to those of potassium [illegible]. Moderate doses of Fowler's solution of arsenic, gtt. ij.–iij., three [illegible] day, in a wine-glass of water, will usually overcome the acne, which [illegible] considerable. I believe that cases which do not yield to the bro[illegible]tment will fail to improve by any other, except that, in my experi[illegible] few cases will do well with borax when the bromide has failed. [illegible] maximum-dose treatment in alternation with the treatment just [illegible] occasionally very useful. This consists of doses of two or three [illegible] of bromide of potassium every second or third morning, increasing [illegible] to four drachms every fourth morning, and six drachms every fifth [illegible] these doses being given after breakfast in a tumblerful of water, as [illegible] pain and vomiting may be caused if they are not well diluted. He [illegible] increase the dose beyond that which produces transient dulness and [illegible]. A great variability is shown in the susceptibility of patients to [illegible], some being unable to bear more than four drachms, whilst no [illegible] symptoms will be induced in others by the adminis [illegible] I have myself observed. A period of six or seven w [illegible] in this treatment, reaching the maximum dose in two [illegible] gradually diminishing the doses during the remainder o [illegible] the patient may be left without treatment for several [illegible] I have never seen such long periods of freedom fi [illegible] claims.

Except as an adjuvant, belladonna has proven of little efficacy in my h
One or two minims of the drug should be given three times a day, usin
fluid extract, and carefully noting the effects. In some cases borax is as
ful as the bromide treatment, though this is not usual. This treatment sh
be carefully tried in all cases where the bromides disagree with the patie
fail in effect. The dose should be ten to twenty grains three times a da
a half-tumbler of water.

The salts of strontium, the lactate, and the bromide, in the same dosa
the borax, have been spoken of in the last few years as being of conside
efficacy in the treatment of epilepsy. I only mention the fact, as I
myself had no experience with them. An attack which is preceded b
aura may sometimes be arrested by nitrite of amyl; but as this warning oc
very seldom, I have almost abandoned the use of the drug. Preparatio
zinc, which were much in favor twenty years ago, have proven of little v
in my hands.

CHAPTER XII.

GENERAL DISEASES OF THE BRAIN.

ARRESTED DEVELOPMENT, MALFORMATIONS, HYDROCEPHALUS.

By N. E. BRILL, M.D.

The brain is a frequent seat of deviations from the laws of normal growth. These deviations may present either an arrest of the development of the brain or of some of its anatomical divisions, or an overgrowth of the brain as a whole or of some part of it. The defects and malformations resulting from these deviations may be limited in area or may be generally distributed throughout the entire central nervous organ; thus a fœtus may present an entire absence of the brain, a condition to which the term anencephalia is applied, or only an absence of the cerebellum or of the corpus callosum. Again, instead of an absence of this organ or one of its component anatomical divisions, there may be present an anomalous condition of the growth of any of these, such as an abnormal smallness of the brain or of some cerebral lobe and its convolutions, a hypoplasia, to which the term micrencephalia and microgyria have been respectively given. Again, these deviations and defects may vary in degree from a general anencephalia to a simple convolutional atypy.

That the brain should be so frequently the seat of these malformations and abnormalities is not to be wondered at. For preponderating as it does, not only in mass, but in rapidity of growth during embryonic life, it is the organ most exposed to the shocks of those traumatic and pathological disturbances which may arise during the early months of intra-uterine life. The disturbing influences may arise from within or without, and to them must be ascribed the causes for the various defects in growth which appear at the birth of the fœtus.

Many of these developmental defects are inconsistent with continued extra-uterine life. In these cases a fœtus is born which may or may not breathe. It may be said, as a general rule, that when the developmental defect is a gross one the fœtus is non-viable. These gross defects and results of developmental arrests have a pathological rather than a physiological interest.

The general causes of cerebral malformations are to be sought, *first*, in a perversion of growth of the ovum, due to inheritance or to an original defect in either of the parental contributions to the development of the embryo; *second*, in a pathological disturbance of the mesoblastic or epiblastic layers of the embryo; or, *third*, in a direct disturbance of the fœtus after the development of the neural tube;—the last factor may be the result of a psychic disturbance on the part of the mother, *e. g.*, a shock or fright (maternal impressions), a traumatic or other disturbance to the mother operating from without;—or, *fourth*, to certain diseases of the fœtus which may develop in utero or after birth. Of course, we are unable to determine the existence of an original defect in the developmental units, or even a pathological disturbance in the blastodermic membranes. We must infer, however, that such conditions must have existed to have produced the malformations and arrests of development, which are so frequently found, on the ground that no other known causes may be reasonably assumed. In fact, certain cerebral malformations and cerebral developmental arrests are so constantly occurring that they assume a fixed and definite type. Where this is the case, it may be laid down as a general rule that the causes producing such changes are to be sought among the first two enumerated. When an atypical malformation occurs, either due to an arrest of development or an overgrowth of some cerebral element, the cause must be looked for either in a disease or some pathological condition of the embryo or fœtus, or in a disturbing factor which has influenced the mother and has been indirectly reflected upon the fœtus.

The character and the degree of cerebral malformation depend also upon the nature of the disturbing factor and upon the period of gestation at which the latter operates. Thus, such an influence operating before or during the formation of the neural tube will be attended by a greater amount of cerebral developmental arrest than at a period when the hemispheric vesicles have already been developed. In the former case an anencephalia may be produced; in the latter, some other forms of malformation which may preserve some portion of the original hemispheres. Should the disturbing influence be an original defect on the part of one or both parents, such as an atypical formation of the cerebral convolutions, a microgyria, or a micrencephalia, such defects may be transmitted to the offspring.

Authors generally have neglected to give the proper weight to disturbing psychical factors in producing the defects of which this chapter treats. These factors are not infrequent causes of the production of cerebral monstrosities. Fright, fear, or a persistence of conceptional visual impressions on the part of the pregnant mother which excite any of these emotions in her are occasionally attended by developmental cerebral defects in the offspring.

The literature of the subject abounds in cases of these defects ascribed to this cause. Encephalocele, microcephalia, cyclopia, and other malformations and defects have all been reported as the result of maternal shock upon the fœtus in utero.

Any deviation from the fixed type of a normal brain is a malformation. When this term is applied to cerebral pathology it includes all the various forms of deviations, to wit: cerebral hyperplasia, cerebral hypoplasia, and cerebral heteroplasia.

Cerebral hypoplasia includes the following pathological conditions: 1. *Anencephalia,* or absence of the brain. This condition is always the result of an arrested development, and should not include that pathological condition of the brain due to a hydrocephalus internus, in which, by reason of the pressure from the enormous quantity of fluid secreted within the cerebral ventricles, the substance of the brain becomes atrophic, and finally coalesces with the cerebral meninges, so that it is difficult to separate them, or even to determine by gross examination that any cerebral cortex is left. In true anencephalia there is almost always an involvement of the structures of the mesoblast formation, the bones of the cranial vault, which may be wholly or in part wanting. 2. *Micrencephalia,* small or rudimentary brain. Under this head we must include that condition of atrophic or rudimentary development of the cerebral convolutions, which is called microgyria. Micrencephalia and microgyria usually, if not invariably, accompany each other, so that a micrencephalic brain usually shows an atrophic or rudimentary development of the convolutions. 3. *Absence, or original rudimentary, or* *atrophic development of any of the component parts of the brain,* such as the cerebellum, the corpus callosum, a cerebral lobe.

ANENCEPHALIA. This is a condition which is usually associated with a defective development of the meninges and cranial vault. Pathologists differentiate between two degrees of this condition, total anencephalia and partial anencephalia. The former is that form of cerebral malformation in which the brain is totally absent, as is also the cranial vault. The cavity which the brain was destined to fill is usually occupied by a mass of dark vascular membranous tissue. Partial anencephalia is a condition in which some brain tissue is present, and some definite cerebral substance can be determined. It may or may not be associated with a defective cranium. The partial anencephalies are alone of interest to the neurologist, for, unlike the total, some are consistent with life and present certain definite indications of their existence which can lead one to form a diagnosis of the condition of the brain *intra-vitam.* While total anencephalia is due to a *complete* arrest of the development of the hemispheres, partial anencephalia is the result of merely a *defect* in their development. The former is produced by a pathological disturbance of the forebrain of the embryo, which prevents the evolution of the hemispheres; the latter presupposes a defect of certain cells in the forebrain, which preclude their complete evolution. Thus partial anencephalia may vary in degree and extent. There may be an almost complete absence of both hemispheres, an entire absence of one hemisphere, or a defect in one or both hemispheres. The last defect may be a loss of brain substance, leaving a cavity, sometimes communicating with the ventricles within the hemispheres and sometimes with the subarachnoidean space. This last condition was first described by Heschl in 1859, and was called by him *porencephalia.* (See also Chapter XVII.)

Investigators have recently been devoting their attention to the etiology of anencephalia. It is the opinion of many that intra-uterine disturbances are the most important factors in its production. There can be no doubt that irrespective of heredity and primary defects in the ovum, both of which are determining factors in the production of anencephalia, pathological conditions of the amnion are the most prolific cause for this cerebral defect. Long ago Dareste[1] and Geoffroy-St. Hilaire called attention to the fact that a small amnion exercised an injurious influence on the growth of the embryo. In fact, the latter regards arrest of development as the result solely of mechanical causes.[2]

Adhesion of the amnion to the head end of the embryo must exercise a causative factor in the production of anencephalia. The anencephalia will be complete or partial according to the time of embryonic development that this adhesion takes place and according to the condition of adhesion. Should the adhesion take place before the cerebral vesicles are formed, the constriction at the cephalic end and the pathological changes which the cells there undergo will prevent the evolution of the hemispheric vesicles, and a total anencephalia will be produced. Should the adhesion be small in extent and involve but a limited area partial anencephalia will ensue. It seems to me that this is the only explanation of the development of that form of partial anencephalia in which one hemisphere is developed while the other is absent.

Almost all who have been investigating teratological phenomena have come to the conclusion that an abnormal constriction of the amnion at the head end of the embryo gives rise to the forms of arrested development shown in anencephalia, exencephalia, cyclopia, and arhinencephalia. It would appear to me that an excessive amount of amniotic fluid could likewise exercise a restrictive influence and determine the development of cerebral malformations.

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[1] Dareste: Recherches sur la production artificielle des monstruosités, Paris, 1877.

[2] Geoffroy-St. Hilaire: Hist. gén. et partic. des anomalies de l'organisation chez l'homme et les animaux. Paris, 1832-1837.

Pressure exercised on the fœtus in utero by pathological conditions on the part of the mother may give rise to adhesions between the amnion and neural tube, and serve as indirect causes for these conditions.

Attention has recently been paid to the artificial production of malformations. Thus L. Gerlach, Fol, Warynsky, Richter, and Roux have instituted experiments on the ova of chickens and frogs with such disturbing factors as variations of temperature, injuries to the ovum, changes in the position of the ovum, removal of parts of the ovum, etc., and have produced monstrosities as a result. These experiments serve to prove that a normal cerebral development requires a normal ovum perfectly adapted to its environments, one which receives no shock or trauma, and whose evolution proceeds without any pathological conditions arising in its integral units.

The question of discriminating between the pathological features of partial anencephalia and porencephalia has not yet been settled. It is certain that every porencephalia is a partial anencephalia in a general sense, and as such should be described in connection with the latter developmental defect. Irrespective of the causes enumerated, partial anencephalia may be the result of certain pathological factors produced by morbid changes occurring in the brain of the fœtus toward the end of uterine life, and in the brain of the child immediately or soon after birth. These morbid changes may be the result of a thrombosis or embolism of some cerebral vessel, resulting in the formation of a focal softening, and subsequent disintegration of the softened area, with serous transudation into the cavity produced by the softening. Cerebral hemorrhage may likewise give rise to the same result. Encephalitis is also a cause for these defects and hiatuses. Traumatism, either instrumental or otherwise, during parturition must not be overlooked as a cause for this condition.

One of the most frequent and important factors in the production of partial anencephalia is a disease of the ependyma of the ventricles, an *ependymitis*, which results in the formation of subependymal morbid changes and the transudation of large quantities of fluid into the ventricular cavities of the brain.

Early obliteration of cerebral vessels, either from disease of the cerebral vessels of the fœtus or from a fault in the development of the vascular system of the embryo, may also be a cause of anencephalia.

Encephalitis is regarded by most authors as a potent factor in the production of cerebral defects like porencephalia. In fact, some are inclined to believe that the vast majority of cases showing partial anencephalia or porencephalia are the sole results of this pathological condition.

These causes may operate on the fœtal brain as well as on the brain of the newborn, in the former case producing a congenital porencephalia, and in the latter a so-called acquired porencephalia.

Syphilis on the part of the parent and its transmission to the fœtus, producing changes in the fœtal cerebral vessels favoring the production of thrombotic and necrotic changes in the brain, must not be overlooked as an important factor in the production of partial anencephalia and porencephalia.

It is thus apparent that a more rational classification of anencephalia would be into true anencephalia and spurious anencephalia; the former being confined to the cerebral defects, due to changes inherent in the ovum and embryo, the result of hereditary or transmitted influences; the latter, to all pathological causes arising from within or without the fœtus. Under the former classification, of total and partial, the partial anencephalies are bound to become confounded with those defects due to pathological causes, which ought not to be considered as originial defects in development. Thus, a defect due to a thrombosis, to a hemorrhage, to an encephalitis, or to an ependymitis should not be called an anencephalia; for, before the pathological process giving rise to this defect was established, the cerebrum may have been perfectly normal. The resulting defect is in such cases, therefore, not an arrest of development, but a destructive change in an already developed brain.

PORENCEPHALIA. Inasmuch as the signs of partial anencephalia and porencephalia have much in common, the latter will be described here, but it should be remembered that, notwithstanding the fact that pathologists differ as to what shall constitute a porencephalia, when it is spoken of in this chapter it is meant to signify a loss or defect of brain substance, due to pathological causes arising in the fœtal brain, and which shows signs of its presence from birth; hence, a congenital defect in the brain. Many pathologists regard the absence of a hemisphere as a porencephalia. Others regard the defects due to hemorrhage, or thrombosis, or to hydrocephalus internus, whether produced before birth or during the first two years of infantile life, as a porencephalia. We insist, however, that such an acceptation of the term is unscientific and unphilosophical; its use should be restricted to congenital pathological defects only.

Since Heschl[1] first described the pathology of this form of cerebral malformations much attention has been paid to the subject. Kundrat,[2] in an able monograph in which he called general attention to Heschl's case, collated a number of his own, giving minute details of all the pathological findings in each case. This monograph has been the basis of most all subsequent contributions to the subject.

Heschl, who suggested the term, confined it to those cases of defects in the cerebrum which presented an absence of cortical and subcortical cerebral tissue, either communicating with the arachnoidal space or shut off from it, the loss of tissue extending down more or less deeply into the cerebrum, even as far as the endyma of the ventricles, and sometimes communicating with an opening into the ventricular cavities. Thus, there resulted a pore or funnel-shaped depression in the cerebrum, hence the name.

The defect is confined to the convexity of the hemispheres or to the Island of Reil. The mesal surface of the hemispheres is never involved, and no case, to my knowledge, has as yet been reported in which that surface has been implicated.

In porencephalia there is usually associated a developmental defect in the hemisphere which contains the lesion. This hemisphere is usually smaller, its convolutions and fissures atypical. This relation of convolutions and fissures exists in most cases. In exceedingly few cases can no disturbance of the cerebral convolutional architecture be determined. In congenital porencephalia there is usually a radiating distribution of the convolutions and fissures about the defect. In fact, Kundrat believes that such a relation of convolution and fissure to the defect is the characteßistic feature which differentiates a congenital porencephalia from one which has developed after birth. In no case of porencephalia are the primary fissures absent. They may be involved in the defect, but some portion of them remains. This would go to prove that the pathological developmental defect producing the porencephalia must occur after the fifth month of gestation. The primary fissures are not infrequently changed in their direction, a result which may be directly due to the progress of the associated developmental defect; but their presence has been established in every well-investigated case. The secondary fissures are frequently absent.

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[1] Heschl: Prager Vierteljahresschr., 1859 and 1868. [2] Kundrat: Die Porencephalie, Graz, 1882.

When the porencephalia is intra-cerebral the convolutions over the lesion are smaller and less rounded than normal. They sink slightly below the level of the surrounding gyri, leaving a depression on the surface. Should the porencephalia communicate with the subarachnoidean space, the gyri immediately adjacent to the defect present anomalous conditions. Since they are usually implicated in the pathological process producing the porencephalia, histological changes can frequently be determined in them; thus, a diminution of the neuroglia and atrophy of the nerve fibrillæ are not infrequent. The gyri themselves end with rounded edges, which sink into the cavity of the defect, and becoming gradually thinner, merge with the subcortical substance. In some cases, owing to the loss of sub-cortical tissue, and the further development of the cortex, the latter grows over the defect and leaves only a sinuous track—a linear opening or slit—on the cerebral surface, which, if not closely examined, appears merely to be an unusually wide sulcus.

The relation of the cerebral meninges to the porencephalia is important, and is considered by Kundrat to be characteristic. He considers it a differential mark between a true (congenital) and spurious (acquired) porencephalia. In the former case the pia, where the defect is covered by cortex and does not communicate with the subarachnoidean space, follows closely the depression of the hemisphere, which is arched over by the tense arachnoid. In those cases where the defect communicates with the sub-arachnoid space, the pia dips down into the cavity for some distance, whereas the arachnoid ends abruptly at or near the margins. When the cavity communicates with the ventricle the pia ends almost at the ependymal lining of the ventricle, the ependyma being itself thickened. In the acquired cases the pia does not follow the depression, but ends abruptly at the margin of the depression. Sometimes the cavity in these cases is filled by a vascular connective-tissue formation which imitates in appearance a thickened pia, but which seems to have no connection with it.

In all cases where the defect communicates with the ventricle the latter is found to be dilated, its endymal covering thickened, and its surface to have a granular appearance. In fact, dilatation of the lateral ventricle is one of the most constant accompaniments of porencephalia, and is sometimes found in cases when the defect does not communicate with the ventricle.

The seat of the porencephalia varies, its region of selection being the convexity of the parietal lobe. In ninety-six cases examined by Audry[1] it was found in both hemispheres in 32 cases, in the left hemisphere in 38 cases, in the right hemisphere in 26 cases.

The lobes involved in the single defect showed the following: Parietal lobe, 17 cases; frontal lobe, 7 cases; temporal lobe, 4 cases; occipital lobe, 3 cases.

In the cases examined by Kundrat the temporal lobe was found to be more frequently the site of the defect than the frontal lobe, and the parietal to be most frequently of all affected.

Corresponding with the gross changes, microscopical examination shows an atrophy and disintegration of the tracts connected with the defective area. When the area involved is in the motor region of the cortex the pyramidal tract is rudimentary, and when traced through the internal capsule, pes, pons, and medulla, shows a marked contrast in size to its opposite fellow. (See Fig. 69.) In these cases the isthmus cerebri is asymmetrical, the affected pyramid in the medulla being either not at all elevated or even not recognizable as a distinct formation; in some cases—those in which the entire motor area of the cortex is destroyed—the pyramidal tract is entirely absent. The association fibres of the cortex cerebri show the same atrophic and rudimentary development. It is owing partly to these associated changes in the subcortical connecting strands and partly to the original loss of the cerebral substance in the pathological defect that the affected hemisphere is smaller than the non-affected one. Asymmetry between the two hemispheres is more marked in the congenital cases. In these cases, too, the basal ganglia on the affected side participate in the defective development, the thalamus especially being smaller than normal, as is also the lenticular nucleus.

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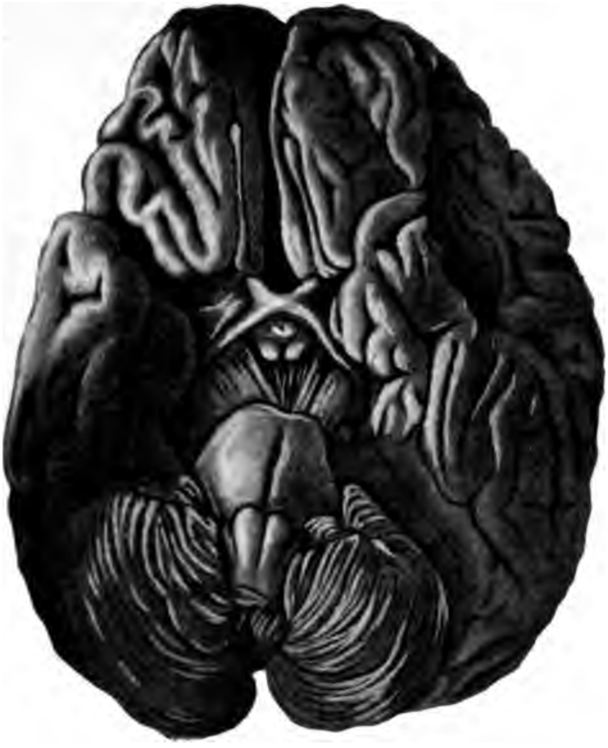
[1] Revue de Méd., 1888, viii. 6, pp. 462-467, 583.

FIG. 69.



Large porencephalic defect in the left insula. View of the brain from the base. Secondary degeneration of the left crus and pons. (After KUNDRAT).

The septum lucidum is often absent, and especially so in those cases where there is a dilatation of the lateral ventricle.

The cerebellum, in a few cases, has been found to be smaller than normal, and also asymmetrical, the asymmetry being confined to the lobe on the same side as the defect in the cerebrum.

The cranium itself stands usually in direct relationship with the lesion. In most cases there is a marked asymmetry. In some cases where the porencephalia involves a large area of the cerebrum there may be no cranial asymmetry, whereas, on the other hand, slight porencephalic defects are attended by a degree of cranial asymmetry beyond proportion to the cerebral defect. Where the cranial asymmetry does not correspond with the cerebral defect, the porencephalia, it may be assumed, occurred late in fœtal l after birth. In the congenital cases there seems to be a direct relationshi tween the degree of cranial asymmetry and the extent of the cerebral l

The character of the asymmetry of the skull varies. In some cases tl volved portion is distended—giving the appearance of a hydrocephalic —while in others it is microcephalic. The latter cases are more num by far than the former. The face very often participates in the asymm

Symptoms. The symptoms of partial anencephalia and porencephali much alike.

Total anencephalia is a condition which does not permit of life.

The character of the symptoms varies according to the seat of the le The most common signs are idiocy or imbecility, hemiplegia, mutism, formation, and contractures of the extremities. In some cases associated these signs is strabismus.

A child who from birth shows a hemiplegia, with or without contrac of the paralyzed side, who, as childhood advances, shows no evidence o cording impressions and makes no progress in intelligence, who does acquire the use of speech, who has a cranial deformity, who shows a pe sion of the emotions, who dribbles saliva, and cannot learn the use of a v to hold its excreta, presents the usual picture of a congenital porenceph

If the child should be born without any apparent symptoms of this nat and if within the first two years of infant life it develops symptoms sin to the above, especially if a history of traumatism can be adduced, o hereditary syphilis, such a child may be said to have acquired porenceph

The most constant of all the symptoms is paralysis. It is present in congenital as well as in the acquired forms, and is a necessary result of destructive process in the brain. Rarely are all the extremities the sea paralysis. The usual form is a hemiplegia contralateralis, which, if seconc degeneration involve the pyramidal tract, is accompanied by contractur the paralyzed limbs. Contracture may also be the result of a defectiv developed pyramidal tract.

Independent of the paralysis, there is often present a malformation of extremities, sometimes in the paralyzed, sometimes in the unparalyzed lin and not infrequently in both. This is present in some form in almost al the congenital cases of porencephalia, and is rare in the post-natal form.

Mutism, or inability to express articulate speech, is not infrequent in congenital form. It is rare in cases of acquired porencephalia. The de in the acquisition of speech may be absolute, nothing but inarticulate c being expressed at times of emotional excitement; or speech may be imp fectly developed, so that the child may be able to express itself in a spe way to those who are accustomed to be about it. It may learn the use some words and phrases. Inability to speak in these cases stands in dir relationship to the psychical disturbance, so that where the idiocy is extre speech is entirely wanting.

Deafness is sometimes present, and is usually found in those cases wh the lesion affects the temporal lobe. When associated with mutism it ser as an additional factor in the production of absolute loss of the power acquiring articulate speech.

Blindness exists only in a few cases, and is found where the porencepha involves a great area of the cerebral hemispheres. It may be the result the cerebral lesion or of an associated rudimentary development of the op tracts.

Strabismus and insufficiency of the ocular muscles are not infreque accompaniments of this affection.

Idiocy is generally present in the congenital cases, and depends for existence on the defect in development of the brain which is associated with the original lesion. It is extremely rare in the acquired cases, the latter producing a lesser psychical disturbance, manifesting itself in an imbecility. The larger the defect the more pronounced will be the mental disturbance, and this is especially so in those cases in which the porencephalia is produced during fœtal life. Every degree of imbecility and idiocy may be presented in the various cases. Thus an absolute absence of all mental life may be present in some of the congenital cases, the afflicted individual showing no emotions, uttering inarticulate cries, passing his urine and feces without knowledge of the act, registering no impressions, not even acquiring the instinct of preservation, apparently only the automatic nervous functions being in operation.

Not infrequently associated with this condition is epilepsy. Convulsions and epileptiform attacks increase the mental hebetude and add horror to the lives of these poor unfortunates. (See also Chapter XVII.)

MICRENCEPHALIA AND MICROCEPHALIA. In the same manner that there may be an *absence* of development of the entire brain or of any of its component parts, an *arrested* or a *defective* development of the brain or any part of it may occur. Thus the brain entirely, or one of its hemispheres or lobes, may present a greater or less diminution in size.

The term micrencephalic is given to that brain which does not approach in weight the lowest standard of weight in man, as well as to a brain which is much diminished in size in some or all of its component parts. When the brain vault is similarly diminished in an individual that person is said to have a microcephalic cranium. The two conditions are usually associated. The diminution in size of the brain depends not only on a defective development of the cortical and subcortical elements, but also on a general arrest of development of the cerebral vesicles after the fifth month of gestation. This arrest, though generally distributed in area over the hemispheres, only partially affects the neural elements. As a result, mostly all of the gross anatomical elements of the cerebrum may present themselves merely diminished in size, and the micrencephalia may represent a miniature brain with small gyri and shallow sulci; or the brain may be fully developed in certain parts and defectively so in others, giving an appearance of a partial atrophy of the brain limited to certain lobes. The distinction between an atrophy of the brain and the results of arrested cerebral development should, in this connection, be always borne in mind; for micrencephalia may be the result of either cause, or both combined. The retrogressive changes occurring in a cerebrum not yet fully developed, due to some pathological process, and resulting in an atrophy of the affected portion of the brain, may itself be the essential factor in the production of an arrest of development in other parts of the same cerebrum. It thus becomes apparent that while both defects may be productive of micrencephalia, this condition may be the result of either cause independently.

Micrencephalia is but a form of partial anencephalia, and is dependent for its production on similar or analogous causes to those which produce the latter. A true micrencephalia is always the result of an arrested development, which may affect the entire cerebrum or some part thereof, and which is due to either an original defect in the epiblast of the ovum or to some acquired pathological process in the fœtal brain.

There is a form of micrencephalia which is the result of mechanical causes. This form is associated with a premature synostosis of the cranial bones. The effect of this deviation from normal cranial growth is to produce a hindrance to the antero-posterior or to the lateral growth of the cranium, or to both. The cranium remains contracted and small, the hemispheres beneath have no room to expand in their growth, and a small or micrencephalic brain is result. This condition can only result when all the sutures of the cran are involved in this pathological condition. If only some of the cranial b be affected, and premature junction of only some of the sutures result compensatory expansion and growth of the other unaffected bones takes pl and the skull and brain become developed in length or breadth, as the maybe, beyond the normal, producing a markedly dolicocephalic or brac cephalic skull.

Virchow[1] classifies the following cranial abnormalities dependent u general or partial synostosis of the cranial sutures:

A. Simple microcephalia may be produced by general premature synost of all the cranial sutures.

B. Dolicocephalic—long heads.
  - *a.* Superior middle synostosis.
    - α. Simple dolicocephali (synostosis of sagittal suture).
    - β. Sphenocephali, wedge heads (synostosis of the sagittal sut and compensatory growth in the region of the large fontanell
  - *b.* Inferior lateral synostosis.
    - α. Leptocephali, small heads (synostosis of the frontal and sp noid.)
    - β. Klinocephali, saddle heads (synostosis of the parietal a sphenoid or temporal bones).
  - *c.* Fœtal synostosis of the halves of the frontal.
    - α. Trigonocephali, forehead small and cuneiform; head, wh viewed from above, presents a triangular shape.

C. Brachycephali, short heads.
  - *a.* Posterior synostosis.
    - α. Pachycephali, thick heads (synostosis of parietal and occipita
    - β Oxycephali, pointed head (synostosis of the parietal with t occipital and temporal and compensatory growths of the ant rior fontanelle region).
  - *b.* Superior, anterior, and lateral synostosis.
    - α. Platycephali, flat heads (extensive synostosis of frontal a parietal).
    - β Trochocephali, round heads (partial synostosis of frontal an parietal in the middle half of the coronal suture.)
    - γ. Plagiocephali, oblique heads (one-sided synostosis of the fro tal and parietal).
  - *c.* Inferior middle synostosis.
    - α. Simple brachycephali (early synostosis of sphenoid and crani base).

The micrencephalia produced by premature synostosis of the crani bones presents usually a brain generally diminished in size—a miniature brai in which all the elements are small, the primary convolutions and sulc as a usual thing typical in form and direction, but interrupted, smaller, an less deep than normal. In these cases the convolutions appear to be flatten by the early pressure exerted upon the growing hemispheres, and the sec ondary gyri are less distinctly marked or absent.

The cranial capacity of microcephalic heads varies according to the ag of the subject. Carl Vogt, who examined carefully this measure in 3 crania, of which 25 per cent. were those of females, gave the followin average: The youngest cranium examined was one of a child who die after birth, the oldest one belonging to a person who lived 44 years. Th average capacity of these crania in children between the ages of five and fifteen years was 382 c.cm., in microcephalic adults 441 c.cm. In other words, the former represented about the normal cranial capacity of a child of six months of age, the latter that of a two to two-and-a-half-year-old child.

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[1] Virchow: Ges. Abhandl., Frankfurt, a. M., 1856, p 901.

These crania show a reduction in size in all measurements. The minimum standard of the circumference of a normal cranium is placed at about 43 cm. Crania whose circumference is smaller than this are considered as microcephalic.

The weight of micrencephalic brains varies to an almost extreme degree. A brain which is below the standard of the minimum weight in man, 960 grms., or 880 grms. in woman, may be said to be a micrencephalic brain. There is a wide latitude in weight, though, in micrencephalia. Thus Sander[1] describes the brain of a micrencephalic infant of five months of age, which weighed in the fresh state only 170 grms., and Jensen[2] a girl of eight years, whose brain weighed, in the same condition, 924 grms. More important, however, than the absolute weight is the relative weight between the brain and general body. This should bear the ratio of 14 per cent. at birth, and only of 2.37 per cent. in adult life (Vierordt).

Let it be understood clearly that only some of the cases of micrencephalia can be referred to cranial causes for their production. These cases are *sui generis*, and ought not to be considered as due to arrested development of the brain. The brain exposed to pressure develops in all its parts, but slowly and incompletely. It is not an arrest of development, but rather an imperfect development. The large number of cases of micrencephalia, however, are the results of a true arrest of cerebral development, arising either from intrinsic disturbances in the ovum or from acquired pathological changes due to local cerebral or extrinsic maternal influences.

It is now generally conceded that the most frequent cause of micrencephaly is an early fœtal hydrocephalus internus. When we come to speak of this condition we will find that the endyma of the cerebral ventricles is the chief factor in its development. Hans Virchow[3] affirms that this malformation of the brain does not lie in the cranium, but in the brain itself, and is the result of a chronic lepto-meningitis, which in addition to establishing a hydrocephalus is productive of an aplasia of those portions of the cerebrum which are associated with the diseased pia.

The appearances of defective or arrested development outside of the diminished size of the cerebrum are apparent at the first sight of a micrencephalic brain. These are referable to the gyri and sulci, to the corpus callosum and to special lobes.

The gyri and sulci not only deviate in size from the normal, but the primary convolutions and fissures may not be distinguishable at all or only with difficulty. Much attention has been given to the occurrence in these brains of a type of gyral and fissural development referable to those occuring in the anthropoid apes and which have been called ape-like fissures.

Ape-like Fissures. On the lateral convexity of the occipital lobe of all anthropoid apes, separating it distinctly from the parietal, running almost directly perpendicular to the Sylvian fossa, is a fissure. This is the opercular fissure of the apes. It is called opercular because its posterior or caudad lip overlaps its anterior or encephalad lip by a thin wall of cerebral tissue like a *cover*. When the lips forming this fissure are separated, gyri concealed by the overlapping tissue are brought to view. These gyri are

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[1] Sander, Julius: Archiv. für Psychiatrie, Bd. i. p. 299.

[2] Jensen, Julius: Archiv. f. Psychiatrie, Bd. x. p. 950

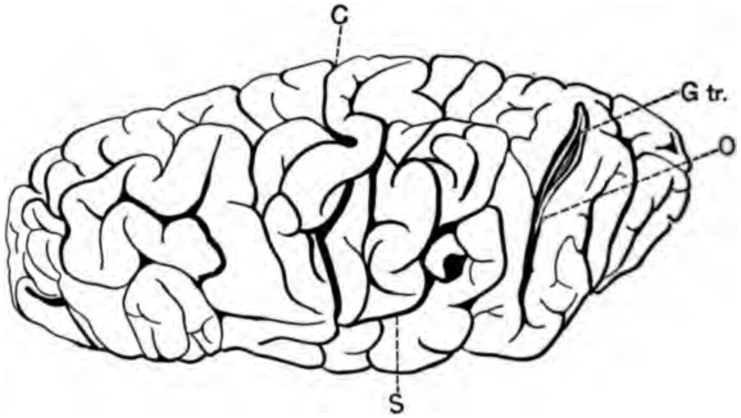
[3] Virchow Hans: Ein Fall von angeborene Hydrocephalus internus. Zugleich ein Beitrag zur Microcephalenfrage. Aus Festschrift v. A. v. Kölliker. 1886. Leipzig.

transition gyri between the parietal and occipital lobes. Gratiolet[1], w
called attention to this subject, showed that in most apes some of these g
descended into the depths of the opercular fissure (fissura perpendicular
and are normally concealed by it and are only visible when the fissur
opened. In these transition gyri Gratiolet undertook to differentiate
various anthropoid apes' brain.

In some microcephalic brains this opercular or *external perpendicular oc*
pital fissure is present. Its usual site is the situation of the sulcus occipita
transversus, from which it proceeds directly downward and forward a
distinctly divides the parietal from the occipital lobe. In its depths are
be found the transition gyri which are given such an important position
both Gratiolet and Bischoff.[2] The transition gyri in the anthropoid apes a
two in number. Gratiolet taught that upon the character of these gy
whether they were visible or concealed, he could establish a basis for the cla
ification of anthropoid brains. He believed that the brain of the oran
outang was to be differentiated from that of the chimpanzee by the absen
in the latter of the first transition gyrus and by the concealment of th
second by the opercular fissure.

Some microcephalic brains have been described, in which in addition
the presence of the opercular fissure some form of transition gyrus has be
preserved, either unconcealed on the surface or only brought to view wh
the opercular fissure has been separated. (See Fig. 70.)

FIG 70.



Brain of an imbecile showing ape-like formation of the occipital lobe. (After WILLIAM SANDER.
O. Opercular fissure. C. Central fissure. G. tr. Transition gyrus. S. Fossa Sylvii.

The explanation of the presence of this fissure is an interesting one. It
well known that the external perpendicular occipital fissure is always prese
in the fœtal brain at the seventh month. In the course of the eighth mon
of fœtal life it disappears.

A brain of an insane patient has been described by W. Sander,[3] in whic
among other anomalies, a convolutional arrest of development of the oc
pital lobe occurred, and in which the opercular fissure and these transiti
gyri were present, as was also another ape-like fissure parallel to the form

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[1] Gratiolet: Memoire sur les Plis cérébreaux de l'Homme et des Primates, Paris, 1854.

[2] Bischoff: Abhandl. der. K. Bayer. Acad. d. Wissenschaft. ii. c. xi. Bd. ii.

[3] W. Sander: Ueber eine affenartige Bildung des Hinterhauptslappen eines menschlichen Gehir
Archiv. f. Psychiatrie, Bd. v. p. 842.

and also in the occipital lobe. This latter fissure, however, has no analogue in the fœtal brain. (See Fig. 70.)

Another ape-like formation of cerebral fissures is sometimes found in micrencephalic brains in the Sylvian. In the normal brain the Sylvian fissure is composed of three legs, forming a figure like the letter Y. The part of the hemisphere bounding its anterior edge is the orbital surface of the third frontal convolution, its posterior, the apex of the temporal lobe. Between the two descends the operculum[1]—not to be confounded with the opercular fissure of the ape's occipital lobe—pressing the two legs apart, but not descending far enough to convert the Y into a V. In the anthropoid apes this part of the cerebrum apparently descends further and causes the Sylvian fissure to assume the latter shape. This V-shaped condition of the Sylvian is sometimes found in micrencephalia. The apparent descent of the operculum is not the only factor in the production of the V-shaped fissure of Sylvius. The evolution of speech in man has been attended by an increase in the development of the lower frontal convolution, adding, as it were, to the formation of the third leg of the Y from the original V-shape, as found in the anthropoid apes. A comparison of the human with the simian brains will establish this proposition. In the latter the frontal lobe in its lower part is flat and relatively small, in the former well rounded, and in part in apposition with the anterior extremity of the temporal lobe, so that it forms a lower stem to the V, converting it into a Y.

Even in some cases where the ape-like formation of the fissure of Sylvius is present, the Island of Reil may be uncovered. As a usual rule, it is covered by the operculum.

On the mesal surface of the cerebrum is sometimes found in micrencephalia another anthropoid ape-like formation of fissures and gyri. This is observable in the formation of those on the mesal face of the occipital lobe. In man the internal perpendicular occipital fissure joins directly the calcarine fissure, forming the wedge-shape lobe, the cuneus. This junction is characteristic of the human brain. In the anthropoid brains the internal perpendicular fissure, while it approaches the calcarine, does not join it, so that a space is left between the terminal extremities of these two fissures, forming a gyrus which has been called by the Germans, *die Zwickelwindung*. This gyrus connects the hippocampal gyrus with the cuneus, lying between the former and the gyrus fornicatus, and is found in all the brains of the anthropoid apes. Its presence in some cases of micrencephaly has been noted by several writers, including Bischoff and Vogt[2].

While the above so-called ape-like fissures and gyri occur only occasionally in micrencephalia, they have likewise been found in idiots who were not microcephalic, as well as in some negro brains. Their appearance in micrencephalia probably led Carl Vogt[3] to the hypothesis that micrencephalia was an atavistic phenomenon, and that the idiots possessing these brains represented a return to the ape state of man, or to that original state before the races of apes and man were divided. Important as their presence is, it should be remembered that all the fissural anomalies described do not occur together in the same brain, and that they sometimes appear in apparently normal brains. In fact, Meynert[4] described the brain of a well-educated engineer, who, during life, presented no mental change, in which the opercular fissure was found exceedingly well developed.

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[1] Operculum or opercular lobule, so called because it covers the cortex of the Island of Reil.

[2] Carl Vogt: Arch. für Anthropologie, Bd. ii., Braunschweig, 1867.

[3] Vogt, Carl: Ueber die Microcephalia oder Affeumeuschen, Archives für Anthropologie, Bd. ii. Braunschwig, 1867, p. 276.

[4] Theodor Meynert: Arch. für Psychiatrie, Bd. vii. p. 281.

Their presence, however, must be regarded as the expression of a partial arrest of cerebral development.

Almost as important as the presence of the so-called ape-like fissures and gyri are the abnormal types of other fissures and gyri discoverable in many micrencephalic brains. As a usual thing, in these cases, the hemispheres are greatly asymmetrical both in size and shape, and the fissures and gyri, while aberrant in each, yet differ from each other. It is even sometimes difficult, by reason of the absence of the divisional fissures, to define the cerebral lobes. The fissures that are usually aberrant are the central, the pre- and post-central, the interparietal, and the occipital. These may be reduplicated or interrupted by so-called bridging convolutions. These convolutions either represent a redundant duplicature of fixed convolutions or are the result of an aberrant fissural development.

The course and direction of these fissures may also deviate from the normal type. Thus the central fissure may cross the post-central gyrus and connect with the interparietal. The internal perpendicular occipital may divide the hippocampal gyrus into two gyri. The calcarine has been described as being continuous with the hippocampal.

The formation of atypical convolutions and gyri is the expression of an inherent primordial defect in the epiblastic development of the neural tube. This defect occurs *ab initio* and cannot be ascribed to any defect in the brain coverings or cranial vault.

That Virchow's belief, that the formation of normal fissures and gyri is the result of the mechanical pressure of the enveloping tissues of the brain upon a more rapidly growing cortex is not the explanation of this phenomenon, is proven by the fact that brains which do not completely fill the cranial cavity and cerebra, that have defective or partially absent crania, show the same tendency to the formation of gyri as those whose envelopes are completed. If pressure were the cause, the latter cases should present no convolutions. Such, however, is not the case. The explanation of convolutional formation must be sought somewhere else. Jelgersma[1] denies the connection or relation of cranial growth and the development of convolutions. He believes that the formation of gyri and sulci is simply the result of the tendency of the cerebral mantle to extend its surface of growth against the restraining factor, of a mutual relation between brain space and the development of the tracts within the cerebrum.

That the development of the central connections of the cortex and the correlated parts is an important factor *per se*, independent of space, is proven by the tendency in the higher vertebrates to show a convolutional arrangement of the intercalar gray masses, like the olive and the nucleus of the trapezium. This convolutional tendency certainly cannot be ascribed to pressure arising from the meninges or cranial vault. The cerebellum itself is also nothing but an intercalar station between cortex and periphery, and is rich in convolutions. It appears to me that the development of neural tracts is alone the important element in producing a convolutional arrangement of the gray matter. Where such tracts develop early in the cerebrum there will be found an early appearance of the convolutions. This is exemplified by the early development of the external perpendicular occipital fissure and the central, representing as they do the areas of visual and motor functions, both important functions in the preservation of the species. It must be remembered that the formation of the cerebral gray is much more rapid than that of the subcortical white tracts; that, also, the connections between the two are early formed, but that these connections and tracts act as a pulling force upon a covering (cortex), which grows more rapidly than the connecting tracts themselves. This seems to me to be more than a probable explanation of the formation of the involution of gray cortex. It coincides with the facts of embryological development; it explains the tendency to the involution of such bodies as the olivary nuclei and cerebellum, and likewise disregards the presence or absence of external pressure, which we have shown cannot have any influence in producing a fixed convolutional type. The relation of cortex to periphery is always fixed, and connecting strands do not deviate in their course through the cerebral axis. The pyramid tract always seeks its cortical connection through fixed and definite areas, and attaches itself, if I may use that expression, to a fixed cortical area. This is also true of the cerebellar connecting tracts and of other intra-cerebral connections. Hence do we normally find fixed depressions in the cortex, the primary fissures and convolutions depending upon the formation of these fissures. These primary fissures and convolutions are present in brains which themselves deviate from the normal in other respects; for instance, in cases of acrania and in cases of cranial asymmetry. If Virchow's explanation of the formation of the fissures and convolutions were correct, then every cranial asymmetry and deformity would have to be accompanied by an aberrant fissural development. This, however, is not the case.

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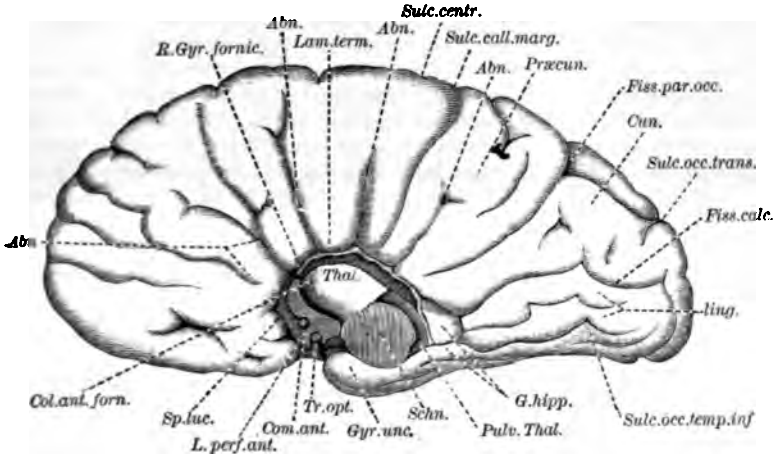
[1] G. Jelgersma: Über den Bau des Säugethiergehirns, Morphologisches Jahrbuch, Bd. xv. p. 83, 1889.

FIG. 71.



Mesal face of the micrencephalic brain of Hofmann, showing absence of corpus callosum, radiatory disposition of the sulci and gyri, and separation of the internal occipital and calcarine fissures. (Case of Onufrowicz.)

Absence of the Corpus Callosum. Other fissural anomalies depending upon the absence of the corpus callosum deserve mention. The corpus callosum is not infrequently found absent in micrencephalia. Cases of the absence of this commissure in other brains not micrencephalic have also occurred.

Onufrowicz,[1] who exhausted the literature on this subject, and who examined the brain of the micrencephalic, Hofmann, mentioned in the title in the foot-note below, comes to the following conclusions: The common characteristics of all brains in which complete absence of the callosum was found were:

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[1] Das balkenlose microcephalengehirn Hofmann. Archiv f. Psychiatrie, xviii. p. 305.

1. Absence of the lyra.
2. Probable absence of the gyrus fornicatus.
3. Absence of the sulcus calloso-marginalis, excepting its dorsad asce ing branch.
4. Fornices and septum lucidum are not joined in their middle.
5. Dilatation of the ventricles, at least the posterior horns.
6. Nervi Lancisi for the most part present.
7. Absence of the tapetum callosi has never been demonstrated.

The corpus callosum may be rudimentarily developed or may be entir absent. In the former cases it is very thin and short, with rudimentary c nections with the frontal and occipital lobes. Its absence must be regard as an arrest of development at a stage before the great commissural conn tions between the two fœtal hemispheres take place.

When it is absent it is naturally accompanied by fissural and gyral abn malities, as well as by absence of other anatomically related parts. Thus most of the cases there is absence of the anterior commissure and a rudim tary condition of the middle commissure. The fornix is also rudimenta The fissures on the mesal face of the hemispheres assume a radial distrib tion at right angles to the centre of revolution of the cerebrum. The int nal perpendicular occipital is no longer perpendicular, but horizontal, a does not join the calcarine. The calcarine is continued forward and joins t hippocampal. (See Fig. 71.)

The ventricles are not infrequently dilated, and sometimes the posteri horn of the lateral ventricle is entirely absent or obliterated.

The occurrence of defective development of the corpus callosum has be explained by Richter[1] as being due to mechanical causes arising from an as ciated defect in cranial development in some cases. The usual angle form by the petrous portions of the two temporal bones, being about 120°, becom enlarged in diseased crania, especially in those of idiots, to more than 150 the tentorium attached to them, on a consequent diminution in the crani base, sinks down further, dragging the falx with it, so that the latter oppo by its presence an obstacle to the junction of the commissural fibres of t fœtal brain, which are sent out from each hemisphere, and which by the junction form the callosum. The sinking of the falx by being dragge down by the tentorium, which cannot develop, owing to the defective deve opment of the cranial base, and the further development of the cerebr hemispheres of the fœtus, cause the callosum to grow against the falx and t be arrested at that obstacle to its growth.

Cases of absence of the callosum without micrencephalia are as numerou as cases occurring with it. Thus cases have been recorded in which durin life no symptoms of any cerebral defect were manifested, the defect havin only been discovered on autopsy.

Other parts of the brain in micrencephalia show an associated retardatio in growth and development. The crura are asymmetrical and smaller tha normal, the pons not infrequently flat, and in one case, which I have read though I have been unable to find the reference since, so defective that th trapezium was exposed. Its defect in most cases depends upon the amoun of arrest which the cerebellum has suffered. The medulla shows a simila involvement. The pyramids are small, sometimes even rudimentary. Th asymmetry of the medulla in some cases is exceedingly well marked. Al these defects depend for their presence upon the nature, the seat, and th extent of the cerebral developmental arrest, so that they vary in the differen cases.

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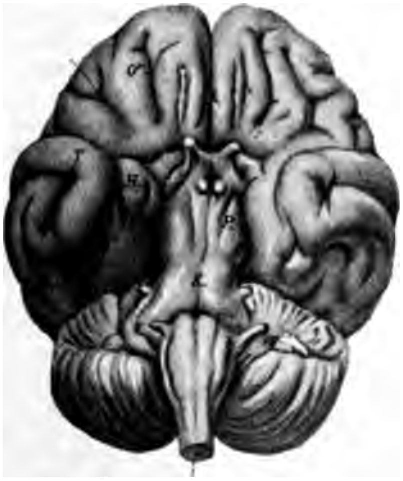
[1] Richter: Virchow's Archiv, 1886, Bd. 106.

Giacomini describes a case of a micrencephalic in whose brain was found a complete junction of both crura in the mesal line, forming apparently one crus. (See Fig. 72.)

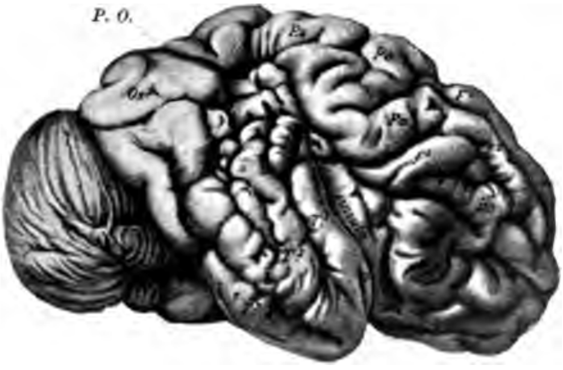
FIG. 72.



Micrencephalic brain, showing deformity and junction of the crura. *Or.* Orbital sulcus. *K.* Pons. *T.* Apex of temporal lobe. (After GIACOMINI.[1])

Microgyria. Associated with micrencephalia and with porencephalia is another abnormality of fissures and convolutions to which the term microgyria has been applied. It is characterized by the development of almost innumerable secondary and tertiary fissures, dividing the affected lobes into minute lobules and gyri. The gyri are very small, numerous, and thin. (See Fig. 73.) In some cases their smallness and thinness give the gyri the appearance of a mere membranous expansion. The degree of microgyria varies in the most of cases. In some but a lobule of the brain may be the

FIG. 73.



Brain of micrencephalic cretin, showing an external parieto-occipital fissure at *P. O.* and microgyria of temporal lobe. Insula is uncovered. (After GIACOMINI.[2])

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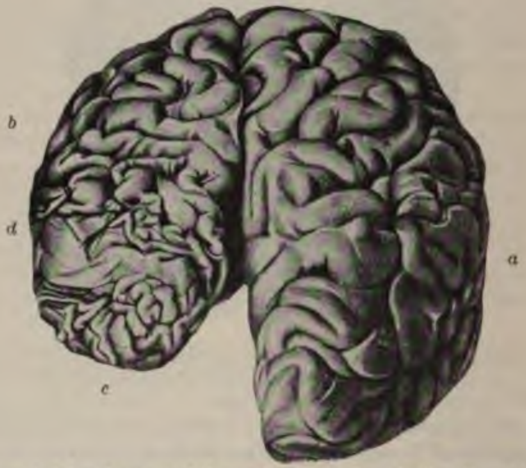
[1] C. Giacomini: I cervelli dei microcefali, 1890.

[2] C. Giacomini: I cervelli dei microcefali, 1890, p 50.

seat of this peculiar malformation, in others an entire lobe, and in still others one hemisphere, or even both in part. This condition is also present in cases of absence of special lobes of the cerebrum, being distributed over the remaining lobes of the affected hemisphere. (See Fig. 74). It was at one time considered that this condition of the crebral convolutions was the result of an encephalitic process, with subsequent contraction and shrinkage of the affected parts, but microscopical examination of the cortex of microgyral brains has failed to demonstrate any evidences of a pathological lesion. Chiari[1] examined carefully the cortex of one of his cases and could find no morbid processes in the cortex or subcortical fields, at least none which could be adduced as due to a chronic encephalitis.

FIG. 74.



Microgyria and arrested development of the occipital lobe in a deaf mute. *a*. Right hemisphere. *b*. Left hemisphere. *c*. Left defective occipital lobe with microgyria. *d*. Membranous cyst in region of the parietal lobe. (After ZIEGLER.)

A peculiar feature of the cortex in microgyria has been demonstrated by Binswanger[2], who found no giant cells in the affected cortex of the motor region, and that the pyramid type of cells was simply approached. In other words, there was but a rudimentary development of the pyramidal layer of cells. This is a good indication that the condition of microgyria is dependent upon a defective or arrested development of the cortex, and not a local pathological process. Anton[3] corroborates to a great extent the demonstration of Binswanger. In his cases the cellular elements of the cortex were not only rudimentary, but were much diminished in number. Recently the examination of two more cases was made by Otto[4], who found in the cortex of the smallest of the convolutions a defective development of the cells, especially the pyramidal cells, and an entire absence of the giant or large pyramids. But in addition to this he describes two zones, light gray in color, beneath the cortex and interspersed in the subcortical layer. The extent of these varied. On examination they proved to be rudimentary ganglion cells.

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[1] Chiari: Jahrbuch für Kinderheilkunde, N. F., xiv.
[2] Virchow's Archiv, 1882, Bd. 87.
[3] Anton: Zeitschrift für Heilkunde, 1886, Bd. vii.
[4] R. Otto: Casuistische Beiträge zur Kentniss der Mickrogyrie, Archiv für Psychiatrie, Bd. xxiii.

Corresponding with the rudimentary character of the pyramidal cells of the cortex is to be found a smallness in these cases of the pyramidal tract, and the pyramid in the medulla oblongata. Heschl[1], who first described microgyria, concluded that its cause was due to an arrested development in the subcortical region, and that by reason of the continuous growth of the cortex, it being restricted in expansion by the arrest below, necessarily curled itself into numerous divisions or gyri.

No exhaustive study of the microscopical features of the cortex in micrencephaly has as yet been made. Thus far there has been observed a decrease in the neuroglia and a defective or imperfect development of the cortical cells. Sachs[2] examined the brain of a child with arrested cerebral development and found marked changes in the structure of the small and large pyramid cells. Almost all of these were defective. Some were without nucleus and nucleolus; in others these were present, but surrounded by a "detritus-like mass." In his case there were no indications of a previous encephalitic process, nor could he demonstrate any morbid changes in the neuroglia. The changes in the cells described by Sachs seem to be analogous to those occurring in microgyria described by Chiari, Binswanger, Anton, and Otto.

Absence of Special Lobes. Associated with micrencephalia and microgyria are cases of cerebral malformation in which partial and complete absence of special lobes of the cerebrum occur. The lobe most frequently absent is the frontal, next in frequency is the occipital. Ziegler[3] describes a case of this form of cerebral hypoplasia in a deaf mute in whom the left occipital lobe was entirely absent, the parietal defectively developed, and its convolutions showing a typical microgyria. (See Fig. 74.)

It is extremely difficult to assign a reason for these cases of malformation. Why an arrested development should be limited to a few lobules or to an entire lobe without any discoverable pathological condition in the hemispheres seems to be inexplicable, excepting on the ground of some local cause. In some of these cases thickening of the pia, indicating a former inflammatory process in that membrane, has been described.

Another malformation occurring at times with micrencephalia is junction of the two cerebral hemispheres. Cases of the same anomalous development also occur independently. The junction is not usually complete, the longitudinal fissure being present in part, so that the cerebrum gives more the appearance of two hemispheres connected by bridges of cerebral tissue which cross the fissure interrupting it. The connection may occur also at the base as well as upon the dorsal surface. (See Fig. 76.)

The somatic accompaniments of micrencephalia are referable to arrests of development in other parts of the body.

From birth a micrencephalic child is easily recognized by the small size of the head and the relatively large face. This difference is more apparent as the child increases in age, for while the head seems to stand still in development, the face grows steadily, emphasizing the original difference.

Very frequently there is an absence of the intermaxillary bone, giving rise to harelip. Cleft palate is sometimes an accompaniment. The nose is broad and appears to sink into the face, giving a peculiar expression. The skull in its entirety shows usually a prognathism which is well marked whenever it occurs. (See Fig. 75).

The extremities are the seat of malformations, such as shortening of the limbs, defective or absent toes. One limb may be fully developed and the other be atrophic. Club-foot and contraction of tendons are not infrequent.

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[1] Heschl: Ueber die vordere quere Schläfenwindung des menschlichen Grosshirns. Wien, 1878.

[2] B. Sachs. On Arrested Cerebral Development, with Special Reference to its Cortical Pathology. Journal of Nervous and Mental Diseases, 1887, vol. xiv. p. 541.

[3] Ernst Ziegler: Lehrbuch der Speciellen Pathologie, 7 Auflage, Jeun, 1892, p. 324.

The general causes mentioned in the beginning of this chapter have their influence in the production of micrencephalia.

FIG. 75.

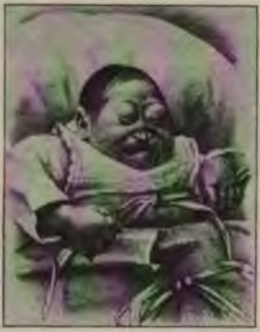

FIG. 76.

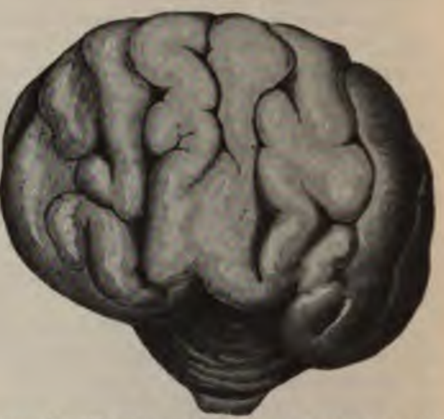

Fig. 75. Photograph of micrencephalic child, one day old (WILLE, *Archiv. für Psychiatrie*, vol. x).

Fig. 76. Brain of the same, showing simplicity in convolutions and fissures. Dorsal view. The hemispheres are asymmetrical and are united in the process of development, the longitudinal fissure being interrupted.

Heredity, alcoholism, and syphilis in the parents are important factors for its development. Maternal impressions, fright on the part of the pregnant mother, are no less influential causative features in the production of this developmental arrest.

The hypothesis of Carl Vogt, that those cases of micrencephalia with apelike formation of fissures and convolutions were simply cases of atavism, can have no claim to our recognition. The researches of Aeby,[1] Bischoff,[2] Sander,[3] Mierjajewsky,[4] Virchow,[5] and others have given this theory its quietus. The generally accepted opinion of all now regards micrencephalia as a result of a cerebral developmental arrest which is occasioned by special causes varying with each case. The special causes may be a hydrocephalus internus, a narrowing, obliteration, or defective development of the carotid artery, a defective cranial development, local pathological processes, such as an ependymitis, or a traumatism occurring in utero. Porencephalia and micrencephalia are not infrequently associated and are etiologically mutually dependent in some cases.

Persons afflicted with micrencephalia rarely reach adult life. A few cases have been reported of micrencephalic idiots who have reached an age of forty years. As a usual thing some intercurrent affection ends their careers early. In fact, micrencephalics have little power of resisting disease, and succumb to pulmonary and infectious affections in infancy and childhood.

The life history of some of these sufferers is the same as that of all idiots.

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[1] Aeby: Archiv. für Anthropologie. vi and vii., 1874; Ueber des Verhältniss der Mikrocephalie zum Atavismus, Stuttgart, 1878; Virchow's Archiv, Bd. 77.

[2] V. Bischoff, loc cit.

[3] Sander, loc. cit.

[4] Mierjajewsky: Ueber einen Fall von Mikrocephalie, Verhandlungen der Berliner Gesellschaft für Anthropologie, etc., Zeitschrift für Ethnologie, Berlin, Bd. iv., 1872.

[5] Rud. Virchow, Menschen und Affenschädel, Bd. iv. Heft 96, der gemeinwissenschaftlichen Vorträge, Berlin, 1870, p. 31.

In them there is an absolute lack of all mental co-ordination. The idiocy is extreme, and all the higher mental functions are absent. They give no sign of receiving or registering any mental impressions. Others show a less high degree of idiocy, and give indications of defective mentality, with few signs of any ideational life. Some of them can be taught simple mechanical pursuits.

The motor innervation is seriously affected in most cases. This defective innervation may vary from a slight paresis to an absolute paralysis. Some are unable to perform any voluntary motion whatever, others are able to walk and even run. The most frequent condition of defective motor innervation is a paretic condition of the extremities of such a nature that while such patients may, when recumbent, move their limbs to some extent, they are, when erect, helpless and unable to walk. (See chapter on Cerebral Palsies of Childhood.)

Convulsions more or less general are occasionally observed. They are usually general in character. True epilepsy associated with micrencephalia is not a frequent manifestation.

Treatment. The treatment of these conditions is practically the treatment given to idiots and imbeciles. It is the subject of anxious consideration to the parents of these unfortunates. The family physician is usually brought into the council, and thus much depends upon his decision in this matter; every physician, be he neurologist or general practitioner, should be acquainted with the essential principles underlying the treatment of the results of these defective states.

Before the lack of mental development becomes apparent the physician may be called upon to treat the child for the paralysis which is so often the most pronounced feature in these cases. This is to be treated by the same means as are paralyses from cerebral disease in the adult, the consideration of which will be found in Chapter XV, and, further, in the chapter on "The Cerebral Palsies of Childhood." The contractures which so often invade the paralyzed limbs are especially troublesome conditions to remedy. Systematic exercise of muscular groups, both actively and passively, should be put in practice early. To assist in this, electricity, massage, calisthenics, and light gymnastic exercise should be brought into requisition. These means should not be neglected, as they are the most important in producing a somewhat useful limb. Especially are they serviceable in spastic conditions of the extremities. Physical exercise and gymnastic work must always be made commensurate with the physical power and strength of the patient. They should never be permitted to be carried to the point of fatigue. There is no doubt that in some of these cases—in those whose mental weakness is not of a high grade—that the potential of mental development is increased by these means.

Still more important should be the consideration given to developing a useful hand. On such a result much of the patient's future contentment depends. In this connection it should be remarked that it is useless to attempt remedial measures late. The earlier they are put into execution the greater will be the chance of success. Such a result will fail in those cases in which the mental weakness is of a high degree. When it is impossible to arouse or excite attention on the part of the subject it is useless to continue treatment in this direction. This is almost invariably impossible in idiots exhibiting a high degree of mental defect. If the faculty of attention can be excited or developed there will be promise of a great degree of success in developing other mental faculties and the physical powers. The elder Seguin, to whom we are certainly indebted for an exhaustive investigation

f this subject,[1] certainly deserves credit for his wo
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of the weak-minded.

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evelopment of the speech centres, or because they ha
s. These defects are all too common among the high
it is but a waste of time and energy to attempt any educ

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and its Treatment by the Physiological Method, 1886.

tional measures. All that should be done in these cases is to pay attention to the control of the sphincters of the rectum and bladder. These patients should be led to the closet at definite periods and kept there until they have voided the contents of the bowels and bladder. In some cases there is a lack of control of these sphincters, and these unfortunates are in a constant condition of filth. Under these circumstances, the rule of having them led to the closet at stated intervals and the careful use of strychnine internally may remedy this feature.

Their attention may be developed by giving them bright-colored objects with which to play, and placing them in the company of other children in the institution. The social element in these places seems to exercise a beneficent influence.

With those having a higher mental development the features of all these educational institutions should be directed to manual training. The use of tools is easily learned by them, and imbeciles may become fair laborers, and even fair artisans. Some have been taught agriculture, others carpentering, plastering, and a few others plumbing. These are fields in which they may become useful members of society. This training should only be attempted in those grades of imbecility in which the educational system adopted has produced the ability to form conceptions and simple judgments. At no time should the physical development be slighted, for it is almost axiomatic in these cases that the potential of mental development is increased by the attention bestowed to acquiring a good physical development. Of course, physical hindrances, such as contractures, should be removed. The orthopedic surgeon should correct by tenotomy and the usual special surgical measures these obstacles where he can.

Success in manual training is aided by the faculty of mimicry and imitation, which these patients possess in a marked degree. We can readily get an imbecile, and occasionally an idiot, to use a tool in the way that he has seen it used. Thus the use of the saw, the hammer, the spade the, plough, may be put to good purpose both for the object of exciting the patient's interest and increasing his physical powers.

As to the moral training, in a large proportion of these patients little can be done. It seems almost impossible to awaken their moral sense. This is owing to their defective cerebral development, and is a common feature among imbeciles. The term of moral insanity has been applied to these cases, a classification which seems to me to be useless. The term imbecility implies the defective development of mental faculties. The moral faculty or sense is certainly included among the mental, and as such is defective to a greater or lesser degree in all imbeciles. Hence, when we speak of imbeciles, that term includes the cases of so-called moral insanity. As just stated, it is almost useless to attempt to awaken in these unfortunates the moral sense. I know of no means yet applied, either philanthropic, educational, correctional, or penal, which has been attended with success. In fact, it is questionable whether in those cases in which the moral sense is lacking, or very defective, it is wise to carry on any educational training, for by it you simply increase the power of these beings to do injury and harm. Tuke believes that this class of imbeciles should be isolated and subjected to detention and restraint in a suitable institution, where, he thinks, that by a uniform, temperate, and positive restriction their existence may be made useful.

During the past few years physicians had been led by the report of successful cases to turn the treatment of some of these cases over to the surgeons, who have performed craniectomy, tapped the ventricles, and performed sundry other operations in their desire to correct these states of arrested development. Notwithstanding the *à priori* absurdity of craniectomy as a corrective agent, surgeons have, nevertheless, performed the operation with the hope of success. Time and experience have proven the falsity of their statistics and the absolute uselessness of such a surgical procedure. I should advise all physicians, therefore, to try other than surgical means. The operation is now falling into disuse in these cases, as it deserves, for it certainly had no rational excuse for its existence.

DEVELOPMENTAL DEFECT OF CEREBELLUM. The cerebellum is likewise the seat of defective development. It may be totally absent or simply show an arrested development of one or more lobes. Asymmetry of the cerebellum without any discoverable pathological process which might occasion it is not rare.

Even the gyri of the cerebellum may be the seat of a microgyria. These conditions are sometimes associated with micrencephalia and may occur entirely independent of any developmental defect in the cerebrum.

Total absence of the cerebellum is rare. The celebrated case of Combetta, cited by Longet,[1] deserves mention. In this case in place of the cerebellum there was found a hemispherical gelatinous mass which was attached to the medulla by two similar pedicles upon which were two masses or accumulations of isolated and separated white substance of the size of peas. There was no fourth ventricle, no trace of a pons.

The most frequent form of cerebellar arrest is a rudimentary condition of one or more lobes. One hemisphere of the cerebellum may be diminished to such an extent that it forms but a rudimentary appendage to the others. When microgyria of the cerebellum is present it shows the same general features as microgyria of the cerebral hemispheres. In these cases the Purkinje cells are small and defectively developed, and the nucleus dentatus is rudimentary.

The fissures which divide the cerebellum into lobes do not run parallel to the posterior margin of the cerebrum, but assume a position at right angles to it.

The most important feature of arrested development of the cerebellum is the rudimentary condition of the tracts to which the cerebellum serves as an intercalar ganglion. Thus the pons is diminished in size, as is the restiform body and the tegmenti brachium. One or the other of the olivary bodies is rudimentary according to the lobe of the cerebellum which is involved. The medulla itself, therefore, becomes involved in the malformation; it is diminished in size, asymmetrical, and presents a defective or absent olivary eminence. If the cerebellum be bilaterally involved, both olivary bodies will be rudimentarily developed; if but one hemisphere be the seat of the defect, the opposite olivary body is the one which is rudimentary. In a case described by Huppert,[2] he found the crura, pons, medulla, and thalami much smaller than normal.

Symptomatology. It is impossible to make a diagnosis of this defect during life, for there are no symptoms which characterize it. Those which have been noticed are common with micrencephalia; thus, there is idiocy or imbecility, a paretic condition of the extremities, and a motor inco-ordination. The ataxia possesses no special characters, however.

HETEROTOPIA OF THE GRAY SUBSTANCE. An interesting deviation from normal cerebral structure is that condition of excessive focal growth of gray matter which occurs in abnormal situations in the white substance of the brain. The favorite sites of this development are: 1. Near the ganglionic masses, the thalamus, and caudate nucleus; 2. Near the walls of the ventricles, more frequently the lateral ventricles; 3. In the white substance of the cerebellum between the corpus dentatus and the cerebellar cortex.

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[1] Hitzig. Ziemssen: Cyclopædia of the Practice of Medicine, vol. xii. p. 844.

[2] Max Huppert: Archiv für Psychiatrie, Bd. vii. p. 98 et seq.

It was first pointed out by Virchow[1] that these insular accumulations of ganglionic material had no direct communication with the cerebral cortex. And yet it appears to us that these masses which in their microscopical appearance do not deviate from the true structure of the cortex, being formed of ganglionic cells, nerve fibres, and neuroglia, must have a direct relation with the gray mantle, for their identity in structure and their situation denote them as offshoots or detachments from the cerebral ganglionic formation. Their position seems to be due to two factors. First, the development of the subcortical tracts may occur in an abnormal or aberrant position, detaching from the parent mass some of the original gray of the hemispheric vesicles, or of the prosencephalon during the invasion of that part of the fœtal brain by the developing fibre systems. Second, the original embryological ganglionic units may have assumed from the beginning an abnormal position, and are subsequently caught or entrapped in the developing fibre systems which developed in a normal course. On any other supposition the aberrant position of these masses cannot be satisfactorily explained.

The cerebellum seems to be more frequently the seat of this abnormality than the cerebrum. Perhaps this preponderance is due to the fact that the fibre systems, because they are concentrated in the cerebellum in a smaller area, are more apt to entrap some of its cortical or ganglionic gray than where these systems are relatively less concentrated.

When it occurs in the cerebrum the site of selection is near the ventricular walls. This is the situation of these masses in two-thirds of all the cases.[2]

Its occurrence in the cerebrum was found by Meschede[3] in cases of epileptic insanity. Otto, however, has found it in apparently perfectly healthy individuals. Spitzka,[4] however, says that "heterotopia of the gray substance in the cerebrum must be always regarded as an abnormal occurrence; in the cerebellum it may be found in perfectly healthy individuals." When heterotopia does occur in persons who during life have shown a defective mental integrity its occurrence must be assumed as an indication of a cerebral morphological stigma.

CEREBRAL HYPERPLASIA. A redundancy in development of the essential cerebral elements may occur independently of a pathological process, and be associated with errors in the developmental growth of both the gray and the white substance. When both are increased there results a condition of macrencephalia, or hypertrophy of the brain.

When the gray matter alone is involved, it is only so in limited area, always on the surface. Here they form small rounded excrescences, which are usually about 2 mm. in diameter,[5] and which are directly connected with the cortical layer beneath. Their most frequent site is over the convexity of the frontal lobes, lying at times free on the gyri, at others on the cortex within the sulci. Their size varies from that of a millet-seed to that of a bean, although the smaller growths are the most common.

Microscopically, these masses show an analogous structure to that of the cortex, the ganglionic cells, however, being usually smaller.

Macrencephalia, or hypertrophy of the brain, is not of very frequent occurrence. It is not to be referred to an acquired pathological process, but to a congenital excess of growth of the brain. The size of the brain may be extreme; thus Ziegler[1] reports a brain of a twenty-year-old girl which weighed 1857 grammes; Virchow,[2] that of a three-year-old child, which weighed 1911 grammes; and Landousi[3] one, 1590 grammes in weight. The enlargement may be partial or general, and is due to an increase in the neuroglia. The brain cortex is thicker than normal, this increase being mostly confined to the superficial or so-called barren layer. Notwithstanding the increase in depth of the gray cortex, the ganglionic elements do not seem to be increased in number. The augmentation of cortical growth depends upon the increase in the connective-tissue formation. This is more apparent, however, throughout the white substance.

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[1] R. Virchow: Virchow's Archiv., Bd. xxxviii. p. 138.

[2] R. Otto: Virchow's Archiv. Bd. cx. p. 81 et seq.

[3] Meschede: Virchow's Archiv. Bd. lvi.

[4] Spitzka: The Somatic Etiology of Insanity, American Journal of Neurology and Psychiatry, 1882.

[5] Otto: Ueber hyperplasia der Hirnrinde, etc., Virchow Archiv, Bd. cx. 1887.

The skull gives evidences of internal pressure, the head showing a commensurate enlargement. The bones of the skull are thin and the fissures separated. The articular cartilages of the base are suffused and reddened. On opening the skull the dura is found adherent to the cranium. The gyri are flattened.

This disease is one of childhood. The child is either born with the anomalous cerebral condition, or it is developed soon after birth. The enlargement of the skull is of such a character that one is apt to make a hasty diagnosis of hydrocephalus congenitus. The shape of the enlargement is, however, different, the outlines of the skull being angular, while in hydrocephalus it is round and the head globular. Fletcher Beach[4] gives the following points of distinction between the two: "The enlargement of the skull in cerebral hypertrophy is more marked just above the superciliary ridges than at the temples, and the fontanelle is often depressed instead of being full and elastic."

Fagge, in commenting on this condition of the fontanelle, judges it as inconsistent with the accounts which have been given of the dura mater bulging as soon as the calvaria was opened.

The etiology of this affection is yet unsettled. Alcoholism of the parents, syphilis, and rhachitis have all been spoken of as a possible cause. Traumatism to the infant's head is also cited as a factor.

Clinically, the disease manifests itself in imbecility or idiocy, paresis of the extremities, as shown by the lolling, tottering walk, drowsiness, and epileptiform convulsions.

ENCEPHALOCELE AND OTHER DEFECTS. There are certain malformations of the brain which are commonly associated with defects in the cranial bones. These, which include anencephalia, which is described on page 317, may be summed up as follows: Encephalocele, cyclopia, and arhinencephalia. The cause of these defects lies in an arrest of development of the structures of the mesoblast, which are involved in the formation of the cranial bones. The cranium, like the spinal column, is formed from the mesoblast by the development of protovertebral masses situated on either side of the neural tube; these finally coalesce and include the central nervous system in their interior. When they fail to unite dorsally they leave a cleft through which the brain or the spinal cord may protrude. To this condition are applied the terms of cranioschisis and rachischisis, respectively. Sometimes both the brain and spinal cord are uncovered, a condition which is called craniorachischisis. The cause of the non-closure of the bony canal is ascribed to an accumulation of fluid in the canal and to external pressure.

When the cranial vault fails to close either from an arrest of development of the bony structures or from causes arising within the cavity, the brain forces itself through the opening, producing a tumor on the surface of the head. The tumor may consist of the meninges alone, which are pressed outward by an accumulation of fluid within the subarachnoidal space, or the brain itself may form with them part of the tumor. In the latter case the condition is called meningo-encephalocele. The membranes at times do not appear in the hernial protrusion, and the brain covered with remnants of pia alone forms the tumor. This is called encephalocele. When the hernia is formed of the meninges alone the condition is called meningocele. Sometimes when hydrocephalus internus is present the distended ventricle forms with the brain substance a part of the tumor. When this occurs there is present a hydrencephalocele. Usually these hernial tumors are covered with the skin of the scalp; in some cases, however, this is even absent, and the tumor is devoid of covering.

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[1] Ziegler: Lehrbuch der speciellen Pathologischen Anatomie, p. 326, 1893.
[2] Virchow: Gesammte Abhandl., 1856.
[3] Landousi: Gaz. méd. de Paris, 1874.
[4] Fletcher Beach: Fagge's System of Medicine, vol. i. p. 572.

The position of the protruding mass varies. Miller,[1] who carefully analyzed forty-two cases which had come under his observation, found that the most frequent position for these hernias was the anterior portion of the skull. Of his cases thirty-four occurred in this situation, while only eight occurred in the region of the occiput. Of the former the most frequent were those in the naso-frontal region. Among these cases there was none that occurred at the base of the skull. The character of these tumors was as follows: Encephalocele, 17 cases; hydrencephalocele, 20 cases; the rest being meningoceles. Another noteworthy feature of his observation was that the occipital herniæ were almost invariably meningoceles, while the sinciput herniæ were either encephaloceles or hydrencephaloceles. In other words, cerebral protrusions were extremely rare in the occipital regions and meningeal protrusions equally rare in the frontal region. These observations would seem to militate against the theory formerly held, that it was owing to the position of the fœtal head in utero that hernial protrusions when associated with conditions of increased intra-cerebral pressure occurred. The occiput being the region of the cranium most dependent in the uterine cavity ought, hence, to be the seat of hernial protrusions more frequently than the sinciput; but the observations of Miller show that occipital herniæ occur in less than twenty per cent. of all cases. That increased cerebral pressure from excessive secretion of fluid within is the cause of these hernias there can be no doubt, but that it is the main cause cannot be affirmed. Some other factor independent of the cranial defect is operative in the causation of these malformations. Adhesion of the amnion to the cephalic end of the embryo is most probably an important etiological factor in the production of this condition when congenital. Changes arising from diseased states of the meninges, which render the membranes of the brain less resistant, may be another cause.

Sex does not appear to have any influence in the production of these herniæ, since they occur about equally as often in the female as in the male infants.

Maternal impressions and fright or other psychical shocks are the chief inciting causes of these malformations.

The size of these tumors varies from that of a hazelnut to that of a two-year-old-child's head (Miller).

Sincipital protrusions, according to their sites of exit from the cranial cavity, are divided into naso-frontal, naso-ethmoidal, and naso-orbital, for each of these articulations may be defective and serve as the origin of escape of the extruded mass. Any of the membranous parts of the infant skull may be the seat of these tumors, though the lateral aspect of the skull does not seem to be a seat of selection for them; still they occur there a little less rarely than at the base. When the hernia occurs at the base it usually presents in the pharynx, and sometimes pushing that structure before it takes its exit from the mouth.[1]

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[1] N. Th. Miller: Ein Seltener Fall von Gehirnbruch, Jahrb. de Kinderheilk, N. F. Bd. xxv. p. 195.

I am aware of the fact that former writers on hernia cerebri, especially surgical writers, have been accustomed to name the occiput as the most frequent seat of all hernial protrusions of the skull contents. This, however, does not appear to be affirmed by more recent contributions to this subject, and cannot be reconciled with the observations of Miller[2] and von Bergmann.[3]

In frontal hernias both cerebral lobes are usually extended, though the apex of a single lobe not infrequently is contained in the tumor. The same relative ratio as to both lobes exists in occipital encephalocele, though a true hernia cerebri is rare in this region.

The character of the tumor depends upon the structures which are contained in it. Meningoceles or hydrencephaloceles are usually large, globular, and constricted at the base, although they cannot be said to have a pedicle. Encephaloceles, as a rule, are much smaller. The protruding cerebral tissue shows a condition of defective or arrested growth, as well as intrinsic evidences of pathological destructive processes. Thus the ganglionic elements may not be recognizable, or they may be small and atrophic; the neuroglia is usually increased in amount and contains at times sclerotic patches. At other times one may find hemorrhages into the cerebral tissue and spots of focal softening in the mass.

The larger size of meningo-encephaloceles and of hydro-encephaloceles and the much smaller size of encephaloceles would lead one to suppose that the defect in embryological development which gives rise to the former must have occurred early in the fœtal life, that which gives rise to the latter later during that period of existence.

It is often very difficult to determine the character of these tumors unless some operative procedure such as aspirating has been adopted; for the tension is often so great in a meningocele or hydrencephalocele that it is impossible to determine fluctuation, and difficult to distinguish it from a solid tumor. As a rule, encephaloceles pulsate synchronously with the normal pulsation of the brain, although exceptions to this are not rare, and fluctuation may often be elicited in the other forms. From a surgical standpoint it is desirable that a diagnosis be made, as the nature of the operation will depend upon the character of the hernia.

Usually the length of life of infants afflicted with this disease does not extend beyond one year, although some cases have been reported in which adult age has been reached. A large number die soon after birth. Where the tumor is mostly fluid rupture occasionally occurs during the process of labor, and death ensues.

The symptoms depend upon the size and portion of the brain extruded and the amount of pressure to which the brain is subjected. Drowsiness, weakness in gait, convulsions, and mental enfeeblement are the most important.

The relief of this condition is to be found in surgical interference alone. Good results have been obtained by such measures in a number of cases. Excision of the mass, especially in the sincipital encephaloceles, has given the best results. Concerning the operations, see Chapter XXXIII., on Brain Surgery.

Cyclopia and Arhinencephalia. The hemispheric vesicles may be arrested in their development from the first cerebral vesicle or may be defectively developed, whereupon the latter vesicle may appear abnormally large. This condition is generally accompanied by a defect in the growth of the other embryonic derivative of the first cerebral vesicle, viz.: the ocular vesicles. The defect in growth may cause but one ocular vesicle to develop. This derivative of the first cerebral vesicle may appear with the latter in front of the skull, a condition which is called cyclo-encephalia.

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[1] Lichtenberg: Path. Soc. Transactions of London, vol. xviii. p. 250.

[2] N. Th. Miller, loc. cit.

[3] Ernst von Bergmann: Die chirurgische Behandlung der Hirnkrankheiten, 2te Auflage, Berlin.

At times both ocular vesicles are formed in close proximity and may appear with the cerebral mass through a central opening in the frontal bone at the root of the nose or in the nasal space. The two ocular vesicles may develop separate eyes, and as such appear in the central opening, or both vesicles may coalesce to form one eye, which may present at the same defective opening in the skull. This abnormality of growth is termed cyclopia, or synophthalmos. Associated with the latter condition is an absence of the nose as such, a rudimentary organ presenting as a cartilaginous and cutaneous attachment or stump to the ethmoid, and situated above the protruding central eye. When the nose is thus arrested in development in cases of cyclopia the condition is named arhinencephalia.

HYDROCEPHALUS INTERNUS. Many of the states of arrested cerebral development of which we have spoken, and of defective growth of the brain, are intimately associated with this morbid condition, but whether the association be one of cause or effect has not yet been satisfactorily determined.

The affection with which we are to deal is characterized by an excessive secretion of fluid in the cerebral ventricles, chiefly the lateral, by reason of which the brain becomes expanded, its walls thinned, and the cranial case greatly enlarged through distention and spreading of the cranial bones and scalp. It may be congenital or acquired. When the disease is acquired after ossification of the crainal bones, no enlargement of the head occurs, nor is there any spreading of the cranial bones.

Authors have divided the affection into two groups, hydrocephalus congenitus and hydrocephalus of the adults. While it is true that the clinical features of the two forms vary, owing to the differences in the cerebral and cranial development between childhood and adult life, the pathological process upon upon which each form depends seems to be similar if not identical. Such a division, therefore, appears to be unnecessary. We prefer to call this affection hydrocephalus internus. Tubercular meningitis was formerly called acute hydrocephalus, but since the last term has fallen into disuse there is no danger of confounding the two terms.

Some authors speak of an original accumulation of fluid in the sub-arachnoidean space, by which the brain becomes compressed and atrophied and the skull itself distended, a condition which they call hydrocephalus externus. But in view of the fact that the occurrence of such a condition, unless it be accompanied by an original defect or arrest of development in the brain, or some part of it, is denied by many, its description will not be treated of in this chapter.

Synonyms. Chronic hydrocephalus, leptomeningitis infantum, meningo-ependymitis, dropsy of the brain, water on the brain.

Effusion into the ventricular cavities may be the result of an arrested development of some portion of the brain, by reason of which, should the cranium not be implicated in the defect, a transudation of fluid occurs, either on the surface of the brain, into the subarachnoidean space, or in the interior into the ventricles, or into both. This is the result of physical causes and is a hydrops ex vacuo. This condition is sometimes called hydrocephalic-anencephalia.

Again, ventricular effusion may be the result of morbid growths in the vicinity of the ventricle. Closure of the foramen of Monro is said to produce an effusion into and a dilatation of the ventricle. Interference with the return circulation in the veins is also mentioned as a cause.

In the great majority of cases the effusion seems to be the result of an anomalous condition of the ependyma of the ventricles, by reason of which an increased amount of secretion or effusion takes place from its surface and fills the cavities, which it lines. In these cases the ependyma is thicker than normal, grayish in color, and it presents a finely beaded or granular condition, entirely distinct from its normal smooth, glistening appearance. In most of the congenital, and more markedly so in the adult cases, is this to be found. Whether the condition can be called an ependymitis is a great question, although the changes in the ependyma and the occasional presence of leucocytes and pus cells in the fluid would lead one to think that the process was inflammatory in character. It is improbable, however, that the embryonic or fœtal brain should be the seat of an inflammatory process.

However, we are still in the dark as to the causation of the disturbance of equilibrium between the vascular walls and the ependyma, which induces the former in these cases to transude the fluid constituent of the blood to such an excessive degree.

The affection in some cases seems to have an hereditary character, the disease appearing in a number of children of the same parents. Alcoholism on the part of the parents is also a supposed factor in its production, likewise is syphilis.

When the disease occurs after the birth of the child, traumatism has been noted in several cases to be the direct cause of its development.

It has also been known to develop after an attack of epidemic cerebrospinal meningitis.

Some of the congenital cases are associated with the developmental defect called porencephalia. This has already been described. (See page 319.)

The fluid which is contained in the ventricles is usually clear, slightly yellow, sometimes almost colorless, of low specific gravity, and contains only a trace of albumin, differing thus very little from normal cerebro-spinal fluid. In some cases, however, the specific gravity is higher and the amount of albumin considerable, varying from 3 to over 11 parts of albumin in 1000. It is owing to the differences in the physical and chemical properties of the fluid in these cases that led Huguenin[1] to believe that a decision as to inflammatoty or inflammatory origin of the fluid could be reached. While it is true that the fluid is sometimes cloudy and contains leucocytes and pus cells, it is a question whether these are the result of an original ependymitis or whether such an inflammation of the ependyma followed from the pressure and presence of an excessive amount of fluid. Huguenin insists upon the presence of a larger amount of albumin in the fluid, when due to inflammatory, and less than 2.5 per 1000 when due to pressure causes. Fagge,[2] who has given a masterly description of this affection, takes issue with Huguenin on this assumption, and mentions the notes of four cases of his own with an inflammatory condition of the ependyma in which only a trace of albumin could be found in the fluid. I am inclined to agree with Fagge in this regard; for it is a well-known fact that the amount of albumin in inflammatory fluids depends directly upon the cellular elements contained therein. The fluid in subacute pleurisy varies in this percentage owing to this cause, and no one will assert that this fluid when the percentage of albumin is small, is not the result of an inflammation of the pleura.

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[1] Huguenin: Ziemssen's Practice of Medicine, vol. xii.
[2] C. H. Fagge: Practice of Medicine, vol. i. p. 574, 1886.

The quantity of the fluid varies according to the length of time the disease has existed in each case. Thus, after death, as much as three gallons of fluid has been found in a case. Numerous cases have been reported in which six and eight pints of fluid were obtained.

Pathological Anatomy. As a usual rule the lateral ventricles are alone involved in the distention; occasionally the third and fourth ventricles participate. The lateral ventricles may be both equally distended, or one may be more distended than the other. Cases have been reported in which the one ventricle alone was expanded and the other apparently of normal size. In the last cases occlusion of the foramen of Monro has been found.

In well-developed cases the brain is distended to a mere shell, the convolutions are flattened, the sulci indistinguishable, and the whole cortex has the appearance of a membranous layer. Microscopical analysis of the cortex has not as yet been satisfactorily made, and observations are not sufficiently numerous to define absolutely the changes occurring therein. Thus far have been noted atrophy and obliteration of the ganglion cells, fusion and disintegration of the medullated nerve fibres.

The basal ganglia are flattened, spread out, and firmer than normal, as are the pons, crura, and the other anatomical structures at the base.

Sometimes the septum lucidum is obliterated, commonly in cases of enormous distention.

The cranial bones are thin, the diploe obliterated, and at times they are translucent. Owing to the intra-cerebral pressure exerted by the fluid, the sutures are separated, sometimes to three-quarters of an inch. The fontanelles are much enlarged and exceedingly tense. The scalp is involved in the distention and is very thin, and is stretched over the skull like a mere membrane. The veins shine through this very prominently, the skin of the scalp having a glistening, bluish appearance. The head reaches, at times, an enormous size, a circumference of 27 and even 32 inches having been noted. In cases of separation of the cranial sutures, *ossa triquetra* have formed in the membranous expansion between them, and in the fontanelles.

In the congenital cases, especially in those in which the third and fourth ventricles were distended, atrophy of the optic tracts, defective development of the pons, the medulla, and cerebellum have occurred, showing that the process had its beginning early in fœtal life.

The bones of the skull are pressed out, so that the frontal projects beyond the orbits at an oblique angle; its orbital plates are flattened, driving the eyes downward to such an extent that the cornea is almost entirely concealed by the lower lids, and the sclerotic above is visible. The occipital bone assumes a horizontal position, and the parietal and temporal are driven laterally to such an extent as to conceal the ears when the skull is viewed from above.

The cerebral meninges show few, if any, pathological changes; occasionally the pia of the base is found to be thickened. The choroid plexus is at times hyperæmic, thickened, swollen, and firmer than normal.

The changes in the ependyma have already been mentioned.

When hydrocephalus is acquired after the cranial bones are united the pathological changes in the brain and skull differ somewhat. Thus, owing to the lack of resiliency of the brain case, all the pressure effects are exerted on the brain cavities, so that the distention of all the ventricles, including the third and fourth, is common. The third not infrequently stands out from the base like a pendant bladder. For the same reason the amount of fluid contained in the ventricles rarely exceeds fourteen or fifteen ounces, as the external pressure seems to act as a check to any large transudation of fluid. Other changes are similar, but not as great in degree as in the co genital form.

Symptoms. Some of the congenital cases show even at birth a distenti of the fontanelles and an enlarged head. The head in these cases may be large as to form an obstacle to the delivery of the child. Not infrequen has rupture occurred during parturition, and death of the child ensued.

In the majority of cases the disease does not show a rapid developme until between the first and fifth months of extra-uterine life.

The head immediately attracts attention; it is large, globular, and giv the child great discomfort. Frequently can the child be seen laying its he upon its hand or supporting it upon a table. Infants are unable to rai their heads from the pillow or bed on which they lie. The skin of the for head is wrinkled into a constant frown, perhaps expressive of pain, for t afflicted patient often gives a moan or whining cry. Headache is complain of by older children. They are irritable and cross. The eyeballs protru somewhat and are directed downward, the cornea being for the most pa covered. Occasionally nystagmus is noticed.

Children with the disease well developed do not like to move about, eve if they can, for motion of the head produces in them vertigo, vomiting, an ofttimes convulsions.

Somnolency and drowsiness are common. The sight is impaired and blin ness, owing to atrophy of the optic nerves, not uncommon. Sometimes sigl is not affected and remains good throughout.

Motion is more or less diminished. There may be a complete paralysis all the limbs, or a paraplegia. Hemiplegia has been noted in a few case When it occurs it is probably due to some other local cause in the brain. Th child may be able to walk. Its gait, however, is shambling and tottering slow and methodical. All signs denote muscular weakness. The child i rising usually places his hands upon his knees, keeps his neck stiff to suppor his head, lifts his upper body with the strength of his arms exerted agains his knees, and then assumes the erect posture. In walking they frequentl hold their heads between their hands to steady it and support its weight.

The voice of older children is peculiarly strident and high-pitched.

The intelligence is usually impaired. When the disease begins in infancy the child shows no aptitude to acquire impressions. They do not learn t talk, to recognize their parents, although a high degree of idiocy is rare. Imbecility is the more usual status. Children with this affection are "backward," as it is termed. That is, they possess a degree of intelligence which belongs to a much younger period of normal life. If the disease be arrested in its course they can acquire speech, and learn to read and write.

Not unusually do tonic and clonic spasms of the limbs appear as well as nystagmus. General convulsions sometimes terminate the scene, or the child falls into a condition of coma, and an epileptiform convulsion ends its life.

The general nutrition of the child participates in the disease. Such children are emaciated, their skin pale and translucent, and generally wrinkled over the body owing to the loss of the subcutaneous fat. The body is usually moist and the head especially bathed in a profuse perspiration.

When the disease approaches a fatal issue the drowsiness increases, the whining, moaning cry appears at more frequent intervals, stubborn vomiting supervenes, and finally life ends with coma or convulsions.

Objective Signs. Fluctuation is easily elicited; sometimes a cracked-pot resonance may be obtained when percussion is made on the fontanelle and the patient is asked to open his mouth. In extreme distention, a light held on one side of the head will be diffused through it, and the glare can be distinguished when the eye of the observer is placed on the other side.

The symptoms of acquired hydrocephalus, when that disease develops after the total ossification of the cranial bones, are not as distinctive. In fact, there is nothing sufficiently characteristic to enable one to form a positive diagnosis.

After a traumatism, which is usually the exciting cause of the affection in the adult, headache is complained of, insomnia, and a loss or impairment of memory soon comes on. These may be soon followed by a succession of epileptiform convulsions, during which a hemiplegia may develop. In fact, hemiplegia in these cases is more frequently observed than the other forms of motor impairment; the hemiplegia may be general or partial. The pupils are mostly dilated and not responsive to light, or only sluggishly so. Sometimes they show an inequality in their dilatation. Strabismus not infrequently occurs. These patients suddenly lose consciousness and have no control of their sphincters, passing their urine and feces in bed. Sometimes an aphasia and complete loss of speech are present before unconsciousness makes its appearance. Coma may supervene or death come suddenly without coma or convulsions. The autopsy alone can demonstrate the cause of death in these cases, as it is impossible to make a diagnosis during life. The presence of the disease may be surmised, but not determined.

Course and Duration. The course of the disease is by no means constant. At times when it is present at birth, no progress is noted until a few months have elapsed. Again it may be rapidly progressive and terminate life within a few months. The disease may slowly progress, and then remain at a standstill, and finally recovery take place; or the progress may be rapid, a considerable enlargement of the head occur, and the infant grow to childhood, and death occur from some intercurrent affection. Rokitansky reports a case in which the distention was so great that spontaneous rupture took place through one of the sutures, the fluid escaping through the scalp and partly diffused itself underneath that covering, and ultimate recovery resulted. The disease may be fatal in early childhood, and occasionally it persists through adolescence even up to the fortieth year, although as a usual thing death results before the age of adolescence.

Prognosis. The prognosis is always grave, the affection usually ending in death. Length of life depends upon the rapidity of development and the amount of compression to which the brain is subjected.

Treatment. Little good is to be obtained by medicinal means. The best results have attended surgical measures. Those usually employed are strapping and compression of the head and draining of the fluid. For an account of the surgery of hydrocephalus see Chapter XXXIII on Surgery of the Brain.

CHAPTER XIII.

GENERAL DISEASES OF THE BRAIN.

(CONTINUED.)

BY F. X. DERCUM, M.D.

PACHYMENINGITIS.

The dura mater of the brain is a dense fibrous membrane consisting of two distinct layers, the outer of which is the periosteal covering of the internal table of the skull. The inner layer alone can be regarded as directly related to the brain. In harmony with these facts these layers are subject to somewhat different pathological processes.

THE AFFECTIONS OF THE OUTER LAYER consist of acute and chronic inflammation. The first occurs as the result of blows upon the skull, with or without fracture; and if air have access to the membrane, as through a wound, the inflammation may become purulent. Acute external pachymeningitis is largely a surgical affection, and does not require extended consideration here. In rare cases it makes its appearance without traumatic cause, and is then probably due to secondary infection from disease of surface structures, *e. g.*, erysipelas of the head or face. If it be the result of trauma, the history, the appearance of the wound, local pain and tenderness render the diagnosis easy. In the rare cases just mentioned the diagnosis is often difficult, and the affection may not be suspected until pus has collected in considerable amount, when focal symptoms, referable to compression or irritation of the cortex, may be present. Fever may or may not be noticed. The treatment, of course, consists in trephining over the affected area.

Chronic simple inflammation of the external layer is of considerable importance. Every now and then we notice in making autopsies that it is impossible to remove the calvarium without at the same time lacerating the dura or without the use of very considerable force. The dura in such cases is firmly adherent to the calvarium, and when we examine the latter we find that it is thicker, denser, and heavier than normal, and that the spongy structure or the diploë has largely disappeared. In other words, there has been an increase in osseous tissue throughout. Chronic inflammation of the external dura is therefore equivalent to *chronic external ossific pachymeningitis*. The existence of this affection is frequently overlooked during life, though occasionally it is of great clinical importance. A certain number of permanent headaches have their explanation in this condition. Among the few neurologists to recognize this fact is S. Weir Mitchell. He places as among the probable causes, trauma, which in young children may be comparatively slight. Thus he says: "A slight injury causes in the young hyperostosis; the bone remains somewhat thickened, with no marked depression, but there is a limited area of adhesions of the dura without clear evidences of present inflammation. These conditions may give rise to nearly constant pain, great sensitiveness of head, incapacity for study, and frequent unendurable additions to the permanent distress."[1]

Instead of being comparatively limited, it may in given cases be widely diffused. It is so found occasionally in autopsies in chronic insanity, epilepsy, trauma of long standing, and in old age. Often it is unassociated with any special symptoms, and is unsuspected during life. However, as we have seen, the possibility of its occurrence should always be considered in chronic headaches, especially if they are more or less limited in distribution and have followed blows upon the skull. The fact that external signs, such as depression or scars, are absent does not militate against the diagnosis. A symptom of value in these cases is tenderness upon deep firm pressure, and upon percussion of the skull. My own observations have convinced me that the condition follows trauma far more frequently than is supposed.

If traumatic external meningitis be severe it does not remain limited to the external layer, but affects the internal dura as well. In such case it also frequently involves the subjacent pia-arachnoid. Bands of adhesions unite the surface of the dura with the soft membranes, and the latter show markedly the signs of inflammatory infiltration. A typical instance of a traumatic meningitis of such severity may make clear the character of this affection.

A man, thirty years of age, fell a distance of some twelve feet, striking upon the head, a little to the left of the vertex. After the immediate symptoms had passed away, though there was not even a laceration of the scalp nor any depression of the skull, chronic headache, referred to the seat of the blow, persisted. This was constant and severe, and failed to yield to internal medication or to active counter-irritation. Finally, epileptic seizures developed which were focal in character. Two years afterward the man died of an intercurrent affection, and the autopsy revealed beneath the site of the injury a thickened dura adherent to the calvarium. It was also adherent by delicate bands to the subjacent pia-arachnoid, which was infiltrated and thickened. Careful examination of the calvarium failed to reveal any trace of fracture. Cases like the above are exceedingly important, inasmuch as they may be improved by early surgical interference.

Traumatic meningitis may therefore involve either the external dura alone, or may involve both layers of the membrane and also the pia-arachnoid. It is not always possible during life to determine whether a meningitis resulting from a trauma involves merely one or all of the membranes. If the pia-arachnoid be involved we may have in addition to circumscribed, constant headache, also the symptoms of focal disease of the brain, such as were presented in the case just cited. Neither is it possible to decide whether inflammation of the membranes is the only lesion present, whether or not there has also been a fracture, with, perhaps, the projection of a spicule of bone from the internal table. In the latter instance, there may be reflex epilepsy due to the constant irritation of the dura, and this, too, when the pia-arachnoid and the cortex are not directly involved. The diagnosis of a given case must, of course, be based upon the special symptoms presented.

Treatment. If the diagnosis of traumatic meningitis has been made, most active treatment should be instituted. Internal medication is of slight benefit. In this conneetion the iodides, bromides, and mercurials naturally suggest themselves, and it is well always to make a thorough trial of them. More important, however, is active counter-irritation. This should consist

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[1] S. Weir Mitchell: "Permanent Headaches," International Clinics, October, 1891, p. 273.

CHAPTER XIII.

GENERAL DISEASES OF THE BRAIN.

(CONTINUED.)

BY F. X. DERCUM, M.D.

PACHYMENINGITIS.

The dura mater of the brain is a dense fibrous membrane consisting of two distinct layers, the outer of which is the periosteal covering of the internal table of the skull. The inner layer alone can be regarded as directly related to the brain. In harmony with these facts these layers are subject to somewhat different pathological processes.

THE AFFECTIONS OF THE OUTER LAYER consist of acute and chronic inflammation. The first occurs as the result of blows upon the skull, with or without fracture; and if air have access to the membrane, as through a wound, the inflammation may become purulent. Acute external pachymeningitis is largely a surgical affection, and does not require extended consideration here. In rare cases it makes its appearance without traumatic cause, and is then probably due to secondary infection from disease of surface structures, *e. g.*, erysipelas of the head or face. If it be the result of trauma, the history, the appearance of the wound, local pain and tenderness render the diagnosis easy. In the rare cases just mentioned the diagnosis is often difficult, and the affection may not be suspected until pus has collected in considerable amount, when focal symptoms, referable to compression or irritation of the cortex, may be present. Fever may or may not be noticed. The treatment, of course, consists in trephining over the affected area.

Chronic simple inflammation of the external layer is of considerable importance. Every now and then we notice in making autopsies that it is impossible to remove the calvarium without at the same time lacerating the dura or without the use of very considerable force. The dura in such cases is firmly adherent to the calvarium, and when we examine the latter we find that it is thicker, denser, and heavier than normal, and that the spongy structure or the diploë has largely disappeared. In other words, there has been an increase in osseous tissue throughout. Chronic inflammation of the external dura is therefore equivalent to *chronic external ossific pachymeningitis*. The existence of this affection is frequently overlooked during life, though occasionally it is of great clinical importance. A certain number of permanent headaches have their explanation in this condition. Among the few neurologists to recognize this fact is S. Weir Mitchell. He places as among the probable causes, trauma, which in young children may be comparatively slight. Thus he says: "A slight injury causes in the young hyperostosis; the bone remains somewhat thickened, with no marked depression, but there is a limited area of adhesions of the dura without clear evidences of present inflammation. These conditions may give rise to nearly constant pain, great sensitiveness of head, incapacity for study, and frequent unendurable additions to the permanent distress."[1]

Instead of being comparatively limited, it may in given cases be widely diffused. It is so found occasionally in autopsies in chronic insanity, epilepsy, trauma of long standing, and in old age. Often it is unassociated with any special symptoms, and is unsuspected during life. However, as we have seen, the possibility of its occurrence should always be considered in chronic headaches, especially if they are more or less limited in distribution and have followed blows upon the skull. The fact that external signs, such as depression or scars, are absent does not militate against the diagnosis. A symptom of value in these cases is tenderness upon deep firm pressure, and upon percussion of the skull. My own observations have convinced me that the condition follows trauma far more frequently than is supposed.

If traumatic external meningitis be severe it does not remain limited to the external layer, but affects the internal dura as well. In such case it also frequently involves the subjacent pia-arachnoid. Bands of adhesions unite the surface of the dura with the soft membranes, and the latter show markedly the signs of inflammatory infiltration. A typical instance of a traumatic meningitis of such severity may make clear the character of this affection.

A man, thirty years of age, fell a distance of some twelve feet, striking upon the head, a little to the left of the vertex. After the immediate symptoms had passed away, though there was not even a laceration of the scalp nor any depression of the skull, chronic headache, referred to the seat of the blow, persisted. This was constant and severe, and failed to yield to internal medication or to active counter-irritation. Finally, epileptic seizures developed which were focal in character. Two years afterward the man died of an intercurrent affection, and the autopsy revealed beneath the site of the injury a thickened dura adherent to the calvarium. It was also adherent by delicate bands to the subjacent pia-arachnoid, which was infiltrated and thickened. Careful examination of the calvarium failed to reveal any trace of fracture. Cases like the above are exceedingly important, inasmuch as they may be improved by early surgical interference.

Traumatic meningitis may therefore involve either the external dura alone, or may involve both layers of the membrane and also the pia-arachnoid. It is not always possible during life to determine whether a meningitis resulting from a trauma involves merely one or all of the membranes. If the pia-arachnoid be involved we may have in addition to circumscribed, constant headache, also the symptoms of focal disease of the brain, such as were presented in the case just cited. Neither is it possible to decide whether inflammation of the membranes is the only lesion present, whether or not there has also been a fracture, with, perhaps, the projection of a spicule of bone from the internal table. In the latter instance, there may be reflex epilepsy due to the constant irritation of the dura, and this, too, when the pia-arachnoid and the cortex are not directly involved. The diagnosis of a given case must, of course, be based upon the special symptoms presented.

Treatment. If the diagnosis of traumatic meningitis has been made, most active treatment should be instituted. Internal medication is of slight benefit. In this connection the iodides, bromides, and mercurials naturally suggest themselves, and it is well always to make a thorough trial of them. More important, however, is active counter-irritation. This should consist preferably of superficial burns made with a white-hot iron, or a Paquelin cautery, over the back of the neck. It is my own custom to make such a burn the size of a silver half dollar, and to destroy simply the superficial layers of the epithelium; then to allow the burn to heal rapidly, and in the course of a week or ten days to burn again. Occasionally it is expedient to make the first burn rather high up, close to the occiput, and to make the second burn a little lower down, but also in the median line. In this way, by making the second burn before the first has had time to heal, and a third before the second has healed, an almost constant active counter-irritation may be maintained. In a limited number of cases I have seen decided relief follow this measure. However, in no instance of well-marked traumatic meningitis have I had reason to believe that a cure had been effected. By all means the most satisfactory way of dealing with these cases is surgically. We can never be absolutely sure that we have only a traumatic meningitis to deal with. It is always possible that the trouble is accompanied by some affection of the internal table, and, therefore, trephining is not only indicated for its therapeutic effect, but also as an exploratory and precautionary measure. It is impossible to foretell whether a given case will be limited to simple chronic local headache, or whether sooner or later there may not be serious complications, such as epilepsy. If trephining be performed for an uncomplicated traumatic meningitis it will be sufficient simply to remove a button of bone without interfering with the dura. The button of bone should not be replaced. If the meningitis be diffused over a rather large area it may be wise, as was done in one of my own cases, to make two small trephine openings at some distance apart. Just in what manner trephining produces amelioration of the symptom is not clear. However, it is probable that the new conditions under which the inflamed membrane is placed, such as the relief of local pressure and altered local circulation, may be the agencies at work. Certain it is that the relief which accompanies this procedure is very decided.

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[1] S. Weir Mitchell: "Permanent Headaches." International Clinics, October, 1891, p. 273.

A variety of chronic external pachymeningitis which has been met with in a few instances needs passing mention. Thus far but two observers have described it, Lannelongue[1] and Alexis Thompson.[2] In every instance the patient has been a child, and its discovery has been either during the performance of an operation, such as craniectomy, or upon the post-mortem table. As in the ordinary form, the dura is very firmly adherent to the calvarium; on its external layer, however, are seen numerous granulations, reddish in color, which are imbedded in the bone; the latter, instead of being thickened, becomes very much thinned, in other words, the inflammation, instead of being attended by deposit, is attended by absorption of bone. The affection is exceedingly rare, and its symptomatology is as yet obscure.

THE INNER LAYER OF THE DURA MATER is far less frequently diseased than the outer layer. However, it is at times, as we have seen, the seat of inflammation the direct result of trauma, being in such cases involved concomitantly with the outer layer. Occasionally such a traumatic inflammation is adhesive, and more or less marked bands of union may be formed with the subjacent pia. Extensive adhesion is, however, rare. Adhesions between the inner layer and pia-arachnoid also occur as the result of syphilitic inflammation. The inner layer of the dura may likewise be the seat of purulent inflammation, and in such instances there is generally a similar involvement of the pia-arachnoid, and the symptoms cannot be separated from those of purulent leptomeningitis. Finally, at the post-mortem table we not rarely find bony deposits in the internal layer, more especially in its prolongations, for example, in the falx. This condition is rather of pathological than of clinical interest.

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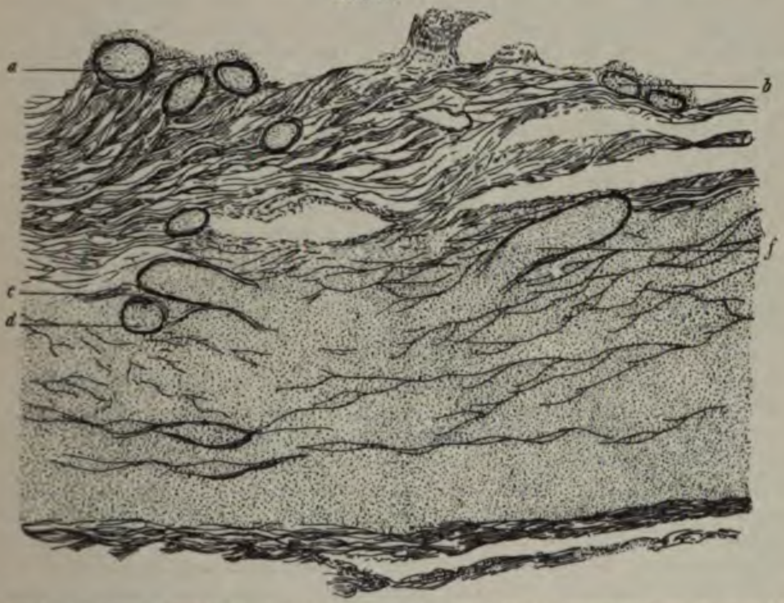
[1] Lannelongue: Congrès Francais Chirurgical, 1891, p. 75.

[2] Alexis Thompson on Pachymeningitis Chronica Externa, Journal of Pathology and Bacteriology, 1893, October 9, p. 75.

Almost all writers describe as a disease of this membrane a condition variously known as internal hemorrhagic pachymeningitis, hæmatoma of the dura, or arachnoid cyst. Every now and then, in making autopsies upon the brain, we find the inner layer of the dura covered by more or less dense layers of reddish fibrin. The deposit looks like an exudation of blood undergoing organization. The exudation can frequently be stripped in layers from the inner dura without difficulty. Occasionally, however, instead of finding merely this deposit, cysts of greater or less size are met with, and these being opened are found to contain altered blood. Virchow and others have considered this affection as inflammatory in origin. Later investigators, however, notably Wigglesworth, Hoyt, Whittaker, and the writer, have attributed the condition to a subdural hemorrhage. It is exceedingly probable that the latter view is correct. The affection, which in itself is rare, is most frequently found among the chronic insane, in just that class of patients in whom degeneration of the bloodvessels is apt to be marked. That a simple escape of blood into the subdural space is sufficient to produce the lesions of this so-called hemorrhagic pachymeningitis has been proved by Sperling, who injected blood into the subdural space in animals. It is probably a matter of little consequence as to whether the initial hemorrhage come from the pia or from the dura. It is true that the exudation is most frequently found more or less adherent to the dura. However, that the hemorrhage does at times come from the pia has been absolutely demonstrated by Hoyt. It appears that after the exudation has taken place that some attempt at organization is made (see Fig. 77), and that sooner or later vessels

FIG. 77.



Section of false membrane in hemorrhagic pachymeningitis, showing excessive vascularity of dura at *a* and *b*, and newly formed vessels at *c*, *d* and *f*.

are formed with poorly differentiated walls, which, in turn, give rise to renewed bleedings. Such vessels are readily seen in microscopic sections of the exudation in the dura. Hoyt in one instance has also traced such a vessel in the exudation from the pia. As to the cause of these hemorrhages, writers agree in attributing them to the degeneration of bloodvessels and especially to the lessened intra-cranial pressure, which, in all probability, accompany degeneration and atrophic changes in the convolutions. That this, however, is not the entire truth is proved by an observation published some years ago by the writer. In this instance there was no mental disease whatever, no degenerative affection of the nervous system, and, notwithstanding, large hæmatomata of the dura were found covering both hemispheres at the autopsy. In this instance, also, the affection was not limited to the internal dura, but affected the external dura as well, and with it the calvarium, that is, the vessels of the external dura were exceedingly enlarged and the calvarium was soft and spongy and riddled with a large number of foramina containing distended vessels.[1] It is probable, therefore, that there are other agencies at work in the production of this affection, agencies which are trophic in character, and the writer has advanced the view that hæmatoma of the dura is really trophic in origin, and that it is in every way comparable to such allied conditions found in the insane as hæmatoma of the cartilages of the ear or of the cartilages of the nose.

As has been stated, the condition has been most frequently met with in autopsies of cases of chronic and terminal insanities. Occasionally, however, it follows an old trauma of the head, and sometimes a sunstroke. It is also occasionally met with in alcoholic subjects. It has at times followed exhausting fevers, and rarely is met with in autopsies upon cases of anemia. Doehle[2] describes the occurrence of a pachymeningitis, which, from the description, was undoubtedly hæamatoma of the dura, in young children. Northrup[3] adds similar cases to the literature.

Owing to the nature of the affection it is rarely recognized during life. If met with in patients who are not insane, or at least are able to give an account of their symptoms, we find headache most frequently complained of. The fact that the headache is continuous would, of course, suggest meningeal or other serious organic disease. Special symptoms may be present, depending upon the locality and the extent of the exudation. Thus, for instance, if the exudation be large and limited to one hemisphere, a more or less marked hemiplegia may be the result. Such a hemiplegia may be ushered in by an increased headache, vomiting, somnolence, coma, convulsions, conjugate deviation of the eyes, contracted pupils, or nystagmus. Instead of the hemiplegia there may be simply a general muscular weakness. Eye-ground changes are not often present, though optic neuritis, it is claimed, occasionally occurs. These symptoms, however, may be caused by various lesions, and the diagnosis is at best unsatisfactory. Should it, however, be reasonably established, surgical measures should be instituted (see Chapter XXXIII), and if doubtful, a treatment based upon general principles should be adopted. This should include absolute abstinence from alcoholic stimulants, a carefully regulated diet, laxatives, alteratives, and possibly counter-irritation to the back of the neck.

Rarely pachymeningitis is tuberculous. Such a case is recorded by Gussenbauer[4] in which the disease followed a blow upon the skull in a man with

[1] Dercum: University Med. Mag., October, 1889.

[2] Doehle: Ueber Chronische Pachymeningitis bei Kinder, etc. Verhandlg. d. X. Internationalen Congresses, 1890, Bd. v. Abthl: xvii. p. 40. Doehle, in 395 autopsies on children below the age of ten, found 48 instances; of these, 30 occurred in infants under one year.

[3] Northrup: Journ. New York Path. Soc., 1891, pp. 59-67. Three cases in starved and emaciated infants.

[4] Gussenbauer: "Ueber Pachymeningitis Circumscripta" Prag. Med. Wochenschr. 1892, xvii. 91.

a bad family history. The dura may also be involved in syphilitic disease of the cranial bones. Primary specific pachymeningitis is generally diffuse and characterized by headache, and often by tenderness of the skull on percussion. Other syphilitic stigmata are present. (See Chapter XXIV.) In children pachymeningitis appears to be occasionally due to inherited syphilis.[1]

LEPTOMENINGITIS.

Because of their close anatomical relations inflammations of the arachnoid and pia mater are considered together under the name of leptomeningitis. These inflammations are various in character. A leptomeningitis may be simple, purulent, tuberculous, or syphilitic, or it may be secondary to pneumonia, scarlet fever, typhoid fever, ulcerative endocarditis, measles, variola or septicæmia. Further, it may be rheumatic in origin or the result of some toxic agent, as alcohol. Sunstoke is another undoubted factor in the production of meningitis. Traumata of various kinds, especially blows upon the skull, are also not infrequent causes of meningitis. Such a meningitis may or may not be accompained by a wound or fracture of the skull. Various diseases of the cranial bones and of the sinuses of the dura mater may give rise to meningitis by contiguity. A very common instance of this is the meningitis which has its origin in disease of the petrous portions of the temporal bone. Finally meningitis may be present without special or discoverable cause, and in such case is spoken of as idiopathic.

Every now and then a cerebral leptomeningitis invades also the pia-arachnoid of the spinal cord, and in such a case the symptoms closely resemble those found in the epidemic form of cerebro-spinal meningitis. (See page 203.) Leptomeningitis varies greatly as to its distribution. It may be widely diffused, but is generally most marked over some one portion of the brain, and at times is even limited to certain areas. Thus it is occasionally most marked or limited to the vertex, in which case we speak of a vertical meningitis. At other times it is most marked at or limited to the base, and in this instance we speak of a basal meningitis; or, it may be limited to some area either of the base or vertex, and then it is spoken of as a local meningitis.

Prominent among predisposing causes are general ill-health, mental overwork, and especially the ages of early childhood and youth and the male sex. It is well known that children suffer far more frequently from meningitis than adults, and boys more than girls.

Symptomatology. When we reflect that the etiology of the affection is so various, and when we consider the great extent of the meninges, that they cover not only the lateral and mesial surfaces of the brain, but also line it at the base, and further are intimately related to the various cranial nerves, we can readily understand that the symptoms and course should vary greatly in different cases. We see at once that, while there may be general symptoms common to all forms, the detailed symptoms depend largely upon the character of the inflammation and especially upon the locality to which the disease is limited or at which it is most accentuated.

In order that we may intelligently study the subject it will be well to consider the symptoms present in the various diffused forms of meningitis. The patient may present for a variable period preceding the more profound symptoms, general malaise, hebetude, and irritability; but at times the onset is sudden, being ushered in by a chill. Among the earlier symptoms are vertigo and vomiting. Soon fever makes its appearance. The headache is almost always of great severity and is unceasing. At the same time the patient becomes hyperæsthetic to touch, to noises, and to bright light. The general surface of the body is everywhere very sensitive, as are also certain muscles, especially those of the back of the neck and of the calves. The vomiting occurs independently of the presence of food in the stomach or of any attack of indigestion. In a short time delirium, muscular twitchings, and, perhaps, convulsions manifest themselves. The tongue at first shows nothing peculiar, but later in the general adynamic condition it becomes heavily furred. There is also obstinate constipation and retraction of the abdomen. Retention of urine is frequent. As the case progresses somnolence and coma may supervene, together with involuntary evacuations of urine and feces. The temperature in the average case is not very high, ranging generally from 100° to 102°, although sometimes as much as 105° or more is noted. The pulse, instead of being increased in frequency, as we would naturally expect it to be, is markedly slower than normal, its rate falling to seventy, sixty, or fifty beats to the minute, or even less. In addition it is frequently irregular. If the case goes on from bad to worse the rate finally increases, and at last becomes excessively rapid. The respiration is at first but little affected. Later it becomes frequent, sighing and interrupted, and may even assume the character of the Cheyne-Stokes respiration. In addition to these symptoms others are present depending upon the special seat of the inflammation. Thus we are apt to have signs of involvement of various cranial nerves. This is true especially of the ocular nerves, strabismus being as a rule an early symptom. In addition there is almost always some modification of the pupils. At first contracted, they may later on be unequally dilated. Sometimes there is ptosis, though this is generally slight. There may also be signs of involvement of the facial nerve, and there may be more or less distinct facial palsy. Instead of paralysis, however, marked twitchings or spasms may be present. If the inflammation involve the posterior cranial fossa there is more or less marked spasm or rigidity of the muscles of the back of the neck. This symptom is rarely if ever absent in posterior basal meningitis, and is one of the most valuable guides that we have. The head is retracted and nearly immobile; attempts at movement and handling of the neck give rise to pain. Further, trismus, gritting of the teeth, rigidity of the muscles of the abdomen, spasms of the muscles of a limb or localized convulsive movements may also be present. Optic neuritis, and if pressure be great, as from extensive exudation, blindness may also supervene.

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[1] Moussons: Journ. de Méd. de Bordeaux, 1891-2, xxi. 3, 17.

The knee-jerks are at first increased, but later diminished, and finally lost. Vasomotor disturbances are occasionally noted. Thus, if the skin be lightly stroked by the finger a persistent red streak may make its appearance. This is the so-called "tâche cérébrale." It depends upon a weakness of the vasomotor apparatus, and is not pathognomonic of meningitis, but is found in other adynamic conditions. Small reddish spots may make their appearance spontaneously. Herpetic eruptions also occur, and bed-sores are not infrequent. Occasionally albumin or sugar is found in the urine, and at times there is an actual or relative increase in the amount of phosphates eliminated. Rapid general emaciation also is apt to take place, and is often one of the most alarming symptoms of the disease.

The detailed symptoms in a given case depend largely upon the position of the inflammation. When the inflammation is most marked at the base, vomiting, basal or occipital headache, more or less marked interference with the various cranial nerves, and especially rigidity of the muscles of the back of the neck, are prominent symptoms. Optic neuritis, is also likely to be present, coming on, as a rule, after the affection has lasted for some time, a week or more. Occasionally a paresis of one side, or even a well-marked hemiplegia, is noted. It is due either to pressure upon the crus of one side or to interference with the vascular supply of one of the hemispheres. Aphasia is also occasionally present.

In meningitis of the vertex, delirium and convulsions are perhaps more marked than in meningitis of the base. Headache is also present and is very severe. It is referred to the top of the head. Vomiting is much less frequent in verticalar meningitis than in the basal form. Gross motor disturbances, such as palsies of an entire limb and well-marked hemiplegias, are more apt to occur. Disturbances of the various cranial nerves are much less frequent. Optic neuritis also may remain entirely absent or occur only as a very late symptom; similarly rigidity of the back of the neck may be wanting.

Limited forms of meningitis are to be distinguished by the fact that the headache is referred to some one portion of the head, and that there are often focal cerebral symptoms, such as disturbances of motor centres or interference with special cranial nerves.

The various forms of meningitis present special features.

PURULENT MENINGITIS. In purulent meningitis the onset is apt to be rapid, and is often marked by a chill. The symptoms soon become pronounced, and when once established the case frequently runs a very rapid course, a fatal termination may take place in twenty-four or forty-eight hours, though at other times the duration of the disease may extend to a week and sometimes longer. The temperature is apt to run higher than in other forms, and may be very irregular. Optic neuritis is infrequent.

SEROUS MENINGITIS, to which Quincke[1] has especially called attention, is characterized by a rather mild and prolonged course, a short febrile period and symptoms indicating diffuse pressure rather than localized symptoms. Headache and rigidity of the back of the neck are less pronounced than in the purulent form. Optic neuritis, on the other hand, is much more common.

TUBERCULOUS MENINGITIS is characterized in the vast majority of cases by a very gradual onset. For days and weeks preceding the outbreak of symptoms the child seems to be in ill health. It seems to be growing thinner, is irritable, nervous and suffers from broken sleep, and often complains of headache. It is only after these symptoms have persisted for a long time that the actual signs of meningitis are noted. However, rarely, the onset, instead of being gradual, is quite sudden, vomiting and convulsions being among the earliest symptoms. In tubercular meningitis, further, the headache is intense and agonizing, and when disturbed the child often gives vent to a shrill scream, the so-called "hydrocephalic cry." Signs of basal trouble, such as gritting of the teeth, strabismus, etc., are common. Optic neuritis, also, is present. Regarding the ophthalmoscopic appearances, it is also of importance to remember that if tubercles be detected in the choroid that the diagnosis must be considered as established The temperature is but slightly raised and pursues an uneven course. The pulse is at first slow, but may suddenly increase in rapidity, especially in the later stages. It is subject to considerable variation. The respiration, likewise, becomes frequent and irregular. The disease may run an acute and rapid course, the case terminating fatally at the end of a few days. Much more frequently, however, its course is subacute and chronic, the duration extending over two, three, four or many weeks. It may, indeed, be exceedingly protracted, and temporary remissions may even take place.

SYPHILITIC INFLAMMATIONS of the pia-arachnoid are in the majority of cases chronic, though cases of acute specific meningitis have been described.

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[1] Quincke: Sammlung Klinischer Vorträge, 1893, No. 67, p. 655.

Specific meningitis is rarely, if ever, diffuse, but almost always localized. It often consists of welt-like or flattened gummatous formations. The symptomatology varies according to the locality of the disease. Most frequently, however, we find the lesion situated at the base, and more pronounced on one or the other side. Basal headache with localizing signs of pressure are, therefore, among the most constant symptoms. A gummatous inflammation of the membranes at the base may involve the vessels of one hemisphere of the brain, and frequently also one or more of the cranial nerves, especially the oculo-motor. The symptoms, therefore, are often those of a crossed hemiplegia; the leg and arm are paralyzed on the opposite side, while the oculo-motor or other cranial nerve is paralyzed on the same side as the lesion. If the disease be situated upon the upper surface of the brain, symptoms indicative of interference with various cortical centres may be present. Common among these are palsies limited to one limb, convulsive attacks resembling focal epilepsy, local headache and tenderness to deep pressure or percussion. If the disease be not well localized, but rather diffuse, nocturnal headache, somnolence, and impairment of the mental faculties may be the only symptoms presented. Optic neuritis may also exist in specific meningitis as in other forms of organic intra-cranial disease. For a more detailed account of the symptoms the reader is referred to Chapter XXIV.

ALCOHOLIC MENINGITIS is chronic in character, and is simply to be looked upon as one of the many lesions to which alcoholics are liable. The affection is vertical and diffuse, and therefore focal symptoms are lacking. Constant headache, dull in character, and dementia, slight or marked, together with an alcoholic history, are the features presented. Convulsions resembling ordinary epilepsy also frequently occur. (See also page 191.)

SIMPLE MENINGITIS is not readily distinguished from the other forms. However, cases of meningitis are constantly met with in which, at the autopsy, neither pus, tuberculous deposit, nor other specific cause is observed. It is probable that of these cases some at least are due to undiscovered general infection. For practical purposes, however, simple meningitis must be admitted into our classification. It is found mainly as an acute affection. In rare instances, however, a chronic form, basal in distribution and symptoms, is met with among infants. It may exist for months and in its milder degrees for much longer periods. In these cases the possibility of inherited syphilis is always to be considered.

MENINGITIS SECONDARY TO DISEASE OF A CONTIGUOUS STRUCTURE is generally localized in character. It is to be distinguished by localized headache and various focal symptoms. A common instance of such a meningitis is furnished by the basal meningitis following necrosis of the temporal bone in otitis media. Here intense basal headache on the side of the affected ear, facial palsy, and perhaps involvement of the ocular nerves, may be the symptoms presented. Often this form of meningitis is complicated by the symptoms of thrombosis of the lateral sinus (see page 366), or by the symptoms of brain abscess. (See page 374). Very rarely a basal meningitis, instead of being due to necrosis of the temporal bone, is due to necrosis of the sphenoid.[1]

Pathology and Morbid Anatomy. The pia-arachnoid may present in cases of meningitis the following peculiarities: First, it may be the seat of more or less milky or white opacities, which are indicative of chronic thickening or simple chronic leptomeningitis, such as is found in alcoholic subjects. Frequently this condition is accompanied by œdematous infiltration of the membranes and by loose attachment of the membranes to the convolutions, and by the formation of subpial lymph spaces here and there; at times, also, by a marked increase in the size and number of the Pacchionian bodies. Secondly, the membranes may be injected and hyperæmic and the seat of serous exudation, and may be slightly clouded. This is a condition sometimes met with in meningitis secondary to the exanthemata. Far more commonly, however, the signs of acute meningitis are more marked, and the meshes of the pia-arachnoid are infiltrated with yellowish exudation. This exudation is purulent, and is most frequently found along the course of the veins. The latter are often outlined by yellowish or yellowish-white borders. Occasionally, however, when the pus is on the surface of the membranes, the veins are entirely hidden from view. These changes are as a rule more marked over the vertex, though they may be found at the base. Collections of pus are also found in the sulci and around the nerve-roots and, at times, over the surface of the dura. Distinct inflammation of the intra-cranial portions of various nerves may also be present, and there may be marked infiltration or even minute hemorrhages into their sheaths. The cortex of the brain may be reddened here and there, or it may reveal minute areas of softening. The ventricles may be somewhat dilated and filled with clouded lymph. The ependyma may also show signs of inflammation, and may be slightly granular and thickened.

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[1] Mackenzie, St. Thomas Hosp. Rep., 1889-90, N. S. xix. p. 350.

In tuberculous meningitis we note in the pia-arachnoid of the base, rarely of the vertex, a yellowish gelatinous infiltration. This is at times firm, at times soft. Especially is it seen about the optic chiasm, and it may extend for some distance into the Sylvian fissure on either side. Small tubercles can, as a rule, be readily detected along the base of the frontal lobe and island of Reil, where they follow the course of the vessels, and are less obscured by the infiltration of the surrounding tissues. Instead of these appearances, we may simply have a very diffuse infiltration of the membranes. The lateral ventricles are much dilated in the majority of cases. This may be either due to direct tuberculous infection of the ependyma or to closure of the foramen of Majendie.

In syphilitic inflammation the membranes may be the seat of more or less extensive flat, gummatous formations. Sometimes the gummata are large, and present signs of caseous degeneration. At other times they are dense and fibrous. They are most frequently found at the base.

Bacteriological examination has revealed in the epidemic form of cerebrospinal meningitis the presence of the diplococcus described by Leyden in 1883. Fraenkel identified this germ with the pneumococcus in 1886. This fact is exceedingly interesting when we remember that meningitis is not infrequently secondary to pneumonia. In such cases the diplococcus has also been found in the meningeal exudation. The streptococcus pyogenes has been found in the purulent form, though Zoerkendoerfer[1] holds that the latter is most frequently caused by the pneumococcus.

In the various secondary forms of meningitis the infection may be carried to the membranes either by the bloodvessels or by the lymphatics.[2] In the meningites complicating ulcerative endocarditis, typhoid fever, typhus and relapsing fevers, cholera, variola, scarlet fever, measles, pneumonia and septic infection generally, the germs probably gain access to the membranes through the blood. In ulcerative endocarditis it is probable that a minute fragment of valve containing pyogenic micro-organisms is carried to the meninges, and that as a result there is a small hemorrhagic enfarct. Inflammation is thus set up which by extension leads to more or less general involvement of the meninges. It is possible also that other cases are due to general infection of the blood with micro-organisms. In typhoid fever it is probable that the meningitis is generally due to a secondary septic infection; in some cases probably to the typhoid bacillus itself. Regarding typhus fever, it is probable that the meningitis is generally due to streptococcus infection. This is probably, also, the case in relapsing fever. In variola we must remember that in addition to meningitis arising from general infection we may also have a meningitis the result of middle-ear disease. This is also true of scarlet fever and measles. Meningitis complicating pneumonia is generally due to the pneumococcus of Fraenkel, as just pointed out. Post-mortem diagnosis of this meningitis can, it is said, be made by simple inspection.[1] The pus is thick, viscid and consistent on account of the abundance of fibrin present. The various forms arising in the course of general septic infection are all streptococcus meningitis.[2]

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[1] Zoerkendoerfer: Prag. Med. Wochenschr., 1893, vol. xviii. p. 211.

[2] Huguenin: Correspondenzbl d. Schweizer Aerzte, 1890, vol xx. p. 789.

The various forms of meningitis having their origin in the lymph stream are almost always due to streptococcus infection. The course may be that of the lymphatics of the sheaths of the various cranial nerves and bloodvessels. Erysipelas of the face and head, carbuncles, etc., may give rise to meningitis by infection through the veins.

To the above channels of infection we should also add cerebral meningitis arising by extension from spinal meningitis. Such cases may occur as a result of infection from bed-sores, coxitis, sacral diseases, etc.

Many of the symptoms can be readily explained by the lesions found. Thus it is probable that the headache is caused by the direct pressure of the swollen membranes upon the sensitive dura mater. Similarly, the symptoms of interference with the various nerve-trunks are due to pressure and irritation and to the infiltration of their sheaths. It is exceedingly probable that the rigidity of the muscles of the back of the neck is due to the irritation of the roots of the spinal accessory nerves, and in some cases also to irritation of the roots of the upper cervical nerves. The delirium, convulsions and coma are to be ascribed to a direct interference of the inflammation and exudation with the brain cortex. The slowing of the pulse can reasonably be ascribed to irritation of the vagus.

Diagnosis. The recognition of meningitis, especially of the basal form, is as a rule not difficult. Various special symptoms, however, may be absent, and at times, though rarely, headache is not marked. In such cases it is necessary to proceed with caution. The entire absence of headache, however, should always throw doubt upon the diagnosis, as headache is one of the most constant symptoms. Vomiting is also a rather constant symptom, especially in the basal form. Coma, delirium and convulsions, however, are common to a number of affections, and here difficulties may occur.

Thus the question may arise as to whether a given case is suffering from uræmia or meningitis. A moment's reflection will convince us that these two affections present numerous symptoms in common. Among these are headache, vomiting, vertigo, delirium, coma, convulsions, either general or limited, and even local or hemiplegic palsies. Further albumin is occasionally present in meningitis. Should, however, a given case present the signs of acute nephritis, that is, numerous tube-casts and blood corpuscles, œdema of the face and of the extremities, the question would be easy of solution. The more chronic forms of renal trouble would reveal by the ophthalmoscope albuminuric neuro-retinitis. Difficulty could only arise in the vertical form of meningitis, and even here the age of the patient and the history of the affection would aid us.

Sometimes it is difficult, to differentiate meningitis from typhoid fever.

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[1] Hutinel: Semaine Méd. Par., 1892, vol. xix. p. 776.

[2] Huguenin, loc. cit.

We have here, however, such points to serve us as the temperature-curve of typhoid fever, the regular pulse, the enlargement of the spleen, the diarrhœa and the rose-colored spots. We should remember, however, that in meningitis spots somewhat resembling the latter may also be present. Finally we may avail ourselves of Ehrlich's reaction, that is the diazo-reaction of the urine which, when present, would throw the weight of our decision in favor of typhoid fever Doubt, however, is thrown upon the value of this symptom, as it is found occasionally in other diseases with high temperature.

Occasionally it is difficult to decide whether the patient is suffering from acute meningitis or from delirium tremens. If the meningitis runs its usual course, and especially if it is basal, the diagnosis is not difficult; but if it be mainly or exclusively vertical, so that delirium and restlessness are prominent, delirium tremens may be simulated. We must be on the lookout in such cases for focal symptoms, for if they are present they point at once to meningitis. Rigidity of the neck, intense headache and optic neuritis do the same. Similarly it is at times difficult to distinguish between *delirium grave* (typhomania) and meningitis. The early appearance of somnolence, rigidity of the back of the neck, localizing phenomena, optic neuritis, hyperæsthesia and convulsions point to meningitis.

Occasionally infants who have suffered from exhaustive attacks of cholera infantum present symptoms which closely simulate meningitis, though no affection of the membranes is found on autopsy. These cases have been termed by Marshall Hall, "Hydrocephaloid." They are distinguished from true meningitis by the previous history and by the fact that the fontanelles, instead of being full and prominent, are sunken.

Differential Diagnosis of the Various Forms. It is necessary to differentiate the various forms of meningitis from each other. Epidemic cerebro-spinal meningitis is to be distinguished from the other forms by the presence of spinal symptoms and by the existence of the malady in an epidemic form. However, we now and then meet cases of cerebro-spinal meningitis in which brain symptoms predominate, and also cases of other forms of meningitis in which, while brain symptoms are present, the spinal cord is also involved. Further, cases are met with in which the symptoms of the epidemic form are closely simulated, though no epidemic may exist at the time. We see at once that the differential diagnosis as regards epidemic cerebro-spinal meningitis from the other forms is not always possible. However, in addition to the general facts already stated, we should remember that the course of the disease in the epidemic form is usually very rapid; sometimes, indeed, it is lightning-like, yet in many instances the diagnosis of meningitis alone can be made, and its special character must remain a conjecture.

Purulent meningitis, we should bear in mind, is sometimes the result of ear disease with concomitant necrosis of the petrous bone, less often the result of suppurative catarrh of the sinuses of the frontal bone, and more rarely still, of disease of the orbit. Traumata, such as complicated fractures of the cranial bones are also prominent factors in its production. To these causes must be added septic thrombosis of the sinuses and brain abscess. Further, it may be secondary to septic processes elsewhere, such as puerperal fever. As regards meningitis the result of ear disease, frontal sinus disease, traumata, etc., we have much in the history of the case to guide us in making our diagnosis. However, if such a history and localizing symptoms be wanting, and the case do not accord in symptomatology with cerebro-spinal meningitis, causes or evidences of general septic infection should be sought for; in such cases we may find endocarditis, inflammations of the joints, septic nephritis, abscesses of the lungs, etc.

The features which distinguish serous meningitis have already been indicated. In addition, it is important to remember that it occurs most frequently in early childhood and in youth, though it is occasionally met with among adults. The symptoms frequently resemble those of ordinary hydrocephalus. In adults the error is not infrequently made of mistaking the condition for brain tumor. The presence of focal symptoms, in the latter, will serve in many cases to make the distinction.

Tuberculous meningitis is distinguished by the fact that it is much slower in its course than the other forms and that the temperature is rarely much elevated. When, therefore, we have symptoms pointing to a meningitis which are slow in their development and subacute in course we should be very careful to search for evidences of general tuberculous infection. The lungs, the pleura, the joints, the bones, the lymphatic glands, especially those of the neck, should be examined. Further, we should remember that tuberculous meningitis is that form which occurs most frequently in children. It is apt to have a long prodromal period, during which there is a gradual loss of appetite, emaciation, sleeplessness, general malaise and apathy, and there is also a predominance of symptoms referable to the base of the brain. Persistent vomiting,[1] marked irregularity of the pupils, irregularity of the respiration, more or less marked interference with the ocular nerves, papillitis, transient or persistent facial palsy, monoplegia, hemiplegia, aphasia,[2] are among the symptoms witnessed. Should doubt arise as regards the differentiation between this and the purulent form of meningitis some assistance may be given to the diagnosis by testing the urine for peptones. Peptones will very probably be present in the urine if marked suppuration is taking place.

A not unimportant caution remains to be given regarding tuberculous and even other forms of meningitis when they occur in young girls. In such cases the symptoms are often hysteroid in character, and the mistake has at times been made of regarding them as hysterical, due consideration not being given to the possibility of organic disease. Again, it is important to bear in mind that the course pursued by tuberculous meningitis is in rare instances latent. Autopsies have been made in cases of sudden death which have revealed an entirely unsuspected and symptomless tuberculous meningitis.[3] Finally, tuberculous meningitis, though occurring most frequently in infants and the young[4] may occur much later. Thus Rendu and Bulloche[4] report a case occurring in a woman aged thirty-eight years. Bastian[5] reports a case of tuberculous meningitis in a man aged fifty-two years. Matthes[6] reports a case of miliary tuberculosis involving the meninges in a man aged sixty-seven years, and Archambault, the case of a man sixty-eight years of age.

Rarely tuberculous meningitis involves in addition to the membranes of the brain also those of the cord,[7] and in such case spinal symptoms are present.

Alcoholic meningitis differs markedly in its ordinarily chronic course. There is always present a marked history of alcoholism, chronic headache, occasional slight delirium, mental weakness, and sometimes slight papillitis.

In localized meningitis, such as occurs from blows upon the skull, we have not only the history of the accident but also the symptom of headache, more or less severe and local in character. Very frequently there are also focal symptoms pointing definitely to some cortical centre. A meningitis having its origin in disease of the sinuses or of the bones of the skull also presents more or less definite localizing symptoms. Of these affections, the meningitis arising from middle-ear disease and consequent necrosis of the petrous bone may be taken as an example. In such a case we have the history of an ear trouble of some standing; often there is a chronic purulent discharge from the external auditory meatus. The involvement of the meninges is made evident by intense unilateral basal headache, the pain being so great that it is with difficulty, if at all, relieved. If the case persist, as is most likely, we have not only deafness upon the side affected, but also the symptoms of a peripheral facial palsy. This palsy is shown to occur through pressure on the nerve in its course through the Fallopian canal, by loss of taste upon the anterior two-thirds of the tongue of the side affected, and by unilateral paralysis of the soft palate. Other cranial nerves may become involved, especially the various ocular nerves. Even the crus of a hemisphere may suffer, and in such an instance we may have the symptoms of a crossed hemiplegia. Abscess of the brain is very frequently a direct outcome of this form of purulent meningitis, and it is often associated with thrombosis of the lateral sinus.

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[1] Northrup: Trans. Am. Pediatric Soc., 1890, 1891, ii. 68-69; also Jacobi, ibid (Discussion), Jacobi maintains that vomiting is present only in meningitis of the base.

[2] Among unusual affections of speech noted in tuberculous meningitis must be mentioned echolalia. Perret, Echolalie dans la méningite tuberculeuse, Lyon Médical, 1891, lxvi. 577.

[3] Herzog: Internat. Klin. Rundschau, Wien, 1892, vi. 545.

[4] Rendu et Boulloche: Bull. et Mém. Soc. méd des hôpitaux de Paris, 1891, viii. 458.

[5] Bastian: Trans. Clin. Soc. London, 1891, xxiv. 29.

[6] Matthes: Muench. med. Wochenschr., 1892, 869; Archambault (Dict. encyclop. des sc. méd Paris, 1873).

[7] Mertz: Deutsche med Wochenschr, 1893, xix. 206.

Prognosis. The prognosis in meningitis varies greatly according to the character of the case. In the various forms of diffuse meningitis and in the local suppurative form the prognosis is very grave. Even in severe cases, however, the termination is not necessarily fatal. Indeed, recovery ensues occasionally from even purulent and tuberculous forms. However, when recovery does result, the patient runs the risk of suffering from some permanent injury either to the brain or cranial nerves. We need only be reminded of the well-known case of Laura Bridgman, in which a meningitis secondary to scarlet fever was followed by destruction of both optic and auditory nerves. Again, we may have a condition of chronic internal hydrocephalus as a permanent result.

Non-purulent forms of traumatic meningitis, meningitis following sunstroke, serous meningitis, and alcoholic meningitis do not immediately threaten life. Complete recovery from them, however, is uncertain. Purulent traumatic meningitis and meningitis from extension of neighboring inflammation is very fatal. Syphilitic meningitis presents a prognosis good in proportion to the shortness of the interval between the origin of the condition and the beginning of treatment.

Treatment. The treatment of meningitis must be based upon general principles. In the acute forms rest in bed is, of course, imperative. Various general and special measures must be used to combat the disease, to maintain the strength and to relieve the suffering of the patient. Among the most distressing symptoms is the intense headache, and to control this antipyrin, antifebrin, phenacetin, with or without the bromides, should be freely used, at least in the beginning of the case. Antipyrin and its congeners are not contraindicated unless there be marked depression. They should, however, be used for a short period only. Morphine is theoretically contraindicated, but in a large number of cases it is the only drug that gives relief to the patient. Application of ice to the head or of cold to the general surface of the body, if the temperature be high, is important. We should remember that occasionally ice is not well borne by young children. Buch,[1] for instance, found that cold applications in cases of meningeal tuberculosis in children have a tendency to produce spasm of the glottis. In many cases, blisters to the back of the neck or back of the ears may be applied with advantage. Especially is this the case in children. In adults the application of a large number of wet cups to the back of the neck, or of leeches to the temple constitute an excellent expedient. General principles must, of course, guid us with regard to these measures, particularly with regard to blood-lettin and to the special form of counter-irritation to be adopted. In anæmic c tuberculous subjects the local abstraction of blood is contraindicated.

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[1] Buch, St. Petersburg. Med. Wochenschr., 1891, N. F. viii. 203.

The patient being constipated, it is necessary to bring about active purgation, and for this purpose no drug is better than calomel. Not only does i produce revulsion by acting upon the bowels, but it has also a tendency t favorably influence the inflammation of the membranes. With the latte end in view, mercurial inunctions should also be used. Children tolerat mercurials very well. In adults, however, considerable caution should b exercised.

Special symptoms, such as a persistent vomiting, demand and often baffl our best efforts. Small doses of bismuth, or bismuth with cocaine, the carbonated waters, or iced champagne are among the measures at our disposal After the more violent symptoms subside, supporting measures should b used to their fullest extent. Alcohol must be used guardedly, if at all Digitalis and strophanthus may be used, provided the period of initial slow pulse has been passed. Strychnine, for obvious reasons, is not a suitable tonic, and this is also true of quinine, full doses of which increase meningeal irritation. If the case has passed into a subacute stage, iodides and mercurials may be used sparingly for a long time. Experience inveighs against the use of tuberculin in tuberculous meningitis. Brehm[1] found that the remedy made the disease more *foudroyant.*

Surgical measures[2] also have been resorted to in the treatment of tuberculous meningitis. C. A. Morton tapped the arachnoid space in four cases and noted slight improvement in two of them. Wallace Ord and Waterhouse trephined through the cerebellar fossa in a child of five years. A small quantity of fluid escaped, a drainage-tube was inserted, and the child did well.

The treatment of syphilitic meningitis does not merit special description. It is to be based upon general principles, again bearing in mind, however, to make free use of the mercurials. The chronic forms of meningitis which result from insolation and other causes are much benefited by the actual cautery applied to the back of the neck at frequent intervals in the manner already indicated in the discussion of pachymeningitis (p. 349).

The surgical treatment of traumatic meningitis has already been alluded to (see p. 350), and is considered in detail in Chapter XXXIII.

MENINGEAL HEMORRHAGE.

Hemorrhages of the meninges of the brain may occur in various situations. They may take place first into the meshes of the pia mater, and in such cases they almost of necessity involve the cortex. Secondly, they may occur in the subdural space; and, lastly, they may occur between the external layer of the dura and the cranium, *i. e.*, be supra-dural. They may have their origin in lesions of the arteries of the brain, of the cranium, of the veins of the pia mater, or of the sinuses of the dura. They may occur either as a result of disease of vessel walls or gross intra-cranial disease, but far more frequently they have as their cause injuries of the skull.

Spontaneous Meningeal Hemorrhage. When due to vascular degeneration meningeal hemorrhages arise under conditions similar to those which occasion ordinary cerebral hemorrhage—that is, middle or advanced life, renal and arterial disease. Spontaneous meningeal apoplexy is, however, quite rare. The symptoms presented are, of course, very variable, depending entirely upon the seat and extent of the extravasation. Headache followed by coma, convulsions, or other palsies may be the symptoms produced. Occasionally, as we have seen, when occurring in the arachnoid space, in degenerative states, such as are present in the chronic insane, the hemorrhage may give rise to a condition known as hæmatoma of the dura. (See p. 351.)

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[1] Brehm, Kinder Artz. Berlin, 1891, ii. 1-7.

[2] C. A. Morton, Brit. Med. Journ., 1891, vol. ii. p. 840; also Wallace Ord, and Waterhouse, Brit. Med. Journ., March 10, 1894.

In rare cases a small hemorrhage may suddenly take place on the inner surface of the dura in plethoric persons. The symptoms presented are so violent as to demand the most active interference, and it is therefore important that the condition should be promptly recognized. Occurring in persons of full habit, often just after eating a full meal, or while in the act of defecation or other effort requiring muscular strain, the symptoms are those of an agonizing headache, often localized and coming on with great suddenness. The pain is so great that the patient becomes pale and depressed. Often vomiting is present, or sudden and repeated movements of the bowels occur. Sometimes other signs of meningeal irritation, such as contracted pupils, are seen. Photophobia and excessive sensitiveness to noises may also be present. At times the pain increases paroxysmally, and may radiate down the back of the neck and trunk. Indeed, many of the symptoms actually suggest a meningitis. However, the fact that the attack occurs in a plethoric individual, after eating and after muscular exertion, as well as the course of the affection, will enable us to make the differential diagnosis. Localizing symptoms may be absolutely wanting, as may also somnolence and coma.

Treatment. The treatment of spontaneous meningeal hemorrhage is practically that of ordinary apoplexy (see Chapter XV). However, in those rare instances in which a small sub-dural hemorrhage occurs, and in which the symptoms resemble those of a meningitis, most active treatment should be instituted. Wet cups should be freely applied to the back of the neck, and it may even be necessary to resort to general blood-letting. Morphia sufficient to relieve pain, ice to the head, and mercurial purgation are among the other measures indicated. The headache is only with difficulty relieved, and is apt to persist for days, though with a lessening severity.

TRAUMATIC MENINGEAL HEMORRHAGE. The traumatic form of meningeal hemorrhage is comparatively frequent. In the great majority of the cases there is fracture of the skull as a result of a blow, the fracture being accompanied by the rupture of some vessel. Cases, however, are met with in which no fracture is present, and in which, notwithstanding, meningeal hemorrhage occurs. The fracture may be exceedingly small and difficult to recognize, even when the skull is exposed. It may be limited to one table. In almost half the cases, according to Jacobson's[1] studies, there is not only a fracture of the vault, but also of the base. The middle meningeal artery is more frequently the seat of traumatic hemorrhage than any other vessel. Its main trunk is seldom injured, the rupture usually involving one of the branches. Blows upon the skull of sufficient violence to cause fracture, or meningeal hemorrhage, are at times accompanied by ecchymosis or contusion of the brain substance. Frequently the contusion is not immediately beneath the site of the fracture, but is found at some distance. Thus a blow upon the side of the head may be accompanied by contusion of the tip of the opposite temporal lobe; a blow upon the occiput may be accompanied by ecchymosis of the tips of the frontal lobes. Extensive softening and even abscess have been described as occurring in these situations.

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[1] Jacobson: Guy's Hospital Reports, 1885-86, xxviii. p. 147.

Symptoms. In extensive injuries to the skull the symptoms of hemorrhage are often complicated and obscured by those of concussion. However, in those forms in which the hemorrhage follows a blow at the side of the head, and is due to the rupture of the middle meningeal artery, unconsciousness and coma do not immediately supervene. The hemorrhage in such cases takes place between the dura and the bone—that is, is entirely extra-dural. Time is required for the outpouring blood to accomplish extensive separation of the dura and for compression symptoms to make their appearance. There is, therefore, almost always a distinct interval of consciousness. This interval varies from a few minutes to several hours. It is not uncommon to learn that after receiving the blow the patient was quite himself, and even attended for some time to his ordinary duties, and that gradually he became somnolent, more and more difficult to arouse, and finally comatose.

In some cases the unconsciousness never deepens into absolute coma. In others, again, instead of coming on gradually, it may come on very suddenly. According to Weismann,[1] it may be exceedingly delayed, one instance of eleven days being given. Associated with the significant fact of an interval preceding the loss of consciousness, we may have also certain motor disturbances dependent upon the seat of the hemorrhage. Among these may be hemiplegia or aphasia. Sometimes, too, there is rigidity of one or both legs, or of both legs and arms. Often with this rigidity we have associated muscular twitchings. The hemiplegia may be only temporary, and varies greatly in degree. According to Jacobson,[2] paraplegia may also be present in these cases. Finally, we should remember that motor disturbances may be entirely wanting. There may be no paralysis whatever.

Interesting and important pupillary phenomena are also apt to be present. Not infrequently, as I have myself seen, the pupil upon the injured side is dilated, perhaps widely so, while that of the opposite side is either normal or contracted. This dilated pupil, termed Hutchinson's pupil by Jacobson, appears to be a pressure symptom. At times both pupils are dilated, and at others, again, both are normal, but if unilateral dilatation is present the symptom is of great value.

The pulse is slow, though it may become more frequent later on. The respiration is apt to be slow, labored, and even stertorous. Vomiting, also, may be present. A rise of temperature may take place, and this is sometimes very high. Thus, according to Weismann 108.8° F. has been reported.[3]

Diagnosis. If meningeal hemorrhage occur in the arachnoid space, that is, be sub-dural, such as may arise from injury of a sinus, the onset of symptoms may be so sudden and so complicated by concussion, that attempts at localizing the extravasation may be futile. However, the value of a careful study of the localizing symptoms in every case is shown by the experience of Starr and McBurney,[4] in which a traumatic hemorrhage from a vein in the pia mater gave rise to apoplexy, partial right hemiplegia, and hemianæsthesia. Trephining, with removal of the clot, resulted in recovery. In supra-dural hemorrhage, as we have seen, a number of distinct and valuable signs are present. Among these is to be placed first and foremost the interval of consciousness which precedes the somnolence and coma. Secondly, the occurrence of a hemiplegia on the side opposite to that upon which the blow upon the head has been received, is of the greatest significance. A dilated pupil upon the injured side ranks next in importance. If associated with these symptoms we have a slow pulse, labored breathing, vomiting and rigidity of the limbs, with twitching and possibly a rise of temperature, a supra-dural hemorrhage is indicated. In the majority of cases some evidences of trauma are also present upon the scalp or skull, and if these are marked the diagnosis is still further assured. We must remember, however, that all signs of external injury may be wanting. This was the case in a patient reported by Bremer, in whom the principal symptom was aphasia, and in which trephining was followed by recovery.[1]

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[1] Weismann: Deutsche Zeitung f. Chirurgie, vol. iv. 1-3.

[2] Loc. cit.

[3] Loc. cit.

[4] Starr and McBurney: Brain, 1891-92, xiv. 284.

Prognosis. The prognosis of traumatic meningeal hemorrhage depends largely upon its seat and whether it is surgically accessible. Extensive effusions of blood in the sub-dural space are, for reasons already stated, not only difficult to recognize, but offer less hope of relief by trephining. In the case of supra-dural hemorrhage great good can be accomplished, provided the case be recognized sufficiently early.

Treatment. The treatment of meningeal hemorrhage is, of course, a surgical one purely. As Jacobson correctly says, "We are to trephine and to trephine early."[2] Delay greatly increases the patient's danger. That, however, trephining at even a late day may be successful is shown by the experience of Stokes,[3] who reports a case of secondary operation nine days after the accident, with recovery, although at the time of the operation the patient was comatose. In this case the patient had the associated symptoms of brachial and facial paralysis. (See Chapter XXXIII on Surgery.)

DISEASES OF THE SINUSES OF THE DURA MATER.

As a result of various causes, thrombosis of sinuses may occur, and because of the peculiar relation which the sinuses bear to the venous circulation of the brain as well as to certain portions of the extra-cranial venous circulation, symptoms arise which it is very important to recognize. A thrombus forms in a sinus as a result of one of two conditions: first, as a consequence of an altered state of blood and feeble circulation, conditions present in various adynamic states; for example, in typhoid fever, grave diarrhœa, in the last stages of phthisis, cancer, chlorosis, grave anæmia, etc., and, occasionally, in generally septicæmia. This form of thrombosis is more frequently found in the superior longitudinal sinus than elsewhere, and more often in children than in adults. The conditions present in the superior longitudinal sinus are peculiarly favorable to the formation of a clot. The blood current is extremely slow, the cavity of the sinus is angular in shape, and it is traversed by numerous fine trabeculæ.

The second cause of the formation of a thrombus is the direct extension of inflammation to the sinus from some contiguous structure, or by direct pressure, as from a tumor. It is in this manner that the thrombi arise which are occasionally met with in brain abscess, purulent meningitis, suppurative inflammation of the cranial bones, of the middle ear, of the orbit, or of the nasal cavity. Inflammation of an external vein sometimes gives rise to a thrombus. This every now and then takes place in phlebitis of the face, scalp, or neck.

If a clot form in a sinus it is apt to extend into the veins of the pia mater. As a consequence, the circulation in the veins is arrested, and the portions of the brain supplied by them may undergo destructive softening. If veins communicating with the surface of the skull, face, or neck be involved, varicose local swellings may occur, constituting striking symptoms.

The thrombus generally consists of a laminated clot which may or may not show signs of purulent changes. Sometimes when septic it may give rise to metastatic disease of the lung and to a general pyæmic infection.

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[1] Bremer: American Journal of the Medical Sciences, 1892, ciii. 134.

[3] Stokes: British Medical Journal, 1888, p. 747.

Alport,[1] after collecting 169 cases of all kinds, showed that most cases occur between seventeen and twenty-six years of age, though quite a number occur in the first three years of life. Of all the cases in which the fact was stated, namely 128, 118 followed chronic otorrhœa.

FIG. 78.

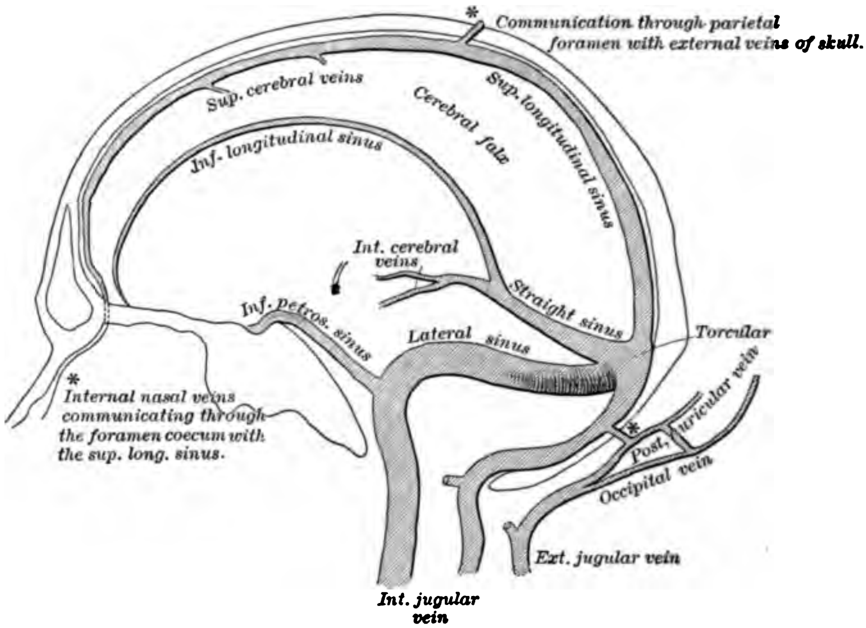

Diagram showing the communications existing between the superior longitudinal and lateral sinuses and the external veins, indicated in the figure by *. (LEUBE.)

General Symptoms. A sinus thrombosis, especially when it involves the longitudinal sinus, may be masked by other conditions, and may only be detected at the autopsy. More frequently it gives rise to decided symptoms, among which may be very severe and often localized headache, vomiting, rigors, high temperature, slow pulse, convulsions, mental confusion, and rigidity of the back of the neck. Occasionally more or less marked palsies, hemiplegic or monoplegic in type, are added. When present they are due to secondary cortical softening. Very often the signs of thrombosis are complicated by those of a meningitis or brain abscess, and it is therefore often difficult to diagnosticate. In fact it can only be recognized when certain circulatory disturbances pointing directly to the blocking up of the sinuses are present. A knowledge of the anatomy of the sinuses and of their venous connections, is, therefore, absolutely necessary. The most important clinical forms met with are thromboses of the lateral, longitudinal, and the cavernous sinuses.

Special Symptoms. THROMBOSIS OF THE LATERAL SINUS. Disease of the middle ear—caries of the petrous bone—is the most common cause of disease of the lateral sinus. Consequently, signs of chronic otitis media are almost invariably present. Intense headache, sometimes preceded by agonizing earache, are the usual accompaniments. Vomiting, optic neuritis, and fever pyæmic in character, may also be present. Even nystagmus may be noted. The involvement of the sinus is especially indicated by venous fulness and œdema back of the ear, in the mastoid and occipital regions.

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[1] Alport. Journal of American Medical Association, 1892, xix. pp. 690, 725, 744.

FIG. 79.

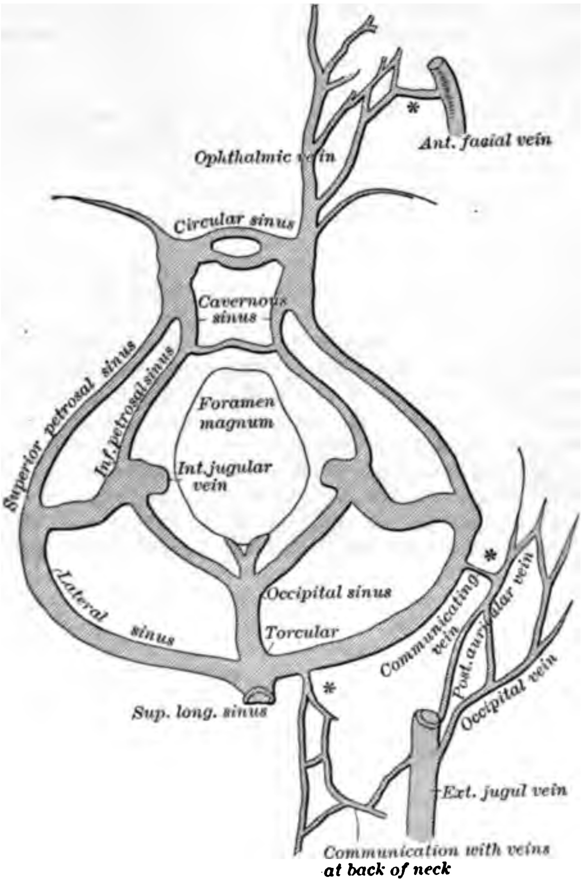

Diagram showing the communications existing between the lateral and cavernous sinuses and the external veins, indicated in the figure by *. (LEUBE.)

This œdema is due to the extension of the thrombus to the small veins which pass through the posterior condyloid and mastoid foramina. These veins connect the lateral sinus with the veins of the scalp back of the ear and over the lower portion of the occiput. In adition there is decided tenderness over the œdematous area, especially, as Bennett[1] has shown, over the mastoid foramen. Another symptom that has been observed is that the external jugular vein on the affected side seems less full than its fellow, the difference being brought out especially by efforts at inspiration (Gerhardt).[1] Inasmu as in lateral thrombosis the internal jugular of the same side receives blood from the cranial cavity the external jugular empties itself more readi into the common trunk. However, in some cases the thrombus extends in the internal jugular for such a distance that it blocks the communicati with the external vein. In such a case, the latter, instead of being compar tively empty (Gerhardt's symptom), is over-distended. When the cl extends for some distance into the internal jugular, the latter is felt as a fir and painful cord. Sometimes the patient holds the head inclined towar the affected side, as in this position the muscles are less apt to cause pressu upon the inflamed vein. Rarely it happens that in cases where the inflam mation is purulent and of such severity as to involve the tissues external t the sinus, the various nerves which accompany the jugular vein in its passag through the jugular foramen may be affected. Symptoms referable to irri tation of the pneumogastric, spinal accessory, and glosso-pharyngeal hav been described, *e. g.*, excessive slowing of the pulse, respiratory disturbanc hoarseness, aphonia, spasm of the sterno-mastoid, and trapezius, difficulty o swallowing, etc.

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[1] Bennett: Lancet, London, 1893, ii. pp. 619, 1001.

THROMBOSIS OF THE CAVERNUS SINUS. Thrombus of the cavernous sinus like that of the lateral sinus, is frequently due to purulent inflammation i its immediate neighborhood. Both sinuses are occasionally involved at th same time. Because of the communication existing between the cavernou sinus and the ophthalmic vein, the special symptoms presented are very striking; thus, there is œdema of the eyelids of the same side, of the conjunctiva, and, later on, of a great part of the side of the face. The latte symptom, however, may not be present, or may be transient in character. In adition there may be more or less marked exophthalmus, and if we examine the fundus of the eye we find that the retinal veins are distended and unusually tortuous, and there may also be œdema of the retina and of the papilla. These appearances are all accentuated if the thrombosis extends to the central retinal vein. There may also be venous pulsation, retinal hemorrhages, etc. If the thrombus be septic in character the symptoms may be those of a phlegmonous inflammation of the connective tissue of the orbit. (Leube.)

Inasmuch as the oculo-motor nerve and ophthalmic branch of the fifth are enclosed in the wall of the sinus, and the pathetic and abducent nerves pass directly through it, various symptoms indicative of interference with these nerves are present. Paralysis of various ocular muscles, ophthalmic neuralgia, and even neuroparalytic ophthalmia may occur. Deep-seated headache may also exist.

THROMBOSIS OF THE SUPERIOR LONGITUDINAL SINUS. Thrombosis of the superior longitudinal sinus is almost always of "marantic" origin, that is, occurs in low, adynamic conditions. It is not an infrequent complication in various affections of childhood, such as chronic exhausting diarrhœa or long continued fevers. In adults it also occasionally occurs under similar conditions, but the symptoms are frequently masked by those of the general disease, and it escapes recognition altogether, being detected only on the post-mortem table. Special symptoms, however, are always present in complete obstruction of the sinus. Inasmuch as the sinus communicates directly with the veins of the nasal cavity, the latter become very much distended, and not infrequently marked epistaxis ensues. There is also marked fulness and even cyanosis in the distribution of the anterior facial vein. Similarly, because the sinus is in communication with the veins of the scalp, (by way of the emissary veins of Santorini, which pass through the parietal foramina) there ensue fulness of the veins and œdema of both temporal regions spreading up to the vertex. This swelling is especially noticeable in children. In the latter, also, because of the obstruction to the outflow from the cerebral veins and the consequent increase of intra-cranial pressure, the fontanelles become distended and prominent. Various symptoms indicative of more or less profound disturbance of the cerebrum also make their appearance; thus, there may be mental dulness, delirium, somnolence, and later on, stupor and coma. Convulsions and palsies may also occur, and these may be more marked on one side of the body than on the other. Vomiting is also present, and headache is quite constant. Inasmuch as the affection is often complicated by a meningitis, there may be, in addition, such symptoms as strabismus, rigidity at the back of the neck, rigidity of the limbs, etc.

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[1] Gerhardt: Deutsche Klinik, 1857, vol. ix, pp. 437, 445.

Diagnosis. Many of the general symptoms presented by thrombosis of the sinuses are also present in meningitis. Among these are headache, vomiting, chills and fever, mental disturbances, such as delirium, somnolence, or stupor, and at times optic neuritis. The diagnosis of sinus thrombosis depends upon the special symptoms produced by sinus obstruction.

Thrombosis of the Lateral Sinus occurs, as we have seen, far more frequently than the other forms. Associated, as it is, with disease of the middle ear, the latter, in all suspected cases, should be carefully examined. The presence or a past history of a purulent discharge is of the greatest significance. Especially is this the case when headache, rigors and rise of temperature follow the sudden cessation of such a discharge. If in addition, tenderness and œdema be present over the mastoid process, lower occipital or upper cervical region, sinus thrombosis is indicated. Diffuse tenderness may be wanting, but a painful point will always be found directly over the mastoid foramen. It is due to a phlebitis involving the emissary vein, which here makes its exit from the sinus. This point exists at the posterior edge of the mastoid process, or better still, it may be found by placing the finger tip firmly upon a spot one inch behind and about half an inch above the middle of the external auditory meatus (Bennett).[1] Sometimes the œdema also is very slight, is circumscribed and limited to this small area. Lack of fulness in the external jugular vein, cord-like hardness and tenderness over the internal jugular are also to be sought for. We should be careful to differentiate between lateral sinus thrombosis and basal meningitis, or brain abscess dependent upon ear disease. In uncomplicated brain abscess the symptoms of sinus obstruction are absent. However, we should remember that in children the symptoms of obstruction are now and then but slightly marked, and in such case the differential diagnosis from meningitis is exceedingly difficult and at times impossible.

The practitioner should be cautioned in every suspicious case of continued fever of irregular or pyæmic type, to examine the ears as possible sources of infection, and it occasionally happens that in this way a sinus thrombosis is detected. "Earache" in a febrile case should always be regarded with suspicion.

The diagnosis of *thrombosis of the cavernous sinus* is based, in addition to the general symptoms mentioned above, upon the symptoms caused by the obstruction of the ophthalmic vein, namely, œdema of the conjunctiva, of the eyelids, of the upper part of one side of the face, exophthalmus, strabismus, neuralgia, fulness and tortuosity of the retinal veins, etc. In suspected cavernous thrombosis, disease of the ear should also be looked for. What has been said relative to this point in connection with thrombosis of the lateral sinus is also applicable here. Disease of the cavernous sinus is much less frequent than that of the lateral sinus.

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[1] Loc. cit.

The diagnosis of *thrombosis of the longitudinal sinus* is also to be based upon general symptoms and on the œdema of the temporal regions caused by obstruction of the veins of Santorini, the occurrence of epistaxis, marked mental symptoms, somnolence, coma, or delirium, convulsions and palsies, and, in young children, the prominence of the fontanelles.

Prognosis. Thrombosis of the sinuses is always a grave affection. When present in the lateral sinus, however, the termination is by no means always fatal. Not infrequently cases are saved by prompt surgical interference.

Treatment. The expectant treatment of sinus thrombosis offers very little hope. Surgical procedures alone promise relief. Operations have many times been attempted, and often with success. Lateral sinus thrombosis, the most common form, is at the same time that form which admits most readily of surgical treatment. In 1880, Zaufal[1] proposed to ligate the internal jugular on the affected side, in order to prevent, if possible, systemic infection, and also to remove the mastoid process and to expose and evacuate the sinus. He first carried out his suggestion in 1884, though unsuccessfully. Two years later, Horsley[2] recommended a somewhat similar procedure. Since then, Lane,[3] Ballance,[4] Salzer,[5] and others, have operated with more or less success. The operation consists of two parts—first, ligation of the internal jugular below the thrombus by two ligatures, and division of the vein between the ligatures; second, trephining, opening of the lateral sinus, and the washing out of the thrombus from above, or removing it by the curette. For details, the reader is referred to the chapter on Surgery. Successful cases have also been reported by Parker,[6] Pickering,[7] Clutton,[8] Harris,[9] and others. Of twenty cases of operation upon the sinuses collected by Koerner,[10] thirteen were successful. Hansberg[11] suggests that, because in the early stages the diagnosis may be uncertain, we should always open the mastoid if, in the course of suppurative ear disease, pyæmic fever develops.

Regarding operations for thrombosis of the cavernous sinus, but one case is upon record, that reported by Bircher.[12] This case was complicated by thrombosis of both the lateral and the inferior petrosal sinuses. The operation involved the removal of almost the entire portion of the petrous bone. Thorough cleansing and disinfection of the wound was followed by recovery and diminution in the ophthalmoplegic symptoms, but with total facial palsy.

Longitudinal sinus thrombosis, being of marantic origin, is not favorable to surgical interference.

CEREBRAL ANÆMIA AND HYPERÆMIA.

As in other instances in which pathological investigation is difficult or impossible, much difference of opinion has existed relative to anæmia and hyperæmia of the brain. Not many years ago, it was quite common to hear the diagnosis of these conditions made almost daily. At the present time, however, as has been pointed out by Gray, such a diagnosis is rarely recorded.

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[1] Zaufal: Prag. med. Wochenschr., 1880, p. 576.
[2] Horsley: St. Thomas's Hosp. Rep., 1886, vol. xviii.
[3] Lane: Brit. Med. Journ., 1889, i. p. 998.
[4] Ballance: Lancet, 1890, i. p. 805.
[5] Salzer: Wien. klin. Wochenschr., 1890, p. 651.
[6] Parker: Liverpool Medico-Chirurg. Journ., 1892, xii. p. 86.
[7] Pickering: Bristol Medico-Chirurg. Journ., 1891, ix. p. 155.
[8] Clutton: Brit. Med. Journ., 1892, i. p. 807.
[9] Harris: Lancet, 1893, ii. p. 93.
[10] Koerner: Die Otitischen Erkrankungen des Hirns, der Hirnhaeute und der Blutleiter, Frankfurt a. M., 1894, p. 72.
[11] Hansberg: Ann. d. mal. de l'oreille, etc., 1892, xviii. p. 614.
[12] Bircher: Centralbl. f. Chirurg., 1893, p. 483.

This is largely owing to our better understanding of other intra-cranial affections, and to the more frequent recognition of the various nervous phenomena associated with neurasthenia. It is undoubtedly true that anæmia and hyperæmia of the brain as primary affections are excessively rare. However, they are not infrequently met with secondary to, or associated with, some other pathological condition. That cerebral anæmia actually exists, is proven by such simple instances as the cerebral symptoms associated with sudden loss of blood, and that cerebral hyperæmia also exists, at least in the passive form, is proven by the symptoms present in cases of general venous obstruction.

Symptomatology. The symptoms presented by cases of undoubted cerebral *anæmia*, such as are caused by an excessive hemorrhage, consist of ringing in the ears, dimness of vision, attacks of "blackness before the eyes," dilated pupils, vertigo, nausea or vomiting, headache, mental confusion and delirium, insomnia and attacks of fainting. These symptoms are all less marked in the recumbent posture, and may even be relieved when the head is on a lower level than the trunk and limbs. The symptoms presented by an ordinary attack of fainting due to fright, or to sudden depression of the heart's action through any cause whatever, depend upon a relative anæmia of the brain, and resemble those just recounted. Similar though milder symptoms, are occasionally met with in general anæmia, chlorosis, leukæmia, the various cachexias and inanition, and at times associated with neurasthenic conditions. (See Chapter II.)

The symptoms of cerebral *hyperæmia* theoretically resolve themselves into two groups: first, those attendant upon active or arterial congestion; and, secondly, those due to passive or venous congestion. The first group are represented by the symptoms so often prodromal to cerebral hemorrhage, *i. e.*, a sense of fulness and heat of head, headache, throbbing sensations in the head, cerebral excitement, tinnitus, vertigo, and insomnia. From the nature of the case, the symptoms are such as are often present in other affections, and the condition can never be diagnosticated with absolute certainty. It is not improbable that such symptoms as photophobia, auditory hyperæsthesia, confusion of thought, and delirium, which are present in the initial stages of simple febrile affections and in the prodromal periods of meningitis, are due to an active cerebral hyperæmia. Whether the affection exists in an independent and chronic form is exceedingly problematic. The insomnia associated with overwork is probably not the insomnia of hyperæmia, but the insomnia of exhaustion (see chapter on neurasthenia, page 66.) No changes are found in the retinal circulation in the so-called cases of chronic cerebral hyperæmia, while the mere flushing of the face cannot be looked upon as bearing any relation to the circulation within the cranium.

The second group of symptoms, those dependent upon venous fulness, are best illustrated by cases of disease of the heart in which excessive venous obstruction is present. Here we often have a dull headache, some tendency to somnolence, and general weakness, symptoms which in their turn are too ill-defined to enable us to establish an absolute diagnosis. It is probable that to some extent they are due to œdema of the membranes.

In some cases the symptoms of cerebral hyperæmia follow the sudden arrest of an habitual discharge of blood, such as accompanies hemorrhoids in plethoric people. This remark applies especially to women at the menopause. An illustrative case occurring in the experience of the writer was that of a middle-aged woman still at the menopause, who had been operated upon for profusely bleeding hemorrhoids. The operation had been entirely successful, yet some three weeks afterward, at what seemed to be a menstrual epoch, the patient died with all the symptoms of a venous hyperæmia of the brain.

What has been said concerning active cerebral hyperæmia in regard to the existence of the affection in an independent and chronic form applies equally here. It is with cerebral hyperæmia and anæmia, as associated with other states, that we have especially to deal. A reference to the chapter on neurasthenia in this volume will show the reader that in very many neurasthenics numerous obscure cephalic sensations are present, such as throbbing, tension of the head, sense of fulness, etc., which suggest the presence of an increased amount of blood in the cranial cavity. It is not improbable that the exacerbation of the head symptoms in female neurasthenics at the time immediately preceding the menstruation, is to be ascribed to such a cause. While we recognize that hyperæmia of the brain as an independent condition is rarely if ever existent, it is more than probable that it at times complicates other functional troubles, notably neurasthenia.

Treatment. The treatment of a cerebral anæmia associated with loss of blood, chlorosis, leukæmia, general anæmia, the various cachexias or neurasthenia is, of course, the treatment of these various states. Cerebral hyperæmia occurring mainly in connection with other diseases, treatment is to be directed to the latter. Thus a case of heart disease, a case of suppressed menstruation, a case of threatening apoplexy, demands each its appropriate management. The symptoms of heat and fulness, ringing in the ears, and vertigo occurring in a plethoric person, should be met by active purgation, general or local blood-letting, and the administration of bromides.

In neurasthenics in whom we have reason to believe that there is a complicating hyperæmia, we should associate with our treatment the bromides, and especially ergot. In neurasthenic headaches in which this complication is believed to exist, the writer has found ergot quite useful. It is unnecessary to say that this drug is, not applicable in cases of suppressed menstruation, or that it is not to be used at the eve of this epoch. Among the other measures are hot foot or sitz-baths taken at bed-time.

CEREBRAL INFLAMMATION.

Inflammation of the brain appears to be present, as in other viscera, in two forms, a parenchymatous and an interstitial form. Our knowledge of the parenchymatous inflammations is not by any means satisfactory. It is exceedingly probable that they occur in at least three clinical varieties: First, *polioencephalitis corticalis*, an inflammation of the cortex of the brain, the lesion which Strümpel believes to be the essential factor in the cerebral palsies of childhood; secondly, *polioencephalitis superior*, by which is meant an inflammation of the gray matter about the aqueduct of Sylvius, *i. e.*, the nuclei of the various cranial nerves supplying the extrinsic and intrinsic muscles of the eyeball; thirdly, *polioencephalitis inferior*, in which various motor nuclei of the medulla are involved. Polioencephalitis superior and inferior are analogous, it will be seen, to poliomyelitis (see Chapter XX). Polioencephalitis superior, which gives rise to *ophthalmoplegia*, is described in Chapter XXVI., while polioencephalitis inferior, *bulbar palsy*, is described in Chapter XXI. Polioencephalitis corticalis, at times the lesion of *cerebral palsy of childhood*, is considered in Chapter XVII.

Interstitial inflammation of the brain may be either acute or chronic and may result in more or less rapid destruction of tissue with or without the formation of pus, or in slowly progressive changes, such as sclerosis. Acute interstitial inflammation of the brain is very rare, but it appears to result every now and then from severe trauma of the head, and at times it arises in the course of one of the infectious diseases, such as erysipelas, influenza, typhoid fever, diphtheria, variola, typhus fever, etc. It is probable also that a less acute form is occasionally due to syphilis. It may be widely diffused or there may be numerous foci of inflammation scattered through the encephalon. At times a localized interstitial inflammation is the result of direct trauma of the brain as by a stab-wound, or from the irritation of depressed or diseased bone or from the presence of a foreign body, or a neoplasm. The symptoms of acute interstitial inflammation are vague, ill defined, and not characteristic. Among them are headache dull in character, vomiting, delirium, coma, convulsions, and hemiplegia and other palsies. The optic papillæ appear, in the cases thus far reported, to have presented no peculiarities. Various conditions of the pupils have been noted. As a rule, mydriasis has been present, though in a case reported by Schmidt[1] the pupils were distinctly contracted. Their reaction is uncertain. Death is preceded by a period of coma.

Autopsies reveal the brain tissue as a whole to be somewhat reddened. At times this reddening is present only in foci here and there. Upon microscopic examination, and in some cases apparent to the naked eye, are found numerous hemorrhages. These may be capillary or punctiform or somewhat diffuse. In other words red softening may be noted in the white matter, the basal ganglia and less often the cortex. At times the hemorrhages are so marked that the condition has been termed hemorrhagic encephalitis.

The fact that the symptoms are vague and common to other intra-cranial affections renders an absolute diagnosis impossible. However, the history of a blow on the head, such as is present in profound concussion of the brain, the absence of optic neuritis and the generalized character of the symptoms should suggest to us a diffuse encephalitis. Similarly if these symptoms occur in a case of infectious fever a diffuse encephalitis may again be suspected. From the uncertainty of the diagnosis and the nature of the affection treatment can only be tentative and general. The principles laid down in discussing the treatment of meningitis apply equally here.

The various localized interstitial inflammations of the brain occurring in the neighborhood of tumors, diseased bone, hemorrhages, etc., may complicate the symptoms resulting from the original lesion. They cannot be separately considered. Occasionally, however, local inflammation results in the brain immediately beneath the site of a blow upon the head or at some distant point, and is often of such an intensity that it results in the formation of pus. At other times, septic material introduced from without may give rise to purulent inflammation and to consequent formation of an abscess. (See page 374). Among very rare forms of inflammation of the brain must be mentioned the case of anthrax of the brain reported by Merkel.[2] On admission to the hospital the patient was exceedingly pale and anæmic, totally unconscious, and presented epileptiform attacks. An ophthalmoscopic examination revealed the margins of the disk on the right side to be obscured; nothing else abnormal. Death occurred shortly afterward. The autopsy revealed, among other things, an injected pia and cortex with numerous small, red specks, apparently hemorrhages, and small hemorrhages in the basal ganglia. Microscopic examination showed the vessels to be filled with anthrax bacilli. In some situations the bacteria had apparently penetrated the vessel walls and were in the perivascular spaces. No bacilli were found in the brain substance proper.

Chronic interstitial inflammation of the brain constitutes various forms of sclerosis. Insular or disseminated sclerosis (see Chapter XXII) is an affection which appears to have its origin in multiple foci of inflammation.

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[1] Schmidt: Deutsche Med. Wochenschr. 1892, xviii. 703.

[2] Merkel: Muenchener Med. Wochenschrift, 1892, No. 47, 840.

Widely diffused sclerosis, such as leads to atrophy, hypertrophy or gross deformity of the brain as a whole or of its lobes, is probably most often degenerative in character, though now and then it likewise appears to have its origin in an inflammatory process. It is most often seen in the brains of idiots and other cases of arrested mental development. These are considered at length in Chapter XII.

BRAIN ABSCESS.

Etiology. Abscesses of the brain are the direct result of septic infection. One of the most common causes is the extension of some suppurative process from contiguous structures, as from caries of the temporal bone, from otitis media, from suppurative processes in the naso-pharynx or in the nasal cavity, from suppuration in the sinuses of the frontal bone, or in the orbits, and from erysipelas and cellulitis of the face and head. Infection may also be carried from a distance. Empyema, lung abscess, putrid bronchitis, and septic endocarditis are all sources of brain abscess. General septicæmia, pyæmia and influenza are among the less common causes. Frequently a trauma attended by a wound or other injury of the skull, such as a complicated fracture of the cranium with or without injury to the brain, results either in a purulent meningitis or brain abscess, or perhaps both. Occasionally abscess occurs in cases in which no septic infection can be traced. Thus it sometimes happense that an abscess develops after blows to the head when no lesion of either table of the cranium or of the dura has been produced. In such instances the abscess may form immediately beneath the site of the blow or in a distant part of the brain; thus, a blow upon the vertex has been followed by an abscess in the temporal lobe, a blow upon the occiput by an abscess in the frontal lobe. Sometimes a blow upon the head may give rise to an abscess at the seat of the blow and a second one at a distant point.[1]

Judging from the statistics of Pitt,[2] almost one-third of the cases of brain abscess are due to disease of the middle ear or of the temporal bone, less than one-sixth to disease of other cranial bones, about one-sixth to trauma, about one-sixth to pyæmia, and the remaining portion to diseases of the lungs and other causes.

In a statistical study of 13,000 miscellaneous cases of ear trouble Jansen[3] found eight complicated with brain abscesses. Of these only seven can be properly included. These seven occurred in something over 5000 cases of suppuration in the middle ear. One occurred in 2650 acute cases and six in 2500 chronic cases. It is seen, therefore, that chronic suppurative otitis is more dangerous than the acute form. That long continuance of suppurating diseases of the middle ear without complications is no guarantee of immunity, is shown by the case of Ransom[4] in which a discharge from the left ear, present for thirty years and then suddenly ceasing, was followed by brain abscess.

Symptoms. *General symptoms.* The symptoms of brain abscess are of two kinds: First, those which are common to cases of brain abscess as a whole; and, secondly, those which depend upon the location and mode of origin. Among those of the first group are headache, vomiting, vertigo and disturbances of the intelligence, *i. e.*, apathy, somnolence, stupor and delirium. To these symptoms we must add rigors, slight fever, normal or even abnormal temperature, slow pulse and constipation. Optic neuritis, as we will see, is also present at times.

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[1] Norbury: Medical News, Philadelphia, 1892, lx. p. 549.
[2] Pitt: Goulstonian Lectures, Brit. Med. Journ., 1890, i. pp. 643, 771, 827.
[3] Jansen: Berliner klin. Wochenschr., 1891, xxviii. p. 1160.
[4] Ransom: Brit. Med. Journ., 1892, i. p. 863.

Headache is one of the most constant symptoms, and is often so severe as to cause intense suffering. It is usually continuous, though occasionally intermittent, and recurring at irregular intervals. It may be diffuse, or localized in certain areas; the latter is often the case when the meninges in the neighborhood of an abscess are inflamed. It is an error, however, to suppose that the *seat* of headache corresponds to the location of an abscess. Frequently the two are widely separated; for instance, frontal headache may be observed in cerebellar abscess. Vomiting possesses here the same significance as it does in brain tumor and in meningitis. It is present more frequently in abscesses of the cerebellum. Disturbances of the intelligence vary greatly both in degree and kind. In the typical case the patient answers slowly, hesitates, and frequently the question must be repeated a number of times before he appears to thoroughly comprehend it. Often he uses the wrong word or gives the wrong name to objects, though true aphasic symptoms occur rather infrequently, and then of course, have a special significance. It is also difficult to secure his attention for any length of time. He is dull, and sleepy. Delirium is among the less frequent symptoms. Rigors occur in the beginning, and are especially observed in cases secondary to suppuration in one of the cavities about the head; for example, those depending on purulent otitis media or empyema of the frontal sinuses. The temperature also runs a somewhat significant course. In a large number of cases there is a slight rise of temperature in the early period, and it is not uncommon to meet with a temperature of 100° to 101°, or even 102°. However, as the case progresses the temperature falls, and is apt to be subnormal, falling as low as 97° or 96°. It is probable that subnormal temperature occurs, especially in simple and uncomplicated cases. A co-existing meningitis or sinus thrombosis is often met with, and it is probably for this reason that more or less rise of temperature is noted throughout in many cases. Thus in a series of twenty-three miscellaneous cases collected by the writer, sixteen presented temperatures varying from 100° to 103° and higher. In three cases the temperature was normal. In four it was subnormal. The importance of these facts in diagnosis will be again alluded to. The pulse is generally markedly slow, resembling in this respect the pulse observed in meningitis and in some cases of brain tumor. Its rate varies from 60 to 50, or 40 beats per minute, but in lethal cases rapidly increases toward the end. At times convulsions occur, but it is probable that they are dependent upon the location of the abscess. Optic neuritis is much less frequent than in brain tumor and in meningitis, and even when present is rarely severe. It is somewhat more frequent in abscesses of the frontal and spheno-temporal lobes and cerebellum than in abscesses elsewhere. Great physical prostration and rapid emaciation often accompany the other symptoms. Tâche cérébrale is noted in some cases just as in meningitis. In others, again, tenderness or pain upon percussion directly over the spot where an abscess exists is sometimes present. Too much reliance should not, however, be placed upon this symptom.

Special Symptoms. The second group of symptoms, namely, those which depend upon the location and mode of origin of the abscess, necessitate the consideration of the various kinds of abscesses separately. The latter may be roughly grouped: *first*, into those due to trauma; *secondly*, those occurring in cases of general pyæmia, purulent lung affections, ulcerative endocarditis, and various infectious diseases; and *thirdly*, those having their origin in infection from various suppurating cavities about the head, such as the middle ear, the frontal sinuses, the orbits, etc.

Traumatic abscesses consist, first, of those with actual wound of the skull, brain membranes, and perhaps brain tissue itself; and, secondly, those in which no such wound can be detected. In the first, rigors such as are common to purulent infection elsewhere, fever, localized and general headache, together with focal symptoms dependent upon the part of the brain injured, are apt to be present. Among the latter are localized palsies or convulsions affecting the opposite side of the body. These abscesses are prone to be complicated by purulent meningitis, and it is probable that the delirium, convulsions and coma often present in these cases, are due to this cause. Optic neuritis may or may not exisist. As a matter of experience, these abscesses are found more frequently in the parietal and frontal regions. In the second variety of abscess due to trauma, that is, those in which no lesions of the cranium or of the membranes exists, and in which the abscess appears to be due to an acute suppurative encephalitis, the symptoms may be very vague and ill-defined. Thus in cases in which frontal abscess results from a blow upon the occiput, no signs may be present save somnolence, stupor and other symptoms referable to simple destruction of the frontal lobes. If such an abscess occur in the tips of the temporal lobes, as is every now and then the case, the symptoms may be so poorly defined that the abscess may run its course without detection.

Abscesses due to pyæmia, suppurative lung troubles and infectious processes are less apt to present the general symptoms of brain abscess in a marked degree. Usually the patient is already so ill that headache, vomiting and mental disturbances lose much of their significance. These abscesses may occur in almost any situation, though they are more frequently met with in the occipital lobes. Not uncommonly they are found in the distribution of the middle cerebral artery, and, indeed, more frequently upon the left side than upon the right[1]. The clinical picture, as in tumor of the brain, depends largely upon the focal symptoms produced. Thus in abscesses of the occipital lobe various disturbances of vision, especially hemianopsia, are usually present; monocular diplopia was observed in one case.[2] In abscesses in or beneath the motor area, cortical epilepsy is rarely absent. For the same reason localized palsies may exist upon the opposite side of the body. Abscesses due to general infectious processes are most frequently multiple; this fact may also influence the symptomatology.

The third group of abscesses, those having their origin in otitis media and disease of the frontal sinuses, orbits, etc., are next to be considered. Most important are those due to diseases of the ear, the so-called otitic abscesses. A history of chronic otorrhœa is usually present. The discharge which has continued without special symptoms for months, and perhaps for years, suddenly ceases. Often this cessation is attributed to cold or to a blow upon the head. Pain in the ear and side of the head, sometimes of frightful severity, now sets in. At the same time, the patient may suffer from a chill. This is soon followed by fever, generally moderate in degree, and by slightly increased frequency of the pulse. Vomiting also is present, and bears no relation whatever to the presence or absence of food in the stomach. The pain in the head is not characteristic, and does not differ from that which may be met with in ordinary acute otitis media, but its occurrence in a chronic case is of the utmost significance. After a variable period, twenty-four, forty-eight hours or more, the pain diminishes, and in the course of a few days the patient passes into the condition of indifference and apathy already described in considering the symptoms in general.

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[1] Martins: Beiträge z. Lehre vom Hirnabscess, Veroeffentlichungen ueber Krankengeschichten und Leichenbefunde aus den Garnisonlazaretten, vii, Berlin.

[2] Abercrombie: Trans. of the Ophthalmological section of the American Medical Assoc., 1891, p. 217.

About this time, also, such febrile rise as may have been present at first disappears, and the temperature becomes normal or subnormal, and the pulse markedly slow. Otitic abscess may be situated either in the spheno-temporal lobe, the cerebellum, or, less often, in the pons or cerebellar peduncle. The detailed symptoms, therefore, vary according to the location. If the abscess be in the temporal lobe and be sufficiently extensive it may, by pressure upon the basal ganglia and capsules, give rise to paresis of the arm and leg of the opposite side, and other symptoms, such as spasms or convulsions. However, the capsule itself is rarely directly involved. Hemianæsthesia and other sensory disturbances have not been reported. If the abscess presses forward and outward it may act upon the various centres of the motor area, more especially upon the centres nearest to it, such as the facial centre. Paresis or spasms of the face of the opposite side are occasionally observed. If the abscess be upon the left side, motor aphasia may result either directly, by extension of the abscess, or indirectly, by pressure on the third frontal convolution. Most frequently this motor aphasia is not complete. Word-deafness and psychic blindness, due to disturbance of the first temporal convolution and angular gyrus have also been recorded. (Macewen.[1]) In a spheno-temporal abscess various cranial nerves are also apt to be involved, especially the oculo-motor. Complete oculo-motor palsy is not infrequently observed; ptosis, dilated pupil and external strabismus are then present on the side of the abscess. The abducent nerve also occasionally suffers, and in rare instances the trigeminal may be involved. The pathetic nerve, for some reason, escapes.

Optic neuritis, present in some cases and absent in others, will not enable us to distinguish between a cerebellar and spheno-temporal abscess, nor will it aid us in determining the side upon which the abscess is situated. It is, when present, sometimes more marked upon the side opposite to that of the abscess.

If abscess be present in the cerebellum, special symptoms may be altogether wanting. However, vomiting is apt to be more marked, and rigidity of the muscles of the back of the neck is frequently present. The headache is usually occipital, but may be frontal. Symptoms of interference with the vermiform process, titubation, and ataxia are rarely, if ever, noted, due, doubtless, to the fact that the abscess is almost always limited to the lateral lobe.[2] Sometimes, however, the speech is syllabic and jerky, such as is seen in cerebellar tumor. Macewen has noted especially rigidity of the masseter muscles. Optic neuritis may be present, but is of no more significance than in abscess of the temporal lobe. Due, doubtless, to a remote effect of pressure, dilated pupils and general oculo-motor paresis is sometimes observed. It is not limited to the side of the abscess. Hemiparesis, with rigidity of the muscles of the same or opposite side, is occasionally noted. Mastoid tenderness is not usually present, unless there be a complicating thrombosis of the lateral sinus.

If the abscess be in the pons, special symptoms may be wanting unless the abscess be large. In the latter instance, crossed hemiplegia, double hemiplegia, or other lateral, bilateral, or shifting palsies may be present. Oculomotor symptoms may also be observed.

Abscesses of the frontal lobe usually present a significant history. Either they are caused by trauma, or there is more or less marked disease of the frontal sinus. The symptoms consist of somnolence, frontal headache, and stupor, preceded, perhaps, by an initial chill. The temperature pursues a course similar to that in otitic abscesses. If the abscess be large, it may pass sufficiently far back to interfere with the motor area, and thus give rise to convulsions and hemiplegia. If it exist upon the left side, motor aphasia may be one of the symptoms presented, with or without right hemiplegia. According to Paget, there is also voracious appetite and extreme thirst.[1]

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[1] Macewen: Pyogenic Infective Diseases of the Brain and Spinal Cord, New York, 1893, case xxxvii.

[2] Koerner: Loc cit., pp. 105 and 106.

THE COURSE OF BRAIN ABSCESS. The course of brain abscess varies greatly in different cases. An abscess may terminate within a few weeks, or may extend over many months, or even years. In some cases the abscess, even after being encapsulated, may become permanently quiescent. Death ensues in inoperable or neglected cases either from profound exhaustion, which is, perhaps, the most common termination, or from the rupture of the abscess upon the surface of the brain or into the ventricles, or by its encroachment upon important centres, such as exist in the medulla. In those cases in which a complicating sinus thrombosis sets in death may result from general systemic infection. In studying cases of suspected brain abscess, we should always bear in mind that departures from the average symptomatology are not uncommon. This is due not only to such frequent complications as meningitis and sinus thrombosis, but also to the fact that the symptoms are often but slightly pronounced, or that they are at times altogether wanting. Cases in which an abscess has pursued an entirely symptomless course from beginning to end are recorded.

Pathology. Brain abscesses are micro-organismal in origin. This is true of them all.[2] The pathogenic organisms most frequently found are the streptococcus pyogenes and the staphylococcus pyogenes aureus, though there may be an admixture of other forms.[3] In a few cases the oidium albicans has been noted. Among rare causes of brain abscess we should mention actinomycosis, a few cases of which have been reported. Tuberculous disease of the middle ear or of the cranial bones is also among the less frequent causes, the membranes being more likely to suffer in such cases than the brain tissue. Primary abscess due to the direct localization of organisms of infectious fevers is rare. These lead more commonly to a purulent meningitis or meningo-encephalitis. Isolated brain abscesses may, however, be caused by peculiar localization of the poison of cerebro-spinal meningitis or erysipelas. The pus of cerebral abscess is greenish or greenish-yellow in color, and fetid.

An abscess having once made its appearance, it steadily increases in size, and gradually encroaches upon the surrounding brain tissue. In a large number of cases this surrounding brain tissue forms a defensive wall about the abscess in the shape of a capsule or pyogenic membrane. This capsule is generally quite soft and fragile; rarely it is firm. The capsule does not bring about a cessation in the formation of pus, and the abscess may finally burst and infiltrate the surrounding tissue, or discharge into the meshes of the pia arachnoid, the surface of the brain, or into the lateral ventricles. At times, if the rupture extend into the white matter only, a second capsule may form, and at the autopsy one encapsulated abscess may be found inside of another. On the other hand, an abscess may, by the formation of a capsule, remain quiescent for a long period of time, or may even become permanently encysted. Abscesses are much more frequently met with in the cerebrum than in the cerebellum, and are quite rare in the pons and medulla. Further, they occur more frequently in the right hemisphere than in the left, though metastatic abscesses appear to form an exception to this rule. When due to pyæmia or to infection from distant organs, such as the lungs, they are generally multiple. When secondary to disease of the ear, of the frontal sinuses, of the orbits, naso-pharynx, etc., they are generally single. This is also the case when they are due to trauma.

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[1] Paget: Trans. Clinic. Society, 1891, xxiv. p. 192.

[2] Martins, loc. cit.; also Macewen, Proceedings of Eleventh International Medical Congress, Medical News, April 28, 1894, p. 464.

[3] Macewen, loc. cit.

Otitic abscesses are found in the spheno-temporal lobe about twice as frequently as in the cerebellum. This is especially true in children. In adults the disproportion is not so great.

The more frequent complications of otitic abscess are purulent lepto-meningitis and sinus thrombosis. Judging from the statistics of Hessler,[1] in about one-third of the cases, one or the other of these complications exists. Thus, of 106 cerebral abscesses, 26 were complicated by a meningitis and 13 by a sinus phlebitis—that is, in spheno-temporal abscess meningitis as a complication occurs about twice as often as sinus thrombosis. Of 59 cerebellar abscesses, only 6 were complicated by meningitis and 10 by disease of the sinus. In spheno-temporal abscess, meningitis occurs in one-fourth the number of cases, in cerebellar abscesses in one-ninth. In the former, again, sinus thrombosis occurs in but one-eighth, while in cerebellar abscess it occurs in one-sixth.

It should also be borne in mind that external purulent pachymeningitis —*i. e.*, extra-dural abscess—in rare cases complicates otitic abscess.

Diagnosis. In the majority of cases of brain abscess the symptoms are such as suggest organic intra-cranial disease. The headache, the vomiting, and the mental condition are significant, especially when taken in connection with the history of the case. The history of a trauma, of the sudden suppression of a chronic discharge from the ear, or, it may be, of general septic infection, is of the utmost importance. Many of the symptoms are common to both tumor and meningitis; but a history of rigors, irregular fever, and especially of subnormal temperature, points to abscess. We should bear in mind that optic neuritis is either not present at all, or, if present, is rarely as marked as in tumor. Differentiation from meningitis depends upon the fact that the symptoms are rarely as generalized as in diffuse inflammation of the membranes of the base or vertex. Differentiation from a local meningitis is, however, not always possible, inasmuch as such a meningitis is often present as a complication.

In addition to the history, the symptoms of most value in enabling us to make a differential diagnosis are those arising from the location of the abscess. As a rule, they are not difficult of recognition. Thus, in abscess of the temporal lobe we have not only such general symptoms as headache, vomiting, mental disturbance, and, perhaps, subnormal temperature, but also, if the abscess be large, various local phenomena produced by its destructive influence and pressure upon neighboring structures, *e. g.*, oculo-motor and pupillary changes, weakness and convulsions of the opposite side of the body, perhaps various forms of aphasia, and, it may be, optic neuritis.

In cerebellar abscess we may have as focal symptoms excessive vomiting, rigidity of the back of the neck, "cerebellar speech," and, perhaps, rigidity of the masseters, as pointed out in the section on Symptomatology. On the other hand, frontal abscess is characterized by such symptoms as stupor, coma, and, perhaps, voracious appetite, convulsions, and aphasia. The characteristics of pyemic and other abscesses have also been pointed out in detail in considering the symptoms. They may be situated in the occipital lobes, giving rise to certain visual symptoms, or in the motor areas and tracts, giving rise to focal convulsions and palsies.

In order to assist in the diagnosis of a cranial abscess, Macewen has studied the differential cranial percussion note. "The percussion note is obtained by the cranial walls vibrating when struck, the note being modified by the consistency and volume of the contents and their relative position to the bone."[1] The test is applicable especially to children. The percussion note in a healthy child is dull, and nearly equally distributed over the vault of the skull. Any increase of density or of tension of the cranial contents beneath the point percussed makes the note clearer.

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[1] Quoted by Koerner, loc. cit., p. 110.

The possibility of the existence of a complicating sinus thrombosis should always be borne in mind, especially in cerebellar abscess, and the characteristic symptoms should be sought for. (See p. 366.)

Prognosis The prognosis of brain abscess is always serious. Unless surgical interference is possible, a fatal termination sooner or later supervenes in the vast majority of cases. Brain surgery, however, has made such enormous strides that when the diagnosis is made sufficiently early, and the abscess is surgically accessible, the condition constitutes, in the words of Macewen, "one of the most hopeful of all cerebral affections." Many instances are on record in which trephining has been successfully performed, the abscess evacuated and drained, the operation being followed by excellent recovery.

Treatment. The treatment of brain abscess is a surgical one only. Everything depends upon the promptness and accuracy of the diagnosis. As soon as an abscess is diagnosed, or, in fact, even suspected, provided localizing symptoms present themselves, exploratory trephining should be undertaken. For the detailed procedure in these cases, as well as the after-treatment, the reader is referred to the section on Surgery.

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[1] Macewen, loc. cit., p. 146.

CHAPTER XIV.

THE ANATOMY OF THE CEREBRAL CORTEX AND THE LOCALIZATION OF ITS FUNCTIONS.

By CHARLES K. MILLS, M.D.

THE surface of the cerebrum is a convoluted arrangement of gray matter known as the *cortex*, the depth of which varies considerably with age, sex, and in different regions of the same hemisphere, and also in different individuals, in accordance with development or disease. Its variations are usually given as between two and three millimetres, but exceptionally it may be as thin as one-and-one-fifth millimetres, or as thick as four. It varies much less among women than among men at different periods. The maximum depth is at the crest of the convolutions, the minimum near the occipital pole. According to Obersteiner,[1] its maximum depth regionally is attained in the paracentral lobe, its minimum near the occipital pole. Conti[2] found that from the postcentral gyrus caudad to the occipital lobe the thickness of the cortex diminished, and that it was at its minimum in the region of Meynert's calcarine type of eight layers, the diminution being so rapid as to be perceptible in sections one centimetre apart. The thickness of the cortex of the parietal lobe is greater on the mesal than on the lateral surface.

Donaldson[3] compared the cortex of the brain of the blind deaf-mute, Laura Dewey Bridgman, with nine normal brains, and concluded that no figures can be given for the average thickness of the fresh normal cortex. Other conclusions by him were that persons with an acquired defect of the central nervous system have a cortex thinner than normal; that females have a slightly thinner cortex than males; that normally the right hemisphere has a cortex slightly less in thickness than the left, the maximum difference being seven per cent. Laura Bridgman's cortex was abnormally thin, having but eighty-nine per cent. of the thickness of the controls, and containing an abnormally large number of small nerve cells. His conclusions with reference to special areas of the cortex will be referred to later.

The amount of gray matter in the surface of the brain has interested a number of investigators, and it may be of practical value, among other things, to determine, as was done by Donaldson, whether portions of the cortex suspected to be defective, and which belong to one hemisphere, will prove to have a less area when the two hemispheres are compared with one another. Donaldson, besides the report of his own important investigations, has given a summary of the work done by others.

The older attempts to estimate the relative extent of different regions of the cerebral cortex were mostly made with a view of determining the relations of certain areas of the brain to the degrees of intelligence. R. and H Wagner,[1] for example, endeavored to make accurate comparisons in this way between the brains of individuals of acknowledged mental ability and those of persons of ordinary or inferior capacity. In the following table are given comparative measurements of the extent of the surface of the convolutions of two men of acknowledged eminence and ability, and of two others of ordinary capacity:

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[1] H. Obersteiner: The Anatomy of the Central Nervous Organs. Translated by Alex. Hill, Philadelphia, 1890, p. 350.

[2] Alfredo Conti: Sur l'Épaisseur de l'Écorce du Cerveau Humain. Cited by Jules Soury in Les Fonctions du Cerveau, Paris, 1892, p. 334.

[3] Henry H. Donaldson: Anatomical Observations on the Brain and Several Sense Organs of the Blind Deaf-mute, Laura Dewey Bridgman. The American Journal of Psychology, September, 1890, vol. iii. No. 3, p. 293, and December, 1891, vol. iv. No. 2, p. 248.

COMPARATIVE MEASUREMENT OF THE EXTENT OF SURFACE OF CEREBRAL CONVOLUTIONS.

	Surface of each lobe separately.					Free and deep surfaces of convolutions.		Whole surface of cerebrum.
	Frontal.	Parietal.	Occipital.	Temporal	Central.	Free surface.	Deep surface, including surface of insula.	
1. Gauss . . .	89,545	45,493	38,286	44,062	2,252	72,650	146,988	219,638
2. Fuchs . . .	92,380	44,783	37,927	43,468	2,447	72,100	148,905	221,005
3. Woman . . .	84,318	41,838	32,851	42,982	2,126	68,900	135,215	204,115
4. Workman . .	72,890	40,142	32,490	39,880	2,270	62,750	124,922	187,672

The methods of making these determinations are necessarily tedious and troublesome, but by such measurements either new views or corroboration of old ones as to cerebral localization may sometimes be obtained. Various methods have been adopted by different investigators. In the Bridgman case thin sheets of flexible gelatine were laid on the surface, and outlines of the gyri were traced with India ink. Copies were taken on tracing paper, and numbered. The length and depth of the sulci were also taken, and calculations of the sunken surface were made.

The specific weight of the gray substance, whether cortical or ganglionic, is less than that of the white. The latter attains its maximum from the fiftieth to the sixtieth year, the former from the fortieth to the fiftieth year. The gray matter is in larger quantity in man than in woman; the white substance is more abundant in woman than in man. The cortex may manifestly diminish in old age. The preponderance in weight of the white substance does not indicate that it has higher functions than the gray, but this superiority is apparently dependent upon the abundance of its neuroglia.

The following is a table by Baistrocchi[2] of the mean specific weight of the brain and spinal cord:

	Males.	*Females.*
White substance of the hemispheres	1.0278	1.0289
Gray substance	1.0206	1.0239
Entire brain	1.0265	1.0333
Mantle or cortex	1.0278	1.0286
Striatum and thalamus	1.0458	1.0446
Midbrain and cerebellum	1.0479	1.0584
Spinal cord	1.0387	1.0318

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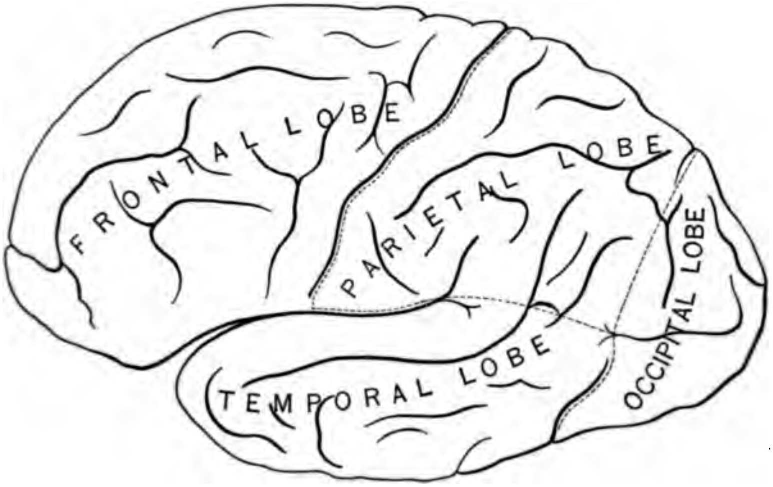
[1] H. Wagner: Maasbestimmungen der Oberfläche des grossen Gehirns, Inaug. Diss. Göttingen, 1864. Cited in Quain's Anatomy, 1893, vol. iii. part i. p. 177.

[2] E. Baistrocchi: Sul peso specifico dell' encefalo umano, sue parti e del midollo spinale e sulla determinazione quantitiva della sostanze bianca e della grigia. Rive. speriment. di fren., 1884, vol. x. p. 193. Cited by Soury, p. 336.

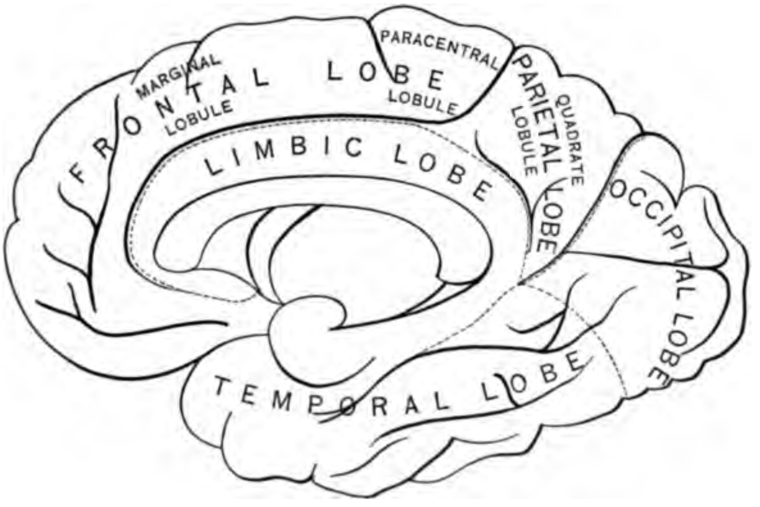
The cerebral cortex should be studied from the points of view of its gross anatomy, hemispherical, lobar, fissural, and gyral; of its minute anatomy, including the nature and arrangements of the elements of the cortex, and its methods of lamination; of its circulation, especially its arterial supply, and the independence of various distributions; and of the embryological, physiological, and clinicopathological facts that indicate its functional subdivisions.

FIG. 80.



Lobes of the cerebrum; lateral aspect.

FIG. 81.



Lobes of the cerebrum; mesal aspect.

As the circulation of the brain will be considered in another chapter, it will only be incidentally referred to here.

The cerebrum is divided into two hemispheres or hemicerebrums by the longitudinal fissure, and each hemisphere is subdivided into lobes, the special method of subdivision varying somewhat with different authors. The old lobar subdivision is into an anterior or frontal, a middle or parietal, a posterior or occipital, and an inferior temporal or temporo-sphenoidal; and for the lateral surface of the cerebrum this subdivision continues to be the one commonly used by anatomists; but on the median surface a limbic lobe has been suggested by Broca, and a falciform lobe by Schwalbe. The falciform lobe consists of that portion of the cerebrum which surrounds the callosum, and, in addition, the fascia dentata, fornix, and septum lucidum, which are not visible to external inspection of the cortex. The terms falciform lobe and limbic lobe are sometimes used synonymously; but the limbic lobe of Broca is only the outer segment of the falciform lobe, the part formed by the callosal convolution or gyrus fornicatus and the uncinate convolution.

FIG. 82.

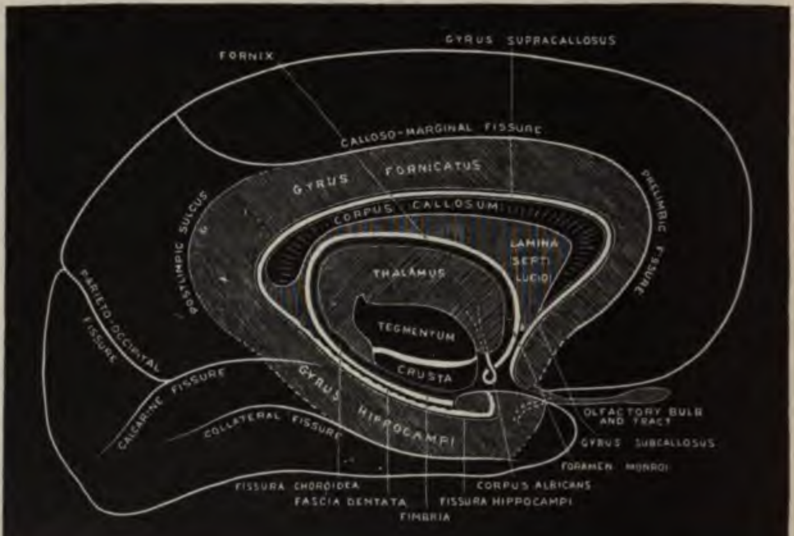


Diagram of the falciform lobe of Schwalbe; limbic lobe of Schäfer. (SCHAEFER, in Quain's *Anatomy*.)

In Figs. 80 and 81 are shown the subdivisions of the lateral and mesal surfaces of the cerebrum into lobes. The frontal lobe on its mesal aspect is sometimes subdivided in the marginal and paracentral lobule as indicated. Fig. 82 is a diagram from Quain's *Anatomy* of the falciform, or, as it has been called, the limbic lobe. To the parts included by Schwalbe, as mentioned above, are added the supercallosal gyrus, represented in man by the longitudinal striæ of the callosum, the peduncles of the callosum, and another rudimentary gyrus underneath the callosum called the gyrus infracallosus or gyrus fornicis.

Instead of this subdivision into anatomical lobes, the cortex, in accordance with its functions so far as they are known, may be conveniently subdivided into physiological lobes—motor, sensory, visual, auditory, olfactory, gustatory, and for the higher psychical functions—a subdivision which will be more fully indicated when enumerating special zones and centres.

A distinction is often made between fissures and sulci, but it is best, following Wilder, to designate all cerebral depressions as fissures, using "sulci" simply as a synonym. The main guide to the study of the most usual arrangement of fissures and gyres of the brain surface has been for many years the diagrams of Ecker,[1] which, with modifications and improvements suggested by Wilder,[2] Eberstaller,[3] Cunningham,[4] and others are still reliable for teaching purposes.

Certain fissures have ental correlatives or colliculi, that is, internal parts corresponding to them, and these are designated as total fissures. They are given by Wilder as follows:

Fissures.	*Colliculi.*
1. Calcarine	. Calcar.
2. Callosal	. Callosal eminence (in the fœtus only). (?)
3. Collateral	. Collateral eminence.
4. Hippocampal . . .	. Hippocamp.
5. Occipital	. Occipital eminence in the fœtus, lamboidal suture in the adult.

Other fissures are called partial, and these fissures may be either constant or inconstant, that is, invariably present or sometimes present and sometimes absent (Wilder).

Deeply placed within the Sylvian fissure, and, as a rule, only visible when its lips are pulled apart, is the insula or island of Reil, which is not as completely covered in the negro as in the white brain, and is more or less exposed in arrested and aberrant brains. Operculum, which means lid or cover, is in common use as a general descriptive term for each of the gyres which overlap the insula. According to Wilder, the insula is a subgyre, having been gradually covered more or less by the converging folds of adjacent regions. The operculum, preoperculum, suboperculum, and postoperculum, parts overlapping it on all sides, are supergyres.

The insula may be regarded as a true cerebral lobe, and is sometimes described as the central lobe. Embryologically, it corresponds to the floor of the Sylvian fossa. It is overlapped above by the operculum and below by the temporal lobe. It is roughly triangular, with the base upward and internal, and is grooved somewhat like an open fan with a series of nearly straight gyres, usually four or six in number, called the gyri operti, or concealed gyres. A curved furrow nearly surrounds the insula. One of the insular fissures, known as the central fissure of the insula, is large and very constant, running in the same general direction as the central fissure of the hemisphere. This divides the island into two parts, an anterior or cephalic, and a posterior or caudal portion, the latter being the smaller of the two. Eberstaller, Cunningham, and others have shown a close correspondence between the gyres and sulci of the insula and those on the lateral surface of the hemispheres. They believe its two central gyres correspond with the two central convolutions which bound the fissure of Rolando, and that its three fissures are comparable with the precentral, central, and retrocentral fissures of the brain mantle; and not infrequently something which approaches very nearly to a continuity of these fissures is observed. Variations of the gyres of the insula go hand-in-hand with variations in the fronto-parietal region. Fig. 83 is a view, after Eberstaller, of the fissures and gyres of the insula. The posterior portion of the insula is seen at 4 and 5. This part is usually termed the pars temporo-parietalis, but, according to Cunningham, a study of the development of this part of the island has shown that it is connected with the falciform lobe, and not with the temporal, and he suggests to call it the pars parietofalciformis.

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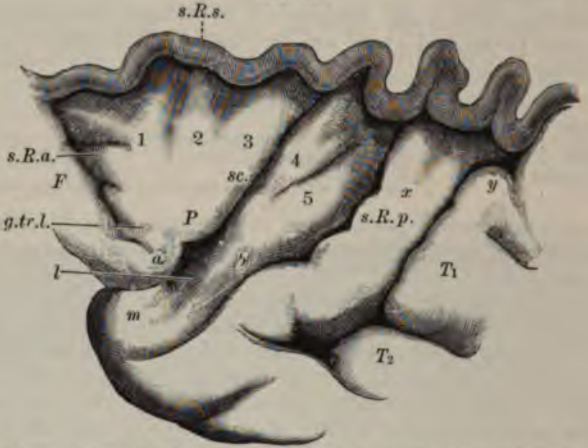
[1] Alexander Ecker: On the Convolutions of the Human Brain. Translated by John C. Galton, London, 1873.

[2] Burt G. Wilder: Brain; Gross or Macroscopic Anatomy. Reference Handbook of the Medical Sciences, vol. viii, 1889, pp. 107-164. Supplement, vol. ix. pp. 99-111. American Reports upon Anatomical Nomenclature, 1889, 1890. Extract from the Proceedings of the American Association for the Advancement of Science, vol. xxxviii. 1889, p 26. The Fundamental Principles of Anatomical Nomenclature, Medical News, Philadelphia, December 19, 1891, vol. lix. p. 708.

[3] Oscar Eberstaller: Das Stirnhirn. Vienna and Leipzig, 1890.

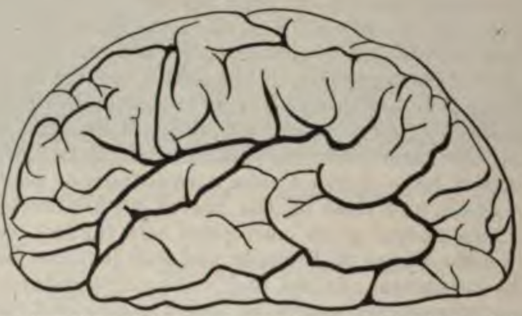
[4] D. J. Cunningham: Manual of Practical Anatomy. Edinburgh and London, 1894. Contribution to the Surface Anatomy of the Cerebral Hemispheres. Dublin, 1892.

FIG. 83.



Fissures and gyri on the surface of the insula: 1, 2, and 3. Three short gyri on the frontal part of the insula. 4 and 5. Two gyri on parietolimbic part. *s.R.a.* Anterior limiting sulcus. *s.R.s.* Superior limiting sulcus. *s.R.p.* Inferior limiting sulcus. *l.* Limen insulæ. *P.* Pole of the insula. *F.* Orbital operculum (for the most part removed). T_1. First temporal gyrus. T_2. Second temporal gyrus. *x*, *y*. Gyri of Heschl. *sc.* Sulcus centralis insulæ. *m.* Gyri on deep surface of temporal pole. (EBERSTALLER.)

FIG. 84.



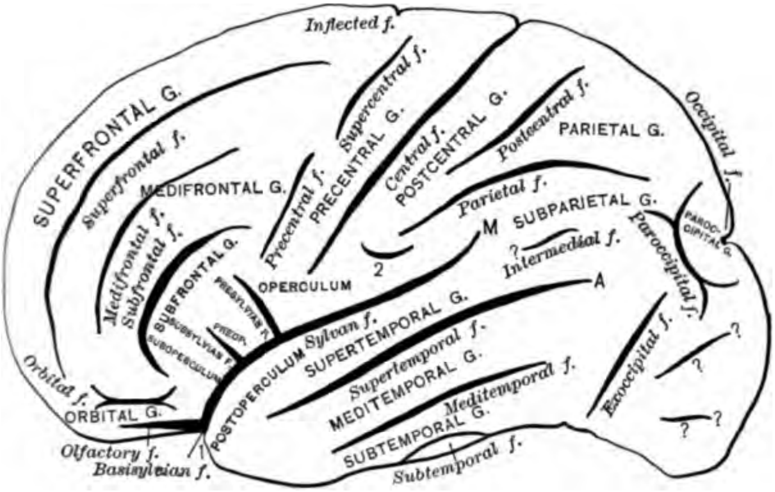
Brain showing cross-formation of the supertemporal gyre, and confluence between the cephalic half of the supertemporal fissure and the fissure separating the most cephalic of the retroinsular convolutions. The other retroinsular gyres blend with the caudal portion of the supertemporal convolution.

Caudad of the insula, at *x* and *y*, are seen two convolutions, or parts of two convolutions, crossing the Sylvian fossa, and uniting the temporal with the parietal lobes. These have been designated as the retroinsular or tempero-parietal convolutions, or the transverse convolutions of Heschl. In a number of human brains I have observed cross-fissuration of the first temporal convolution, indicating a tendency to confluence between the anterior portion of the supertemporal fissure and the fissure which separates the two most anterior retroinsular convolutions. Fig. 84 shows this relationship. The parts played by the insula and the retroinsular convolutions are still obscure and are to be solved in all probability as much by embryological study as by clinical and pathological observation.

The study of the anatomy of the cerebral surface has been made more difficult by the different names which have been given to the same fissures and gyres, and also by the differences of opinion between authorities apparently equally competent regarding the multiplication of fissural integers. One includes in his schemes and diagrams more secondary fissures than others; one describes a principal fissure as single; another as if usually divided into two parts; and still another as if it consisted of three parts.

A close study of diagrams and legends of Ecker, Wilder, and Cunningham will show these differences, but it may be well to emphasize and summarize a few of the most important features. Both Wilder and Eberstaller recognize fissural integers not accepted by Ecker, and in some cases divide fissures and gyres, regarded by the latter as single, into two or more components. Wilder makes of the precentral fissures a supercentral and a precentral; he subdivides the second frontal convolution into two parts, and submits the interparietal fissures to the same process of subdivision. Ebertstaller has carried the multiplication of fissural integers to even a greater length, and yet anyone familiar with human brains, from actual study, will be willing to concede that his anatomical scheme has been constructed from actual observation. Let me call attention to a few of the special features in Eberstaller's diagram. Instead of the single precentral fissure of Ecker, he figures an inferior, a superior, and a median precentral sulcus; the single interparietal fissure of Ecker is divided by him into three parts, which he names respectively the inferior retrocentral sulcus, the interparietal sulcus, and the anterior occipital or outer perpendicular fissure, giving also a superior retrocentral and a transverse retrocentral sulcus. In the lateral prefrontal region are given a superior and inferior frontal sulcus after the

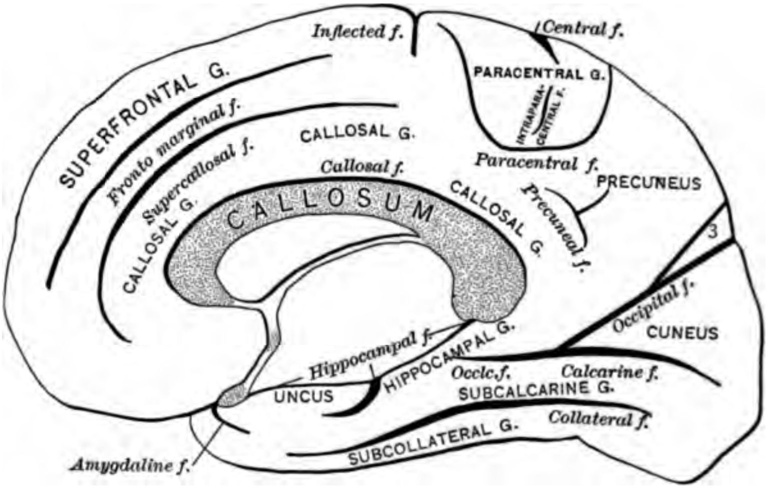
FIG 85.



Lateral aspect of the left hemicerebrum: A. Angular gyrus. M. Marginal (or supramarginal) gyrus. f. Fissure. G. Gyrus. PREOP., preoperculum. 2. Subcentral fissure. The interrogation points near the caudal end indicate Professor Wilder's doubts as to the existence of constant fissures in these places, or as to what they should be called if they do exist. (WILDER.)

manner of Ecker, and, in addition, a median frontal sulcus, in this respect following Wilder; he also figures a diagonal opercular sulcus and a radiating frontal sulcus, and subdivides the frontomarginal or orbitomarginal sulcus into three parts. The subdivision of the second frontal convolution into a median and lateral layer, and of the third frontal into a basal ascending and triangular portion are also peculiar to his diagram, and, judging from my own observations, in accordance with what is usually found in Nature. Instead of a transverse occipital and inferior longitudinal occipital sulcus, he has as an anterior outer perpendicular or ape fissure, and a lateral occipital fissure. These additions of Eberstaller will prove useful in recording autopsies and in making histological examinations of particular regions of the brain.

FIG. 86.

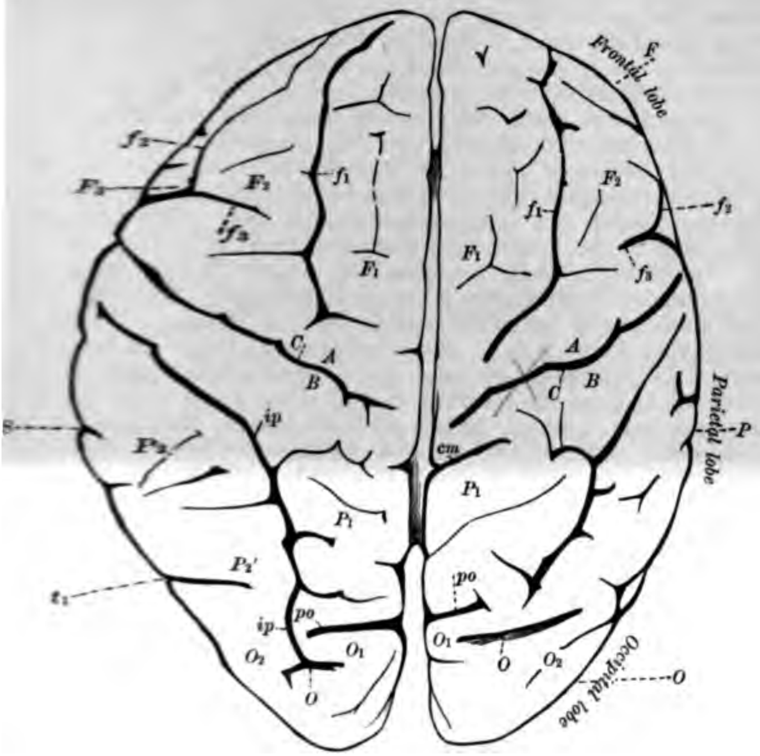


Mesal aspect of the right hemicerebrum: G. Gyrus. *f.* Fissure. *occlc. f.* Occalcarine fissure for occipito-calcarine, the common stem of the fissures demarcating the cuneus. 3. Cuneolus; the fissure cephalad of it is the adoccipital. (WILDER.)

The inflected fissure (see Figs. 85 and 86), first described by Lussana and Lemoigne, is a short fissure or indentation cutting the median edge of the hemisphere a little cephalad of the precentral fissure. It is very seldom absent in the adult human brain; in a large number of human brains examined by me it has always been present. Wilder says that at first glance it suggests the cruciate fissure of the carnivora, but he is not willing to admit this homology without further investigation. The confusion caused by a multitude of names is well illustrated in considering this fissure; it was "first described by Lussana as *solco inflesso*, which was anglicized by Wilder as *inflected fissure;* by Broca it was called *incisure preovalarie*, and by Schwalbe *sulcus paracentralis;* while Eberstaller calls it *sulcus medialis*" (Wilder). The callosal fissure, as already described, is the space between the dorsal aspect of the callosum and the overhanging cortex. The precuneal fissure is a small right-angled or T-shaped fissure in the central portion of the precuneus. The medifrontal fissure, first named by Owen, is an inconsistent fissure which sometimes divides the second frontal convolution into two parts. It is figured by Wilder, but is not always present, or at least is not always distinctive. Benedikt holds that the three-fissure or fourvolution type of the frontal lobe is a peculiarity of the brains of many inals.

The amygdaline fissure (see Figs. 85 and 86), which is usually distinct but ll, appears upon the mesoventral aspect of the temporal lobe, near its tip, nding caudad for $\frac{1}{3}$ c.m. from the horizontal portion of the Sylvan fissure, om the ventrocephalic ends of the hippocampal and collateral fissures lder). Owen and Broca recognized this fissure, the former calling it hinal. Wilder's name was *postrhinal*, but believing it might have a nt ental part, the *amygdala*, he later suggested *amygdaline*.

FIG. 87.



of the brain from above: *F*. Frontal lobe. *P*. Parietal lobe. *O*. Occipital lobe. *S*. End of izontal branch of the fissura Sylvii. *C*. Central fissure or fissure of Rolando. *A*. Anterior l or ascending frontal convolution. *B*. Posterior central or ascending parietal convolution. pper. F_2. Middle. F_3. Lower frontal convolution. f_1. Superior frontal sulcus. f_2. Inferior l sulcus. f_3. Vertical fissure (sulcus præcentralis). P_1. Upper or posteroparietal lobule. P_2. r parietal lobule, constituted by P_2, gyrus supramarginalis. P_2'. Gyrus angularis. *ip*. Inter-al sulcus. *cm*. Callosomarginal sulcus. *po*. Parieto-occipital fissure. t_1. Upper temporal e. O_1. First occipital convolution. O_2. Second occipital convolution. *o*. Sulcus occipitalis versus. (ECKER).

he subtemporal fissure is an inconstant fissure sometimes placed between third or subtemporal convolution and the fourth (subcollateral or fusi- n). Unfortunately, Eberstaller has applied this name to a fissuration the mesal aspect of the cerebrum. The exoccipital fissure of Wilder (see . 85) is an independent fissure between the occipital and temporal regions the lateral aspect of the hemisphere; when present it is nearly vertical, and has been described by Benedikt as Wernicke's fissure. It is also known as the ape-fissure, because it is homologous with the inferior portion of the external perpendicular fissure of the ape. The late A. J. Parker proposed for it the name occipito-temporal fissure. It is best defined in low-type human brains—those which approach ape-like conformations. In the brain of a criminal paranoiac I have seen it almost as well defined as in the ape.

Fissures universally admitted to be of the first importance are the fissure of Sylvius, the central fissure or the fissure of Rolando, the parieto-occipital fissure (occipital of Wilder), and the calcarine fissure. These, with the callosal, the supercallosal or callosomarginal, and the collateral fissures are the boundaries of the lobes. The Sylvian fissure has a well-defined course both at the base and on the lateral aspect of the brain. At the base, passing toward the lateral convexity, it separates the cephalic extremity of the temporal lobe from the orbital surface of the frontal, and on the lateral convexity divides into an anterior ascending limb, which is usually short, and into a longer and more horizontal posterior limb, which forms a boundary between the temporal and portions of both the frontal and parietal lobes.

The central fissure, or fissure of Rolando, is usually deep and unabridged, running from above downward and a little forward, nearly midway between the cephalic and caudal extremities of the cerebrum, as a rule, beginning close to the longitudinal and extending nearly to the Sylvian fissure. Sometimes it really or apparently passes into the longitudinal fissure, but usually the confluence is apparent, bridging fibres being present below the crests of the bounding convolutions. In rare instances it runs into the Sylvian fissure, and it may be duplicated, but this is a very rare occurrence. As both the precentral and the retrocentral fissures are sometimes nearly as large as the central, either of these may, to one not versed in cerebral anatomy, appear to be a duplicated central fissure. This duplication sometimes really occurs. At the meeting of the American Neurological Association in 1894, Professor Wilder exhibited a brain in which apparently two central fissures were present on each side; and other instances of this have been recorded by Giacomini, Calori, and Debierre.

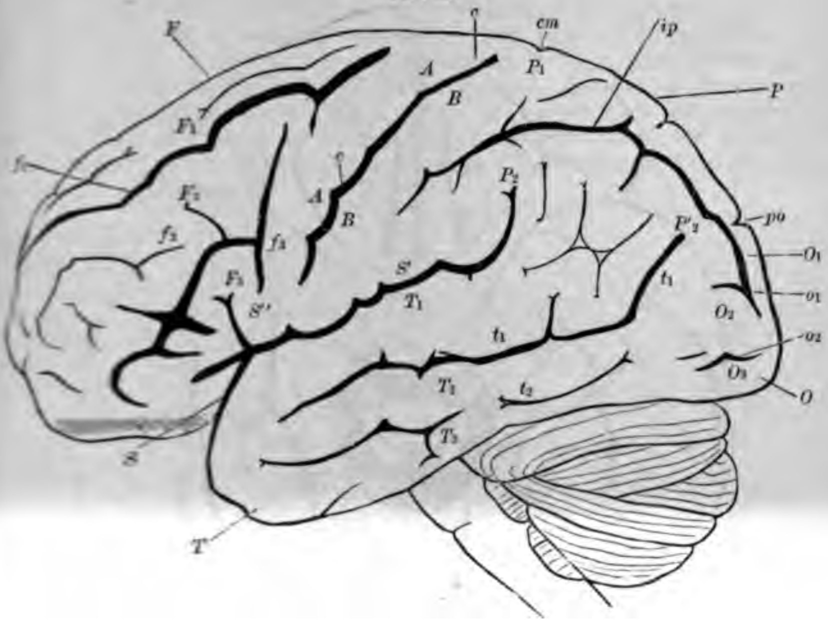
The parieto-occipital fissure of Ecker (occipital of Wilder) is seen from the lateral aspect of the cerebrum in the median edge of the hemisphere, about half-way between the upper extremity of the central fissure and the occipital pole. It extends as a deep and well-defined fissure on the mesal surface, and serves as the main demarcation between the parietal and occipital lobes.

The calcarine fissure, one of the most important cerebral fissures, corresponds in position in its anterior part to the postcornu, and has for its ental correlative the calcar or hippocampus minor. It unites with the parieto-occipital fissure to form a Y-shaped figure, which includes the gyral mass known as the cuneus or wedge. At its posterior extremity it is usually forked, but this extremity is sometimes independent of the main fissure, and has been termed the sulcus extremis by Schwalbe. The stem of the Y, which is the common stem of the calcarine and the parieto-occipital fissure, is generally considered an extension of the former. Wilder designates it as the occalcarine fissure. Occasionally it runs into the hippocampal fissure. In the brain of a paranoiac criminal examined by me this confluence was nearly complete. I have also seen the calcarine fissure bridged.

The callosal fissure is the space between the dorsal aspect of the callosum and the overhanging cortex. The callosomarginal, or supercallosal fissure of Wilder, extends from before backward along the mesal surface of the hemisphere, for the most of its length parallel with the callosum and nearly midway between it and the edge of the hemisphere. Caudad it turns upward until it reaches the edge of the hemisphere, and cephalad it follows the curve of the knee of the callosum. It is occasionally divided into two or even three parts. Its caudal curve forms the posterior boundary of the paracentral lobule. The collateral fissure, commonly described as the fourth temporal fissure, separates the temporal from the limbic or falciform lobe.

Besides these fissures others of less, but still of considerable, importance subdivide the lobes of the brain into gyres, or convolutions.

FIG. 88.



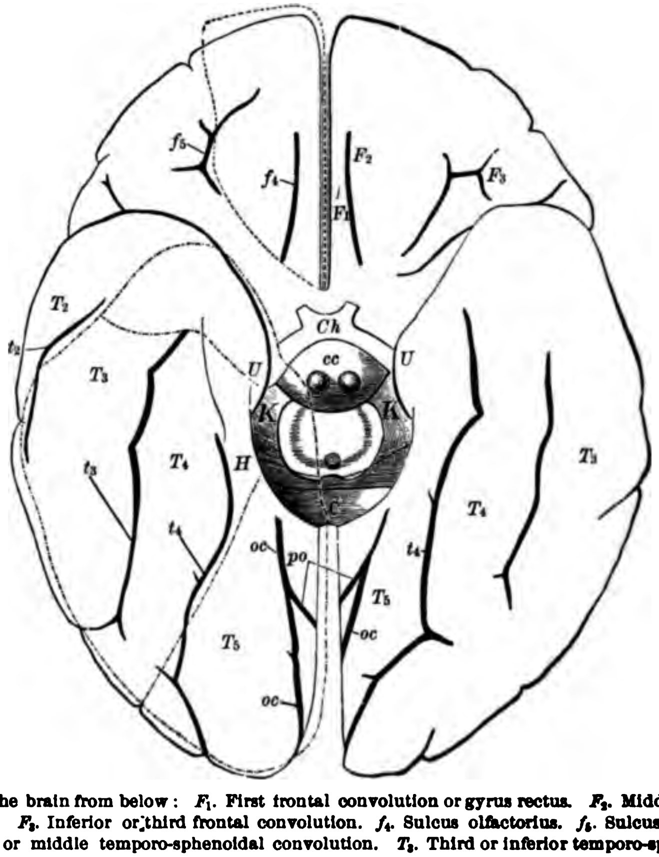
Outer surface of the left hemisphere: *F*. Frontal lobe. *P*. Parietal lobe. *O*. Occipital lobe. *T*. Temporo-sphenoidal lobe. *S*. Fissure of Sylvius. *S'*. Horizontal. *S''*. Ascending ramus of the same. *c*. Sulcus centralis or fissure of Rolando. *A*. Anterior central or ascending frontal convolution. *B*. Posterior central or ascending parietal convolution. F_1. Superior, F_2. Middle, and F_3. Inferior frontal convolutions. f_1. Superior, and f_2, inferior frontal sulcus. f_3. Sulcus præcentralis. P_1. Superior parietal or posteroparietal lobule. P_2. Inferior parietal lobule, viz.: P_2. Gyrus supramarginalis. P_2 Gyrus angularis. *ip*. Sulcus intraparietalis. *cm*. Termination of the callosomarginal fissure. O_1, first; O_2, second; O_3, third occipital convolutions. *po*. Parieto-occipital fissure. o_1. Sulcus occipitalis transversus. o_2. Sulcus occipitalis longitudinalis inferior. T_1. First. T_2. Second. T_3 Temporo-sphenoidal convolutions. t_1. First. t_2. Second temporo-sphenoidal fissures. (ECKER).

On the lateral surface of the prefrontal region two fore and aft fissures are uniformly present, and are commonly spoken of as the first, or superfrontal, and the second, or subfrontal fissures. A short distance cephalad of the central fissure, and more or less parallel with it, is a fissure called the precentral, or vertical frontal, which is not infrequently subdivided into two parts, and when this is the case the lower one can be regarded as the precentral and the upper as the supercentral. By means of these fissures, and the Sylvian and central fissures, the frontal lobe is subdivided into four convolutions, namely, the superfrontal, medifrontal (or the first, second, and third frontal), and the precentral, or ascending frontal, which lies just in front of the central fissure.

Arching over the lateral aspect of the parietal lobe is a fissure, usually

long and well defined, which has been designated as the intraparietal (Turner), interparietal (Ecker), or parietal (Wilder) fissure. It may be subdivided into two parts, and its more caudal portion is regarded by Wilder as a fissural integer, which he has named the paroccipital fissure, because it environs more or less completely his occipital (parieto-occipital) fissure. The parietal lobe is subdivided by this fissure into a superior and inferior portion.

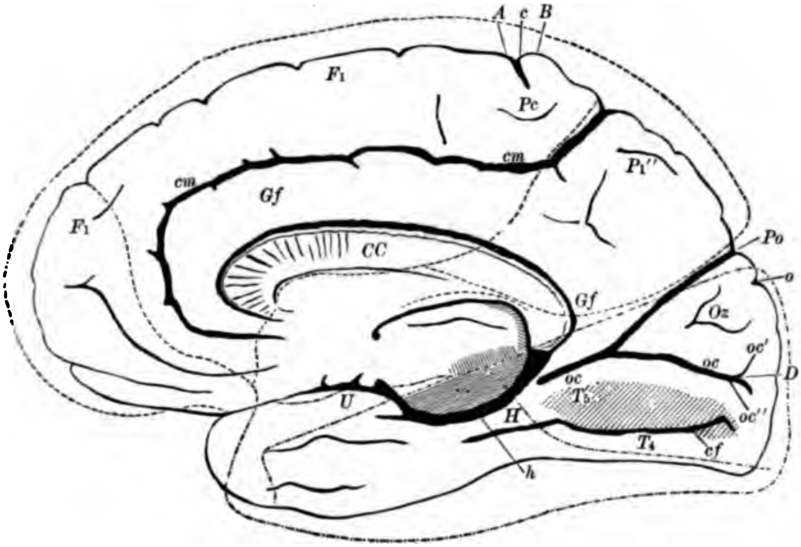
FIG. 89.



View of the brain from below: F_1. First frontal convolution or gyrus rectus. F_2. Middle or second frontal. F_3. Inferior or third frontal convolution. f_4. Sulcus olfactorius. f_5. Sulcus orbitalis. T_2. Second or middle temporo-sphenoidal convolution. T_3. Third or inferior temporo-sphenoidal convolution. T_4. Gyrus occipito-temporalis lateralis (lobulus fusiformis). T_5. Gyrus occipito-temporalis medialis (lobulus lingualis). t_4. Sulcus occipito-temporalis inferior. t_3. Sulcus temporo-sphenoidalis inferior or third temporal fissure. t_2. Sulcus temporo-sphenoidalis medialis or second temporal fissure. *po*. Parieto-occipital fissure. *oc*. Calcarine fissure. *H*. Gyrus hippocampi. *U*. Gyrus uncinatus. *Ch*. Optic chiasma. *cc*. Corpora albicantia. *KK*. Crura cerebri. *C*. Corpus callosum. (ECKER).

In order to understand the fissures and gyres of the temporal lobe, it is necessary to remember that it has a ventral, or ventromesal, as well as a lateral aspect. Passing from the horizontal branch of the Sylvian fissure ventrad, and then mesad, four fissures and five gyres are recognizable, which are enumerated by Ecker in order as the first, second, third, and fourth temporal fissures, and the first, second, third, fourth, and fifth temporal convolutions. The first two of these fissures are described by Wilder as the supertemporal and subtemporal, and the first three convolutions as the supertemporal, meditemporal, and subtemporal gyres. The ventromesal fissures and convolutions have other special names. The fourth temporal fissure is commonly described as the collateral, and the fourth temporal convolution, because of its shape, the fusiform. The fifth gyre has been given the name of lingual, because of its supposed resemblance to the tongue. As shown in the diagrams, these are occipito-temporal, rather than temporal convolutions. The fifth temporal convolution is subdivided into two parts, an anterior, known as the uncus or uncinate gyre, and a posterior, the hippocampal. The hippocampal fissure is situated to the inner side of these gyres, and is concealed from view on ordinary inspection of the brain; but by pulling aside the edge of the temporal lobe the fissure can be seen, and traversing the bottom of it a serrated ridge of gray, the fascia dentata, or dentate gyre. The latter is, properly speaking, a part of the cortex, although largely concealed within the fissure, which it follows cephalad toward the splenium of the callosum, and there becomes continuous with another smooth, gray layer, the fasciola cinerea.

FIG. 90.

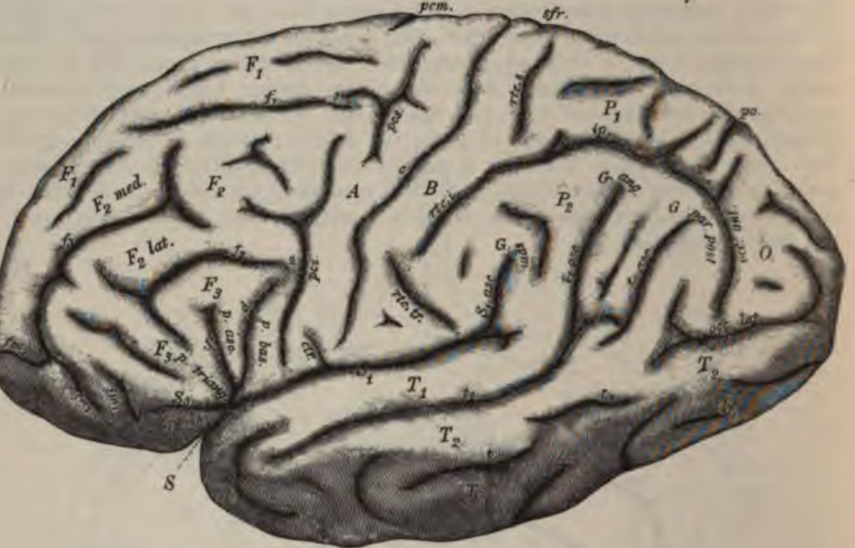


Inner surface of right hemisphere : *CC.* Corpus callosum longitudinally divided. *Gf.* Gyrus fornicatus. *H.* Gyrus hippocampi. *h.* Sulcus hippocampi or dentate fissure. *U.* Uncinate gyrus. *cm.* Sulcus calloso-marginalis. F_1. Median aspect of the first frontal convolution. *c.* Terminal portion of the sulcus centralis or fissure of Rolando. *A.* Ascending frontal. *B.* Ascending parietal convolution. P_1''. Precuneus. *Oz.* Cuneus. *Po.* Parieto-occipital fissure. *o.* Sulcus occipitalis transversus. *oc.* Calcarine fissure. *oc'.* Superior. *oc''.* Inferior ramus of the same. *D.* Gyrus descendens. T_4. Gyrus occipito temporalis lateralis (lobulus fusiformis). T_5. Gyrus occipito-temporalis medialis (lobulus lingualis). *cf.* Collateral or occipito-temporal fissure. (ECKER.)

The fissures and gyres of the occipital lobe do not always follow a well-defined plan in the human brain; but it is usual, following Ecker, to recognize on the lateral aspect of the cerebrum a superior and inferior, or a first and second occipital fissure, and a superior, middle, and inferior occipital convolution. The fissure described by Ecker as the transverse occipital fissure is present in the anterior portion of the lateral aspect of the occipital lobe. It is called the anterior occipital by Eberstaller and Schäfer, while

Wilder regards it as the caudal portion of the paroccipital. It is usually a separate fissure in the fœtus, but in most adult brains it is joined to the caudal extremity of the interparietal. On the mesal aspect of the hemicerebrum the occipital lobe is sharply demarcated by the parieto-occipital and the calcarine fissures, between which is the lobule, or gyre, known as the

FIG. 91.



Diagrammatic representation of the outer surface of the normal brain:

S. Truncus fissuræ Sylvii (trunk of the Sylvian fissure). S_1. Fissura Sylvii (outer portion of the Sylvian fissure; including S_1 *asc.* ramus posterior ascendens (posterior terminal branch); S_2. ramus anterior ascendens (anterior ascending branch). *c.* Fissura centralis (central fissure); including *ctr-* (inferior transverse sulcus). *pci.* Sulcus præcentralis inferior (inferior precentral sulcus). *pcs.* Sulcus præcentralis superior (superior precentral sulcus). *pcm.* Sulcus præcentralis medialis (median precentral sulcus). *rtc. i.* Sulcus retrocentralis inferior (inferior retrocentral sulcus). *rtc. s.* Sulcus retrocentralis superior (superior retrocentral sulcus). *rtc. tr.* Sulcus retrocentralis transversus (transverse retrocentral sulcus). *sfr.* Fissura subfrontalis (median marginal fissure). f_1. Sulcus frontalis superior (superior frontal sulcus). f_2. Sulcus frontalis inferior (inferior frontal sulcus). f_3. Sulcus frontalis medius (median frontal sulcus). *d.* Sulcus diagonalis (diagonal opercular sulcus). *r.* Sulcus radiatus (radiating frontal sulcus). fm_1, fm_2, fm_3. Sulcus frontomarginalis (parts of orbital marginal sulcus). *ip.* Sulcus interparietalis (interparietal sulcus). *po.* Fissura parieto-occipitalis (inner perpendicular fissure). *occ. ant.* Fissura occipitalis anterior (outer perpendicular, or ape fissure.) *occ. lat.* Fissura occipitalis lateralis (lateral occipital fissure). t_1. Sulcus temporalis primus (first temporal sulcus, or parallel fissure); including t_1 *asc.*, ramus ascendens (ascending ramus). t_2. Sulcus temporalis secundus (second temporal sulcus); including t_2 *asc.*, ramus ascendens (ascending branch). *A.* Gyrus centralis anterior (anterior central convolution). *B.* Gyrus centralis posterior (posterior lateral convolution). F_1. Gyrus frontalis superior s. primus (superior, or first frontal convolution). F_2. Gyrus frontalis medius s. secundus (median frontal convolution); including F_2 *med.*, median layer; F_2 *lat.*, lateral layer. F_3. Gyrus frontalis inferior s. tertius (inferior, or third frontal convolution); including *p. bas.*, pars basilaris (basal part of opercular portion); *p. asc.*, pars ascendens (ascending portion of the opercular portion); *p. triang.*, pars triangularis (triangular portion); *p. orb.*, pars orbitalis (orbital portion). 1. Lateral root of the superior frontal convolution. 2. Lateral root of the median frontal convolution. P_1. Lobulus parietalis superior (superior parietal lobe). P_2. Lobulus parietalis inferior (inferior parietal lobe); including *G. spm.*, gyrus supramarginalis (supramarginal convolution); *G. ang.*, gyrus angularis (angular convolution); *G. par. post.*, gyrus parietalis posterior (posterior parietal convolution). *O.* Lobus occipitalis (occipital lobe). T_1. Gyrus temporalis primus s. parallelus (first temporal, or parallel convolution). T_2. Gyrus temporalis secundus (second temporal convolution). T_3. Gyrus temporalis tertius (third temporal convolution). (EBERSTALLER.)

cuneus, or wedge, which is almost invariably of distinctive appearance. Ventrad of the calcarine fissures the occipital and temporal lobes merge more or less.

The convolution just above the Sylvian fissure is sometimes designated as the supramarginal, and the one below it the inframarginal, the former corresponding to the lower extremity of the postcentral and the anterior extremity of the subparietal convolution, and the latter to the supertemporal convolution. Marginal is used by some authors to describe the lobule, or convolution, between the supercallosal fissure and the edge of the hemisphere. Others regard this area as simply constituting portions of the mesal aspects of the frontal and parietal lobes. It is unfortunate that the term marginal is applied to one region bordering the edge of the hemisphere, while supramarginal and inframarginal are used to describe remotely situated convolutions which border the Sylvian fissure. Within the marginal convolutions another, called the paracentral gyre or lobule, is sometimes located. It unites the precentral and the postcentral convolutions, and, therefore, passes around the upper extremity of the central fissure; hence its name, paracentral.

The angular gyre or convolution is one to which frequent reference is made, and, as the term is used with somewhat different meanings, it may be best here to enter into an explanation. Huxley employed the term to describe the convolution which curves around the posterior extremity of the fissure of Sylvius, and Gratiolet applied the term *pli courbe*, which has a similar meaning, to the homologous gyre in the brain of the ape, in which it is limited caudad by the parieto-occipital fissure. In descriptions of the human brain "angular" is more commonly applied to the convolution which curves around the extremity of the supertemporal or parallel fissure, which is in accordance with Gratiolet's recommendation to restrict for the brain of man the term to the region between the Sylvian fissure and the occipital lobe, so that, ordinarily, the angular gyre will be constituted by the arch composed of the posterior uniting portions of the subparietal and supertemporal convolutions; the supertemporal fissure, however, terminates most frequently in the parietal lobe, but sometimes reaches into the occipital, and I have known it to extend without bridging over the median edge of the hemisphere; and a description will not hold good if the extremity of the fissure reaches into the occipital lobe, or elsewhere than its usual position. The difficulties arise largely from the term having been first used to describe appearances present in the brain of an ape, whereas the human brain had developed into a more complex region. It is best to understand that it describes that convolutional area on the lateral aspect of the hemicerebrum where the parietal, occipital, and temporal lobes come together, no matter what relations this area may bear to fissures, although in the majority of human brains this gyre will curve around the end of the supertemporal fissure.

THE MINUTE ANATOMY OF THE CEREBRAL CORTEX.

It has been held that differences in the function are indicated by the shape, size, prolongations, or other peculiarities of the nerve cells in different regions of the cortex. In some regions certain types of cells are prominent, leaving no room for doubt as to their special significance. The motor zone has been thoroughly studied, and for it, for the cerebellar cortex, and to a less extent for the regions assigned to the special senses, and to common sensation, distinctive histological features have been determined.

The views formerly held, and to some extent yet maintained, are those given in the works of Charcot, Ferrier, and Bevan Lewis. Among the most distinctive of the special elements of the cortex are pyramidal cells, comparable in every way to the motor cells of the ventral horns of the spinal cord. These are variable in their dimensions and have been placed in three classes according to their size. The largest or giant pyramidal cells were first carefully studied by Betz and Mierzejewski.

The pyramidal cells abound especially in the motor cortex, although Golgi and others have demonstrated that they exist everywhere in the gray matter. They may reach dimensions of from $\frac{1}{500}$ to $\frac{1}{600}$ of an inch in breadth, and may be four or five times as long as they are broad. They often deserve the name which has been given them of "giant cells." These cells are furnished with prolongations and processes. Some of them are of great length and of great complexity. From their apices processes pass upward to the superficial layers of the cortical cinerea, there to terminate in a tree-like panicle. Long axis cylinder processes reach downward to become constituents of the white fibres of the brain. Numerous small so-called protoplasmic processes spring from the sides of the body of the cell.

While a general agreement exists among authors as to the laminal arrangement of the cortex, the number of layers given differs somewhat according to the point of view taken, and also according to the region of the brain. Baillarger maintained it was composed of six layers, arranged from without inward in alternations of gray and white as follows: 1, white; 2, gray; 3, white; 4, gray; 5, white; 6, gray. The four internal layers are often confounded in a single yellowish-red layer, by some authorities described as a special layer. Duval admitted either five or six, according as the fifth and sixth are counted as one or two.

With reference to different regions of the brain, Meynert enumerated five types of cortical lamination as distinctive in the brain of mammals as follows: 1, a common type; 2, the occipital type; 3, the Sylvian type; 4, the type of the cornu ammonis; 5, the type of the olfactory bulb. Bevan Lewis differs from Meynert both as regards his types of lamination of the cortex in different regions, and in some cases as regards his description of the characteristic types, holding to eight distinct types of cortex, not mere fanciful distinctions based upon trivial peculiarities, but abrupt transitions from one kind of cortex to another, especially to be seen in small mammalian brains.

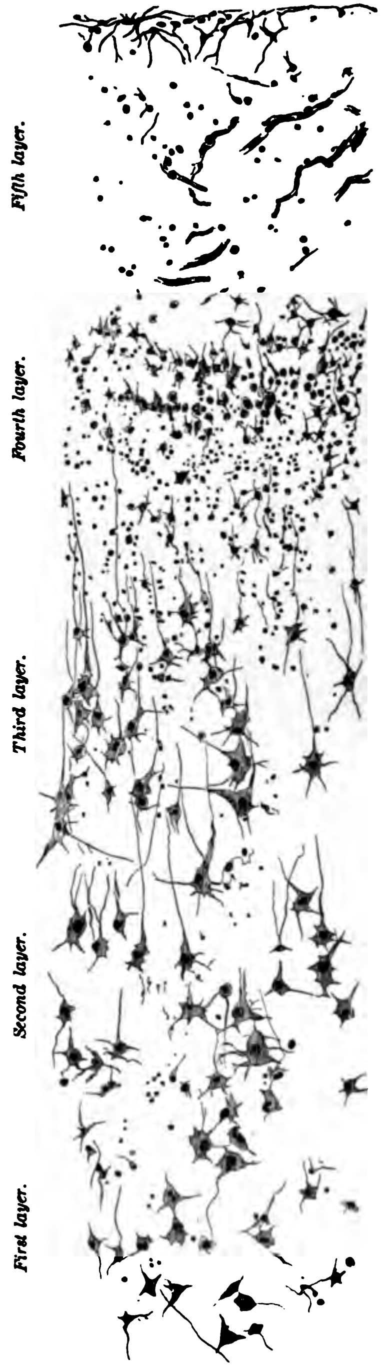
In the motor area, which has received the most attention, usually five or six layers have been enumerated by different authorities, as Meynert, Betz, Mierzejewski, Bevan Lewis, and Clark; but sometimes the layers of the motor cortex are described as only four in number. Five are made by a subdivision of the fourth, and six by a subdivision of the third and fourth. In the occipital region a larger number of separate layers are usually enumerated, as many as eight by Meynert. In other regions, as in the parietal and in the limbic lobe, the stratification of the cortex is apparently different.

The most commonly accepted lamination of the motor cortex has been that of Meynert into five layers: 1, a very thin external layer, formed almost exclusively of amorphous substance containing a few if any nerve cells; 2, a layer almost entirely composed of numerous small pyramidal cells; 3, a layer, also composed of pyramidal cells, but most of these of larger size than the preceding (the most internal portion of this lamina containing the largest cells is sometimes described as a separate layer); 4, a layer chiefly composed of myelocytes; 5, a layer of reddish-yellow color composed of fusiform and irregular cells. Lewis holds to five typical lamina for the motor cortex (Fig. 92) subdividing the third layer of the four type layers. These are: 1, an extremely delicate pale zone, devoid of nerve cells, which limits the cortex externally, and presents the features described as peculiar to the cortical neuroglia; 2, a narrow belt of very closely aggregated nerve cells of irregular marginal contour, oval, pyramidal or angular, with a proportionately large nucleus; these cells vary much in size, and are much more richly developed in some than in other regions of the brain; 3, a deep belt of nerve cells subjacent to the above elements, which are characterized by their more or less elongated contour and by the tendency to gradual increase in their size, as they lie deeper in the cortex; 4, a layer which presents us with highly characteristic nerve elements, usually large pyramidal cells, which Lewis has described as "motor cells," elements which are found modified in different cortical regions; 5, a layer represented by a series of spindle cells, which beneath the summit of a convolution are disposed radially to the surface in regular columns, separated by bundles of medullated fasciculi ascending from the core of the gyrus.

Golgi found everywhere in the cortex three great types of cells, the pyramidal, the fusiform, and the globular or polygonal, but he did not, like others, recognize them as existing in sharply isolated strata. Globular cells were found everywhere in the thickness of the cortex, as were also pyramidal cells, but the former are met with most abundantly near masses of fusiform cells, which are commonly found almost entirely in the deepest layers, while the pyramidal mainly occupy the superficial and middle lamina. Golgi therefore divides the cortex into only three layers, a superficial, middle, and deep. He does this not only for the motor area, but for the first occipital convolution, instead of the six of Lewis, or the eight of Meynert, Clark, and others.

It has been supposed that lamination of the cortex and the peculiar constitution of the lamina in different cortical areas were dependent on the function subserved, in one region motor cells, in another sensorial abounding; also that cells and their processes are large or small or have special traits according to their functions. Golgi maintains that the cells should be subdivided according to their histological charac-

FIG. 92.



Lamina of the cortex from the modified upper limbic type in the brain of the rabbit. (BEVAN LEWIS.)

teristics into motor and sensory cells, as everywhere in the brain and spinal cord these two types are found.

Recent opinion favors a general type of four layers. Ramón y Cajal, who has examined the cortex especially in small mammals, such as rabbits and cats, enumerates: 1, a molecular layer; 2, a layer of small pyramidal cells; 3, a layer of large pyramidal cells; and, 4, a layer of polymorphic cells. Fig. 93 shows a section of the cerebral cortex as described by him.

FIG. 93.

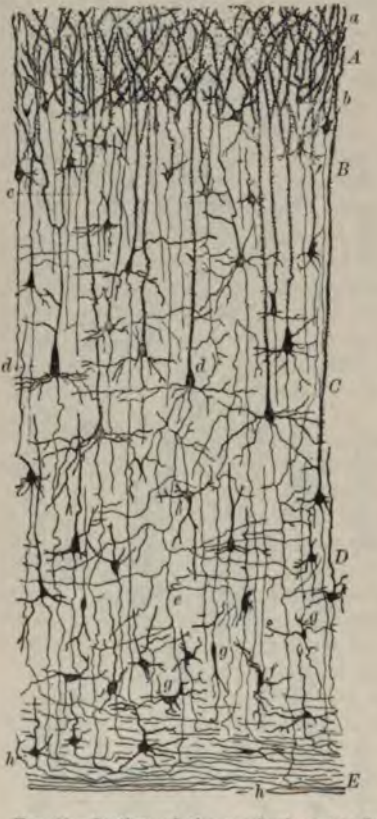


FIG. 94.

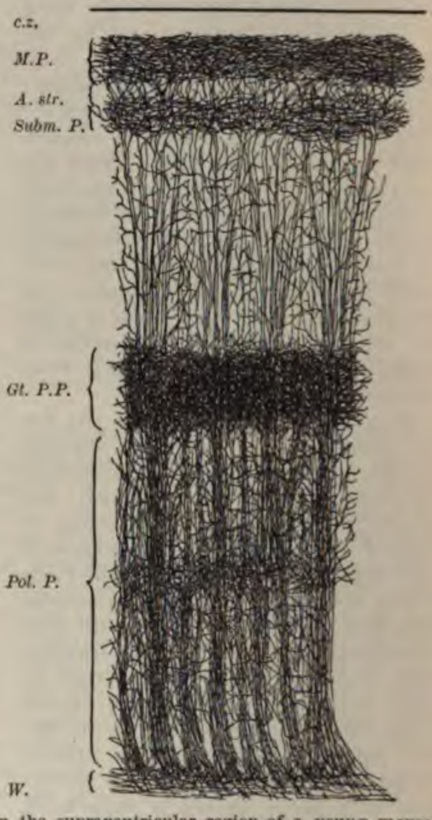


FIG. 93.—Section of the cerebral cortex from the supraventricular region of a young mouse: *A*. Molecular layer. *B*. Small pyramidal layer. *C*. Large pyramidal layer. *D*. Layer of polymorphic cells or irregular corpuscles. *E*. White matter. *a*. Panicles of the pyramids. *b*. Smallest and most superficial of the pyramids. *c*. Axis cylinder from a small pyramid. *d*. Large pyramid. *e*. Its axis cylinder. *f*. Cell with ascending axis cylinder. *g*. Similar but smaller cells. *h*. Cells found in the white matter. *i*. Rounded cell that sends its axis cylinder toward the white substance. *j*. Cell with short axis cylinder process. (RAMÓN Y CAJAL.)

FIG. 94.—Cortex of the human brain, showing the nerve-fibre systems and plexuses (combined Weigert's and Golgi's methods): *c·z*. Clear zone (free of nerve-fibre). *M.P.* Molecular plexus (Exner's), in the molecular layer. *A. str.* Ambiguous cell-stratum. *Subm. P.* Submolecular plexus. *Gt. P.P.* Great pyramidal plexus. *Pol. P.* Polymorphic plexus. *W.* White substance. (ANDRIEZEN.)

Andriezen speaks of the second of these layers as the ambiguous layer, because of the indefinite shape of many of its cells. At least eight types of cells are now recognized in the cortex; and with the new methods of staining and investigating, the different cortical layers have been largely unravelled.

Many structures, for example, have been determined as entering into the molecular or outermost layer. In the most superficial portion of this layer is a system of neuroglia fibre-cells which form a fine feltwork, and underneath this system in the same layer are certain neuroprotoplasmic structures, more than a dozen of which have been differentiated by recent methods of research. The ambiguous, the large pyramidal, and polymorphic layers have also been made to reveal numerous structures hitherto unknown. Seven cell types are described for the polymorphic layer. The intricate nerve-fibre systems of the cortex are well shown in Fig. 94. We are beginning to get new light on the functions both of the different regions of the cortex, and of its different layers, through these microscopic revelations. According to Andriezen, the sensory excitations which reach the cortex by the different systems of sensory projection fibres are distributed mainly in the molecular and submolecular regions, their terminal processes coming in contact with the apical processes of the ambiguous and great pyramidal cells, chiefly in that part of the molecular layer which is known as Exner's plexus.

CEREBRAL LOCALIZATION.

The literature of cerebral localization has attained such vast proportions that even to summarize it would be a vain task. Since the publication, in 1888, of my monograph on *Cerebral Localization in its Practical Relations*,[1] few important additions to our knowledge of this subject have been furnished by physiology and clinico-pathology, although much of the earlier work has been reviewed, new points of departure have been taken, and new methods of regarding phenomena have been suggested. Soury[2] in 1892 devoted more than four hundred pages, which proved scarcely sufficient, to a critical and historical review of the literature of the functions of the brain. Recent work which promises most for the solution of the problems of cortical function is based upon microscopical investigation. Golgi's method of silver staining has revealed a new world to the neuro-histologist, and the work inaugurated by him and carried forward by his pupils, and by Ramón y Cajal, Van Gehuchten, Kœlliker, and others, bids fair to place upon a firm foundation the whole subject of brain functions. It has become possible by this method, and other methods which are its natural outcome, to trace in a manner never before possible the processes of nerve cells.

Whatever views may be held as to the nature of localization, for the practical purposes of the physician and surgeon the cerebral cortex can be divided into a series of physiological lobes, and these lobes into more or less numerous subareas and centres. The lobes are regions for motion, common sensation, vision, hearing, naming, smell and taste, and for the higher mental faculties.

MOTOR LOCALIZATION.

As would be anticipated, the subject of motor representation has been more fully developed than any other branch of cortical localization, having become, for most specialized movements, an exact science. As the proper interpretation of localizing phenomena has been much disputed it may be well first to briefly consider some of the differing views.

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[1] Read before the Congress of American Physicians and Surgeons, Washington, D. C., September, 19, 1888. This paper has been used freely in the preparation of this chapter.

[2] Jules Soury: Les Fonctions du Cerveau, Doctrines de l'École de Strasbourg, Doctrines de l'École Italienne. Paris, 1892.

Franck[1] summarizes the explanations of the action of the excitable regions of the cortex under several heads, as follows: (1) they are true motor centres, (2) they are sensory centres (reflex theory), (3) they are at the same time motor and sensory, (4) they are places of confluence and passage of excitations originating elsewhere, (5) they are simply places which receive and transmit excitations to the regions of movement below.

Ferrier[2] believes that the motor zone contains psycho-motor centres, or centres for movements which involve conscious discrimination; movements which are automatic, instinctive, or responsive are more or less distinctly organized below the cortex. He adheres in his latest work to separate sensory localization. The motor centres, he holds, although functionally and organically connected, are anatomically differentiated from the centres of sensation, general as well as special. The functional connection between sensory and motor centres is necessary to be borne in mind. Ferrier admits that the motor centres are not independent centres of action, but respond only to stimuli which pass from the sensory centres by way of associating fibres. Marique's experiments, which have been confirmed by Exner and Paneth, show that when the motor centres have been completely isolated, by section of the fibres which associate them with the sensory centres of the cortex, paralysis results of precisely the same character as that which occurs when they are actually extirpated; also that the same contractions were obtainable on electrical irritation of the respective centres after, as before, isolation, showing that they still retain their excitability and connection with the pyramidal tracts.

Charcot's views were practically those of Ferrier, namely, that these centres were the cortical substrata of motor activities. Exner originated the theory of *absolute* and *relative* centres, according to which lesions which cause affections of particular parts, as of the upper extremities or of the face, are chiefly grouped in definite localities, but nevertheless the same effects may result from lesions of almost any part of the cerebral convexity. Absolute centres are those destruction of which always causes certain symptoms; while lesion of relative centres frequently, but not invariably, induce these phenomena.

According to Bastian, the Rolandic centres are kinesthetic; he refers the movements to their sensory or centripetal antecedents, believing the only true motor centres are bulbar and spinal; but, as Waller points out, all that he really denies is that these centres are spontaneously motor.

Both Hitzig and Nothnagel looked upon the Rolandic region as related to the so-called musclar sense. Nothnagel regarded this area as containing centres of muscular consciousness, or centres wherein conceptions of the normal progress of movement were formed. Schiff held that these Rolandic centres were tactile, destructive lesions, causing ataxia and irritation, resulting in movements of a reflex nature.

Munk regards the cortical Rolandic region as the sensory area, or *Fühlsphäre*, believing that it stands in the same relation to common sensation, meaning sensation of the whole body, as the cortex of the occipital lobe does to vision, and that of the temporal lobe to hearing. Tripler, Goltz, Luciani and Seppili, Starr, Dana, and others also hold that affections resulting from destruction of the so-called motor zone are in some way related to disorders of sensibility in the limbs affected.

According to Horsley,[3] in the so-called motor region three functions are clearly represented, as shown in the diagram, Fig. 95. They are, 1, slight

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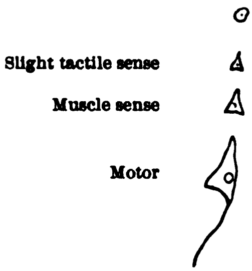
[1] François Franck: Leçons sur les Fonctions Motrices du Cerveau, Paris, 1887, p. 362.

[2] David Ferrier: Croonian Lectures on Cerebral Localization, London. Smith, Elder & Co., 1890.

[3] Victor Horsley: Trans. Cong. Amer. Phys. and Surgs., 1888, Washington, D. C., p. 341.

representation of the tactile sensation; 2, representation of the so-called muscular sense; and, 3, great representation of movement. "These three functions are wrapped up closely together, and in every particle of gray surface there is represented this triune function for a single segment of the body."

FIG. 95.



Horsley's view of sensorimotor representation in the motor cortex.

According to Waller, who has given close attention to both the psychical and physical sides of the subject of localization, the main drift of development is from undifferentiation toward specialization. Secondary connections may become established, but the main and primary movement is disconnection. Localization is the law of all organization, and it would be marvellous if the cerebrum presented an exception. He contends that every centre must be a point to which, as well as from which, an impulse goes. He pictures one undivided change taking place in any centre, resulting in impulses both to it and from it; he avoids the term motor, because it may imply spontaneous origination of force. He speaks of his two laboratories, in one of which he learns, and the other where he teaches, comparing the first to a sensory and the other to a motor region, and the highway by which he travels from one to the other he compares to the internuncial tract between them; but he argues the laboratories themselves are neither sensory nor motor; they are places in which sensation and motion are produced. "The truism that by a sensory centre, or motor centre, we cannot mean a centre which feels or which causes motion, but at most a place where sensation occurs or a motor impulse is elaborated, is worth while making particularly clear, if only as a matter of definition."

The view expressed in the latter part of the preceding sentence is the only one that need to be accepted by the practical physician; he simply needs to know that something occurs in a so-called sensory region, on the one hand, or in a motor, on the other—something which gives rise in each case to clinical manifestations of a special character.

In reality, the special functions of different cerebral zones are determined by the fibres which go to them from the periphery. We are gradually reaching more scientific and more sensible ideas of cerebral structures and function, having been handicapped too long by erroneous views as to the nature of cell action. It has been too much the fashion to speak of the cell as the citadel of nervous energy; to regard it as the true source of power, and to believe that it has spontaneity. The supposed necessity of structural continuity from centre to periphery has also held us back. Wundt, Forel, and Nansen (admirably summarized by Soury) have shown the fallacy of these views. Forel first saw that it was only necessary for the nerve fibrils to be in contact in order that nervous impulses should be conveyed, nerve transmissions being in this way comparable to the method of electrical transmission. He protested against speaking of motor cells and sensory cells, the only differences between motor and sensory cells being that the cell body is central for the former and peripheral for the latter. The fibres from both terminate in arborescent ramifications, the motor nerves in the muscles, the sensory in the cortical cinerea. The nerve processes, according to Nansen, are of two kinds—nerve fibres proper, and protoplasmic processes. The former pass toward the periphery from the cortex; they do not anastomose with nerve cells by means of these prolongations. The true nerve cells have processes always single, but with lateral branches. They directly become nerve tubes, or lose their individuality, and give birth to nerve trees in the network of the cortex. The main point of Nansen's view is that nerve impulses may be transmitted to or from the higher centres without passing directly to the nerve cells—that is, by the cell bodies; also that a reflex can be constituted through the ramifications of the cells. Impulses are conveyed from processes to processes through an entire reflex arc; through the entire length of a cortico-efferent, or a cortico-afferent, projection system. The function of the nerve-cell body is trophic; its nuclei and nucleoli preside over the nutrition of the long or short fibres which pass out of or grow into them. Cells are of enormous bulk, in order that they may be able to sustain these processes. The aggregation of gray matter at various levels of the nervous system are watering and feeding places, not places for renewing nerve activity. In the central fibrillary network of the cortex the degree of development of intelligence will be proportioned directly to the complexity of this reticulum.

The acceptance of views such as these does not call for an abandonment of the doctrine of localization, even as held by its most advanced advocates; at the most it simply requires the subject to be looked at from a different point of view, or to be explained in different terms. At certain levels, and in certain areas of given levels, in the great sensorimotor path or arc occur or can be elicited certain phenomena, which only show themselves outwardly in the one case by the sensory and in the other by motor manifestations.

James compares the motor area to the mouth of a funnel, from which pour out impulses caused by incoming impulses.

According to Brown-Séquard,[1] the most persistent and constant opponent of modern localization, numerous contradictory facts show that nerve cells possessed of definite functions, instead of being united in a certain cerebral territory, are dispersed throughout the encephalic mass, and are joined in some way one to the other, so as to form a functional solidarity; also, that all symptomatic manifestations of cerebral origin arise exclusively from an irritation, which acts either by arresting or exaggerating the activity of the encephalon. Dupuy,[2] who also believes that the doctrine of localization as generally understood cannot be accepted, holds that as no agent but electricity produces movements from application to the motor cortex, when a current of minimum strength applied to certain spots in the cortex produces certain motor effects, it only shows that these points are situated in the line of the least resistance. He criticises the flap experiments of Putnam, and the circumvallation, freezing, and other experiments of Franck, and reproduces the old cases and the old arguments regarding destructive lesions, which have not produced paralysis or aphasia, and also the well-known facts that the cortex is not absolutely necessary for the production of epileptic convulsions. His most interesting argument is drawn from the teachings of Golgi and Nansen, that the cells are trophic elements, and that motion and sensation are the attributes of the peripheric elements alone, the central gray matter simply reacting according to the nature of the impulse.

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[1] Brown-Séquard: Archives de Physiologie, January, 1890.
[2] Eugene Dupuy: The Rolandic Area Cortex. Brain, 1892, vol. xv. p. 190.

Goltz has ridiculed those who consider the brain a mosaic of little brains, and yet he acknowledges that the functions of the forepart of the cerebrum of the dog are not the same as those of the hindpart, and he has never denied the possibility of the existence of different cortical territories related to diverse functions; but he affirms that the most important functions of the brain—those which indicate the passions, the emotions, and the instincts—do not depend upon such circumscribed areas (Soury). He would seem to hold that the phenomena usually attributed to the ablation or irritation of isolated cortical centres or areas are due to inhibition or interference with lower centres, admitting only loss of perception of a general character.

Mott,[1] who believes in the sensorimotor functions of the central convolutions, in summarizing the evidence in favor of this doctrine, cites the embryological work of Flechsig and Hosel, which he believes shows that afferent channels connect the nuclei of the posterior columns, and the sensory nuclei of the fifth nerve, with the opposite Rolandic area either directly by the fillet, or indirectly by the fillet and thalamus. The real claim made by Flechsig is that the sensory fibres terminate in the region *behind* the central fissure; and it is not quite sound to argue that because fibres run from behind forward these embryological facts teach that sensory fibres terminate in the motor cortex.

SUBDIVISIONS OF THE MOTOR AREA.

The motor zone is divided horizontally on the lateral aspect of the hemispheres into three great regions for the face, arm, and leg, which correspond nearly, but not exactly, with the imaginary extensions caudad of the superfrontal and subfrontal fissures. Instead of subdividing the Rolandic region from above downward into thirds, as is often done, it is better to divide it into fifths, placing the area of representation for the lower extremity in the upper fifth, and that for the upper extremity in the middle two-fifths, and that for the face in the lower two-fifths.

The anterior branch of the Sylvian fissure, and an imaginary line extending from it to the longitudinal fissure, may be regarded as the cephalic or anterior boundary of the motor area; the retrocentral fissure may be considered its caudal or posterior boundary, the posteroparietal region being concerned, in part at least, with sensation. The horizontal branch of the fissure of Sylvius is its ventral or inferior boundary. For a long time the longitudinal fissure was regarded as the superior boundary of the motor area, but Horsley and Schäfer, and others since them, have shown that this area extends over the edge of the hemicerebrum. They found that the application of the electrodes at successive points from before backward in the marginal convolutions produced: (1) movements of the head; (2) of the forearm and hand; (3) of the arm at the shoulder; (4) of the upper (dorsal) part of the trunk; (5) of the lower (pelvic) part of the trunk; (6) of the leg at the hip; (7) of the lower leg at the knee; (8) of the foot and toes. These primary movements are almost invariably complicated by secondary movements, produced by excitation of the adjacent parts. The paracentral lobule is the convolution of the mesal surface of the cerebrum, chiefly concerned with the movements of the leg and foot.

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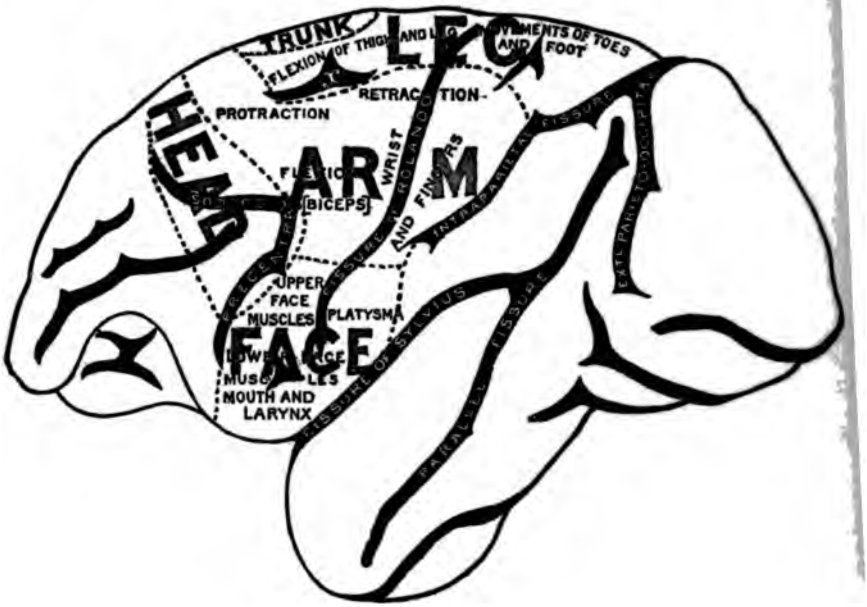
[1] F. W. Mott: Jour. Physiol., 1894, vol. xv. pp. 464–487.

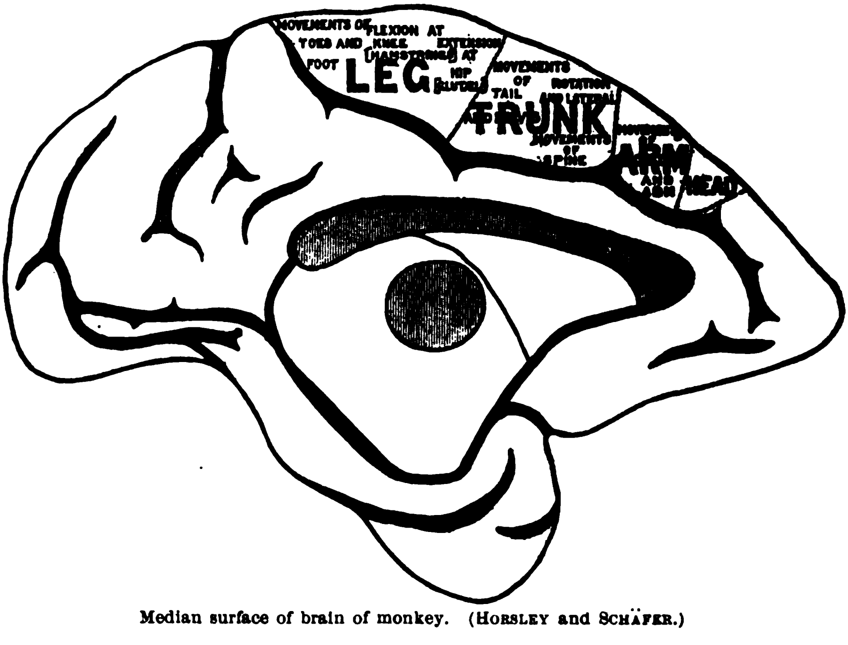
The subdivision of the motor area on the lateral aspect of the hemicerebrum into subareas for speech, the head and eyes, the face, arm, leg, and

FIG. 96.



Lateral surface of brain of monkey. (HORSLEY and SCHÄFER.)

FIG. 97.


MOVEMENTS OF TOES AND FOOT
FLEXION AT KNEE [HAMSTRINGS]
EXTENSION AT HIP [GLUTEI]
LEG
MOVEMENTS OF TAIL
ROTATION AND LATERAL
TRUNK
MOVEMENTS OF SPINE
ARM
HEAD

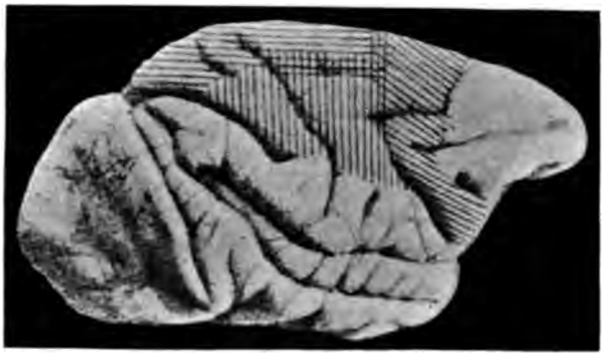
Median surface of brain of monkey. (HORSLEY and SCHÄFER.)

trunk, and the further division of these subareas into a few centres for certain specialized movements of the face, arm, and leg, are represented in Fig. 96. The motor area of the lateral surface of the hemisphere is made to include the caudal portions of the three frontal and both central convolutions, but not to reach backward so as to take in the superior and inferior parietal convolutions. The areas for the arm extend cephalad and mesad to the edge of the hemisphere. The subdivision of the head, arm, trunk, and leg areas in the marginal lobule on the mesal surface of the hemisphere are shown in Fig. 97.

Area for the Movements of the Head and Eyes. Ferrier found that irritation at the base of the superior and middle frontal convolutions gave rise to lateral movements to the opposite sides with dilatation of the pupils, the expression assumed by the monkey being that of attention and surprise; but the same movement occurs with other special reactions, on stimulation of the angular gyrus, and more especially of the superior temporal convolution. With the latter is associated pricking of the ear from stimulation of the auditory centre; but Ferrier argues that these are simply attention movements, the same as would result from stimulation of the motor centres for these movements. Destruction of the oculo-motor centres of Ferrier, according to some experiments, causes conjugate deviation toward the side of the lesion. Bilateral destruction of the centres at first caused inability to turn the head and eyes, but the animal recovered.

A minute physiological study of the representation of the head and eyes was made by Beevor and Horsley,[1] who studied the following list of movements: "I. *Movements of the Head.* The head moves in one of the following modes or in combination of the same: (*a*) simple horizontal rotation to the opposite side; (*b*) rotation with elevation of the muzzle; (*c*) rotation with adduction of the head to the (opposite) shoulder. II. *Movements of the Eyes.* (*a*) Both eyes open; (*b*) both eyes turn horizontally to the opposite side and upward; (*d*) both eyes turn to opposite side and downward; (*e*) both eyes turn to middle line (from the side stimulated), or turn only through a few degrees. III. *Movements of the Pupils.* (*a*) Contraction; (*b*) dilatation."

FIG. 98.



Areas of representation of the movements of the head and eyes (oblique shading), of the upper limb (vertical shading), and of the lower limb (horizontal shading). (BEEVOR and HORSLEY.)

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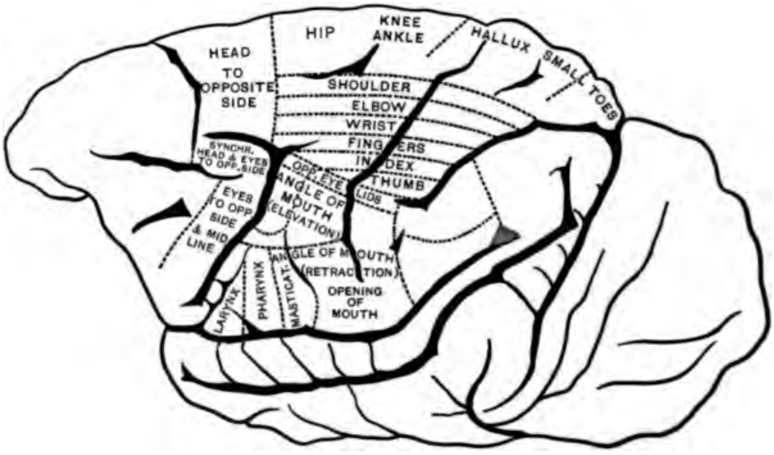
[1] Beevor and Horsley: Phil. Trans. of Royal Soc. Lond., (1888), vol. clxxix. B. pp. 205-256.

The area determined as representing the movements of the head and eyes is indicated in Fig. 98 by the oblique shading. They found it to be a very extensive region, reaching from the longitudinal fissure above almost to the Sylvian fissure below. The turning of the head to the opposite side—so that the face looks away from the hemisphere stimulated—has a much larger area of representation than the synchronous conjugate deviation of the eyes. The area in man is probably much the same. Beevor and Horsley found that simple horizontal rotation to the opposite side occurred most markedly from irritation of the cortex just above the horizontal branch of the precentral fissure. Rotation with elevation of the muzzle, as in the movement when the head is tilted backward, so that the face looks upward as well as outward, was also observed above this fissure. Rotation with adduction of the head to the opposite shoulder is rarely a primary movement, but was observed occasionally from irritation along the lower border of the horizontal limb of the precentral fissure. The movement of opening both eyes was represented just above but close to this fissure, and that of turning both eyes horizontally best in front of the precentral fissure, but to a less degree behind it. The movements of the eyes in other directions mentioned occurred at various points near the transverse limb of the precentral fissure. Dilatation of the pupils, when it occurred, which was but rarely, appeared to be represented around the horizontal limb of the precentral sulcus. In Fig. 99, which indicates the foci of representation of movements of all the small segments of the body, are shown the areas for the movements of the head and eyes together, of the eyes to the opposite side, and of opening the eyelids.

FIG. 99.



Cortical foci of representation of movements of small segments of the body. (BEEVOR and HORSLEY.)

Area for Associated Eye Movements, according to Mott. Mott,[1] as the result of recent researches on associated movements produced by unilateral and bilateral cortical faradization of the monkey's brain, concluded: (1) That the area of the cortex of the frontal lobe, which, when stimulated, gives rise to associated eye movements, can be divided into three zones—(*a*) a middle zone immediately below the horizontal part of the precentral sulcus, faradization of which is followed by simple lateral deviation of the opposite side; (*b*) an upper zone immediately above this, which may extend to and include part of the marginal gyrus; this gives, on faradization downward, inclination combined with lateral deviation; (*c*) a motor zone immediately below the middle one, and sometimes extending nearly down to the margin of the hemisphere; this gives upward inclination, usually combined with lateral deviation. (2) That similtaneous bilateral faradization of the frontal cortex at identical points invariably brings the eyes into position of looking straight forward. (3) Bilateral faradization of identical points in the visual area produces similar results to those obtained in the frontal area. (4) Weak stimulation of the frontal area sufficed to overcome one of the occipital visual area.

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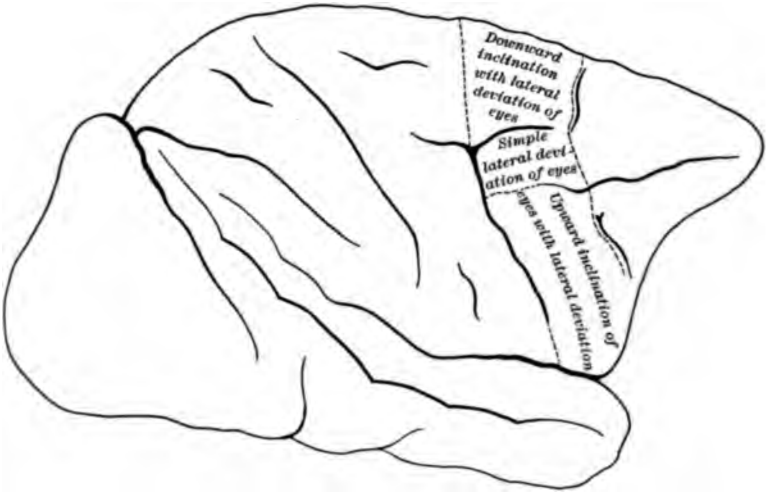
[1] F. W. Mott: Brit. Med. Journ., vol. i. June 21, 1890, p. 1419.

These areas are shown in Fig. 100, and in one of my own cases in which a trephine opening presumably uncovered the lower halves of the two central, the posterior extremity of the second frontal, and the posterior superior corner of the third frontal convolutions, a careful application of a weak faradic current to the cortex, at a point which corresponded to the posterior extremity of the second frontal convolution, caused distinct deviation of the head to the opposite side.

FIG. 100.


Downward inclination with lateral deviation of eyes
Simple lateral deviation of eyes
Upward inclination of eyes with lateral deviation

Cortical areas for associated eye movements, according to Mott.

In one of Horsley's cases, in which operation was performed at the point of meeting of the areas for the movement of the trunk, protrusion of the upper limb, and turning of the head and eyes, the aura was contraction of the abdominal muscles followed by turning of the head and eyes to the opposite side. Other cases have been reported in which turning of the head was the starting-point of the spasm. In some cases at least, when the aura or signal symptoms can be most certainly shown to be turning of the head and eyes to the side opposite the lesion, the probabilities are that the focus or primary seat of the irritation is from a lesion in this oculomotor region. The fact that the cortical oculomotor palsies are not present as a persistent condition, even when we have definite lesions of the second frontal gyre, is not an argument of weight against the existence here of oculomotor centres. Such persistent oculomotor paralysis was not present in one of the best defined cases of lesion in the second frontal gyre ever reported, a case occurring in my wards of the Philadelphia Hospital. Such symptoms do not persist, because of the automatic nuclear mechanism of the cranial nerves related to these centres.

Conjugate deviation of the head and eyes, when a persistent or permanent symptom, is most likely to arise from lesions of the pons, cerebellum, or cerebellar peduncles.

Fraenkel[1] has reported a case in which there was excessive rigidity of the neck muscles, which would seem to bear out this conclusion, as examination showed the existence of a large clot covering the foot of the second and the adjacent part of the third left frontal convolution, immediately in front of the anterior central convolution.

Centre for Elevation of the Upper Eyelids (Action of the Levator Palpebræ Superioris). The experiments of Ferrier, of Beevor and Horsley, and of Mott indicate the existence of a centre for elevation of the upper eyelid in the caudal portion of the second frontal convolution, which would be its most probable position, because this movement is usually associated with movements of the eyes and head, which are represented in this and adjoining gyres. Landouzy, Grasset, Rendu, and others have recorded isolated cases of paralysis of the upper eyelid with cortical lesions in or near the angular gyre; but although a stimulation of this region or an area nearby sometimes causes elevation of the eyelid with other phenomena, this reponse is to be regarded rather as a reflex from sensory stimulation, than as due to excitation of a motor centre. An animal when appealed to through the sense of sight and hearing elevates its eyelids as a sign of attention. Destruction of the angular gyre in animals does not cause paralysis of any kind, and many cases of lesion of this region without ptosis or any other paralysis have been reported by competent observers.

Centre for Laryngeal Movements. In 1877, Seguin[2] reported a case of left hemiparesis, with impairment of speech, and also of phonation, the patient after an attack being never able to control the pitch of the voice, apparently from lack of proper action of the muscles of the pharynx and larynx. Autopsy showed the surface of the right third frontal convolution degenerated, being yellow, tough and elastic, and the same change to a less extent was found in the same location on the left side. A motor centre for laryngeal movements has therefore been assigned to the posterior extremity of the third or subfrontal and the anterior extremity of the precentral convolutions, and this is supposed to be better differentiated in the right than on the left side. Krause,[3] in 1883, on excitation of the cortex, noticed rise of the larynx and movement of the vocal bands to a position midway between expiration and phonation, lifting of the palate, contraction of the constrictor pharyngis, and movements at the base of the tongue. With extirpation experiments he found that eight dogs had lost the power of barking, on attempting which they only uttered a hoarse whine or sound. Garel[4] has reported a case of vocal paralysis in which the inferior portion of the precentral gyre on the *right side* was slightly adherent to the meninges, and the membranes being stripped from the surface beneath presented a light yellow discoloration; so at the foot of the third frontal gyre were two red points of softening. Semon[5] and Horsley found that excitation of the lower end of the precentral gyre at its most cephalic part brought about phonatory closure of the vocal chords, and a fair amount of other evidence has been collected to show the existence of a cortical centre for laryngeal movements. Masini reproduced the experiments of Krause, and found that the laryngeal centre had its principal focus, as indicated by Krause, at the foot of the precrucial convolution of the dog; and yet, according to Masini, the unexcitable areas of the larynx embraced nearly all of the motor zone. To cause paresis of the muscles of the larynx, he found it necessary to practice bilateral extirpation of the entire cerebral mass in front of the crucial fissure.

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[1] Fraenkel: Charité Annalen, 1886, cited by Gowers in Diseases of the Nervous System, 1893, vol. ii. 2d ed.

[2] E. C. Seguin: Referred to by Delevan in Med. Rec., N. Y., Feb. 14, 1885.

[3] Hermann Krause: Cited by Horsley and Schäfer and Delavan.

[4] Garel: Annales des Maladies de l'Oreille et du Langue, 1886, tome xii. p. 218.

[5] Felix Semon and V. Horsley: Phil. Trans. Royal Soc., 1888, vol. clxxix.

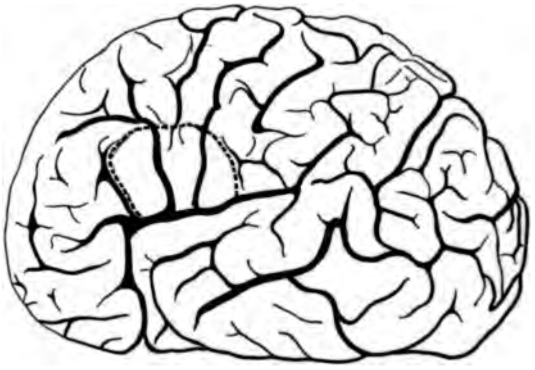
Centre for Movements of the Jaw. The movements of the jaw are probably represented in the immediate vicinity of the centres for the larynx, throat, tongue, lips, and face. In the course of an operation Horsley found that electrical excitation of the precentral convolution, at the junction of the middle and upper thirds of the area for the face, caused lateral movement of the jaw, as well as of the angle of the mouth, such as might be produced by the conjoint action of the platysma and the pterygoid muscles. In Horsley's diagram, Fig. 99, of the cortical foci or representation of movements of small segments, centres for laryngeal, pharyngeal, and masticatory movements are placed one behind the other, between the presylvian or ascending branch of the Sylvian fissure and the foot of the precentral fissure.

The movements just considered—those of the larynx, pharynx, and jaws —are among those which have a bilateral representation of the cortex; they are movements performed by muscles which usually act together on the two sides of the body. The cortical centre on either side is sufficient for the representation of movements of both sides. What is ordinarily termed a hemiplegia is never a complete hemiplegia; the movements of the trunk, of the neck, of inspiration, and some of the movements of the face, particularly those of the jaw, and of the orbicularis palpebrarum and the levator palpebræ muscles, are not affected at all, or only slightly. The double representation of the cortex is more or less complete, according to the degree of the association of the two halves of the body. The orbiculo-palpebral movement is, to a certain extent, and particularly in some individuals under the control of the will, and in such cases will have a distinct unilateral representation in the cortex. This is sometimes greater for one side than the other, as is illustrated by the fact that the individual can wink with one eye voluntarily better than with the other. It is necessary to have double lesions in order to produce complete paralysis of these bilaterally represented movements.

The Face Area. The face area is subdivided into an upper and a lower subarea. In three cases during operation I have observed faradization of the anterior superior portion of this face area produce contraction of the opposite angle of the mouth and face. It is probable that in the extreme upper anterior portion of this area is a subcentre for such movements of the upper face as contraction of the frontalis and obicularis palpebrarum muscles, although as these movements are, as as a rule, bilaterally associated, such a centre does not seem to come out clearly as the result, either of physiological investigation or the experiments of disease. Berkeley fixed the cortical centre for the movements of the angle of the mouth in this upper subarea for the face, but probably lower than the centre for orbicular movements. While it is not usual, paralysis in the upper distribution of the facial nerve sometimes takes place as the result of a cortical lesion.

Orolingual, or lip and tongue, centres are situated in the anterior portion of the lower subarea of the face, a little behind the centres for the throat and larynx. In a case of typical orolingual paralysis, recorded by me, the patient had distinct facial paralysis in the muscles supplied by the lower distribution of the seventh nerve, and also lingual paresis; probably also slight want of control over the right orbicularis palpebrarum. He had some power over the nasal dilator, and good control of masseter, pterygoid, and temporal movements. Articulation was distinctly involved because of oro-lingual monoparesis. He could talk, but pronounced certain words indistinctly. He had no difficulty in propositionizing. A focus of strictly cortical yellowish softening was found involving the lower extremities of both central convolutions (Fig. 101), both on their external and Sylvian surfaces, and a soft one, half-inch in diameter, about the middle of the internal portion of the insula. The softening reached into the central fissure, thus taking in a posterior, inferior strip of the second frontal convolution. Its greatest height was one and one-half inches upward from the Sylvian fissure, its width along this fissure one and one-fourth inches. The anterior limit of the lesion was one-fourth of an inch caudad of the presylvian fissure.

FIG. 101.



Softening of face area.

A more recent report locating the facial centre, is by Brissaud.[1] The patient was a woman aged eighty years, with cardiac disease and catarrhal emphysema. She had right-sided hemiplegia and aphasia, which greatly improved. For two years, subsequently, she was found to have right facial paralysis, with some muscular atrophy on the right side, with hyperæsthesia, and with right ptosis and dilatation of the pupil. Her face was absolutely asymmetrical, the mouth deviating to the right, the right nostril was drawn upward, the arch of the nose on this side completely immobile, and the angles of the cheek effaced. The brain showed a focus of yellow softening occupying the inferior quarter of the left postcentral convolution. The gyral conformations presented some peculiarities, the foot of the subfrontal convolution being situated much in advance of the inferior extremity of the Rolandic fissure.

Centre for Retraction of the Angle of the Mouth (Action of the Platysma Myoides.) Ferrier found that electrical irritation of the lower extremity of the postcentral convolution of the monkey caused retraction of the angle of the mouth, stronger excitation causing the head to draw slightly to one side. A similar effect was produced by excitation of the frontal division of the third external or supersylvian convolution of the dog; and, occasionally, the angle of the mouth was retracted on both sides and the ear drawn forward. Similar results were obtained by experiments on jackals. A limited number of clinical cases have corroborated this experimental result, as one case reported by

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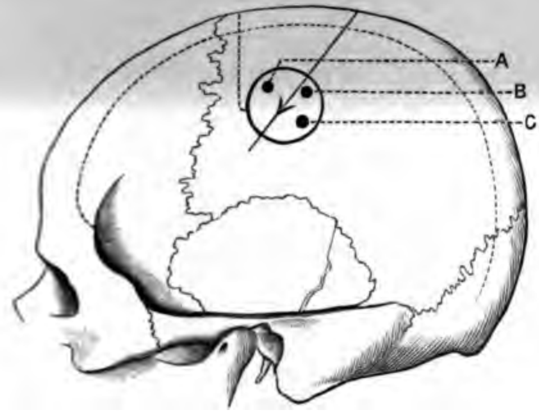
[1] Brissaud : Le Progrès Médical, vol. xix. 2d ed. (S. Paris, Dec. 1893.)

Bramwell[1] of a woman who frequently had convulsions which always began in the right platysma, and often were almost entirely confined to this muscle, and who also had numbness in the thumb and forefinger, followed by paralysis of the right arm and leg. A spicule of bone was found irritating the inferior margin of the postcentral convolution.

Area for the Movements of the Upper Limb. The subdivision of the area for the upper limb is for the shoulder in the upper part, the elbow next below and behind, the wrist next below and in front, the thumb lowest and behind. In the area just above the superfrontal fissure the movements of the lower and upper limb are absolutely blended, most markedly in the hinder sixth of the superfrontal gyre. Sometimes an epileptic fit from a lesion centred here begins by complicated and generalized movements of both extremities on one side. Beevor and Horsley,[2] as one of the results of an elaborate series of experiments by stimulation of the cerebral cortex of the monkey, conclude that "the most intense representation of the movements of the segments of the upper limbs is arranged in a most perfect graduation from below upward. The representation of the thumb occupies the lowest part of the upper-limb area, *i. e.*, opposite the lowest point of the intraparietal sulcus; the fingers have their seat of greatest intensity immediately above that of the thumb; the representation of the wrist is situated just above that for the fingers, while the elbow is represented higher up than the wrist, near the upper limit of the area. The representation of the shoulder forms the upper limit of the upper-limb area, being localized rather higher up than that for the elbow."

FIG. 102.



A. Flexion of thumb, fingers, wrist, elbow, and face movements. B. Shoulder retracted, elbow fixed. C. Flexion of elbow with arm raised; the position of the scar is shown by the triangle.

Some of the experiments and observations made on the human cortex during operations have beautifully isolated the subdivisions of the areas of the upper and lower extremities, and of the face. In a case operated upon for me recently (October, 1894,) by Barton, a trephine opening was made in the position indicated in Fig. 102, and gentle faradization with an antiseptic bipolar electrode at the points indicated in the diagram produced the following results: At A, believed to be just cephalad of the lower part of the middle third of the central fissure, flexion of the thumb, fingers, wrist, and elbow, followed by movements of the face; at B, flexion of the elbow with raising of the arm. At C, retraction of the shoulder and flexion of the elbow. B and C were presumably just caudad of the lower part of the middle third of the precentral convolution.

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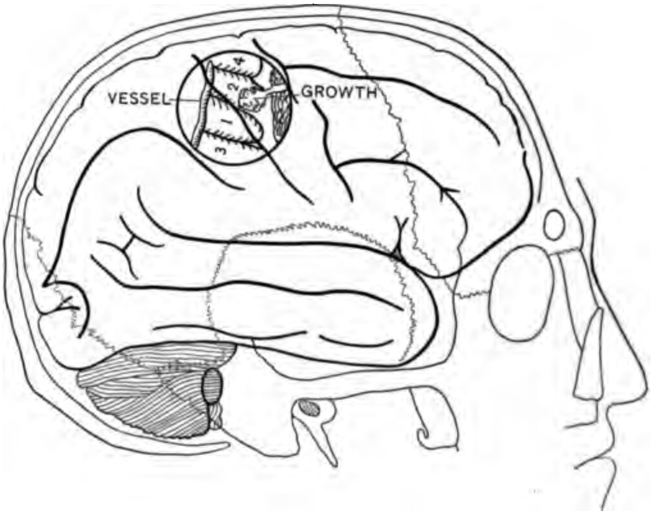
[1] Byrom Bramwell: Brit. Med. Jour., August 28, 1875.

[2] C. E. Beevor and V. Horsley: Phil. Trans. Royal Society, London, vol. clxxix. (1888), B, pp. 205-256.

In one of Keen's[1] operations, on touching the cortex with the electrodes at a position which apparently corresponded to the anterior portion of the central convolution, movements of the wrist and fingers were produced. The hand moved in extension in the mid-line and to the ulnar side at different touches, the fingers being extended and separated. Above the region in which these movements were obtained, application of the current caused movement of the left elbow, both flexion and extension, and of the shoulder, which was raised and abducted. Below the region where the hand movements were excited, the application of the current produced an upward movement of the whole of the left face. In a case trephined by Hearn for the writer, the exact movements described by Keen were produced. These facts of experiments on man would seem to uphold the view that the motor zone, in man at least, is much more extensive in front of than behind the fissure of Rolando. Nancrede, of Philadelphia, before excision of the cortex, fixed the position of the thumb centre by means of the faradic current. The patient suffered from convulsions beginning with strong flexion of the right thumb. The current was applied in the second fourth of the postcentral convolutions, counting from below upward.

FIG. 103.



Centres for the thigh, leg, shoulder, upper arm, elbow, wrist, and hand, as determined by faradization of the human brain.

In another case operated on for me by Keen, the centres for the shoulder and upper arm were fixed with great accuracy, as were also those for the elbow, hand, and wrist. After the removal of the bone and dura a fissure corresponding to the line of the central fissure was seen. The opening was made by a one and one-half inch trephine, the centre pin of which had been inserted in the line of the central fissure, 1.75 inches from the median line. Several spots were selected, as shown in Fig. 103. When a weak faradic current was applied to the spot marked 1, just caudad of the line of the central fissure, at about the juncture of its upper and middle third, the first movement which at once resulted was protraction and moderate adduction of the shouder and upper arm. This was soon followed by a series of jerks, involving the upper, and to a more moderate degree, the lower arm. No differentiated movements of the head and arm, and no face or upper-leg contractions were observed. The left toes and foot, however, slightly flexed coincidently with the shoulder movement. A second application was made at the point marked 2, the effect of which was to again produce decided shoulder and arm movements, with greater adduction and some protraction of the entire arm. The thigh was flexed upon the pelvis at an angle of about 130°, and the leg upon the thigh about the same, with adduction of the thigh and extension of the foot and toes. Excitation at the point marked 3, flexed the elbow, hand, and wrist, with slight shoulder abduction, protraction closely following. Excitation at 4, caused primary movements of abduction and flexion of the thigh upon the pelvis, and the leg upon the thigh at an angle of 135° or 140.° No foot movements or movements of the upper extremities occurred.

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[1] W. W. Keen: Trans. Amer. Surg. Assoc., vol. vi. 1888; also American Journal of the Medical Sciences, November, 1888.

Area for Movements of the Lower Limb. The movements of the lower extremity are represented in the upper portion of the motor area and the adjoining marginal convolutions; probably hip and thigh movements on the lateral and mesal aspects of the hemisphere, well forward in the area, and movements of the legs and toes further back on both the lateral and mesal aspects. Beevor and Horsley[1] described the whole region of representation of the lower limb as a narrow strip running along and forming the upper fifth of the convex outer surface of the excitable region of the hemisphere; further, that the middle of this strip, or, more strictly speaking, the juncture of its middle and posterior thirds, is essentially the seat of representation of the hallux, while each extremity is essentially the seat of representation of the small toes. Horsley[2] has described a case of traumatic epilepsy, the primary movement consisting of flexion of the hallux, followed by gradual flexion of the rest of the lower limb, and that followed by successive invasion of the rest of the lower body in the usual order. A dense and cystic cicatrix was found at the upper end of the precentral gyrus. In another case, in which a tumor was removed and with it the cortex in front of the upper end of the fissure of Rolando, the only permanent complete paralysis of the lower limb was that of the hallux. In one of my cases in which a small gumma involved the upper fourth of the precentral and a smaller segment of the postcentral convolution, the patient had severe attacks of left-sided spasm, beginning with twitchings in the left toe and foot; and she also had partial paralysis of the left leg and arm, most marked in the leg. The leg area, as first shown by Horsley and Schäfer, is also largely situated upon the mesal surface of the hemispheres. According to these authors, the excitation takes effect chiefly upon the ankles and digits, producing most commonly flexion of the foot with flexion of the digits. The most marked movement in front of the upper end of the Rolandic fissure is flexion of the leg at the knee, with the addition, when the electrodes were applied more anteriorly, of flexion at the hip.

Area for Movements of the Trunk. A narrow strip of the anterior portion of the leg area appears both from the results of experimentation and of pathological and surgical observation to be a trunk area, this being larger proportionally on the mesal than on the lateral aspect of the hemisphere, as represented on the diagrams. Horsley and Schäfer occasionally obtained movements of the trunk when the electrodes were applied to the lateral surface near the margin of the hemisphere. On the adjoining mesal surface excitation produced rotation and arching of the lower spine and the pelvis, and extension of the hip, movement of the tail to the opposite side, and flexion at the knee.

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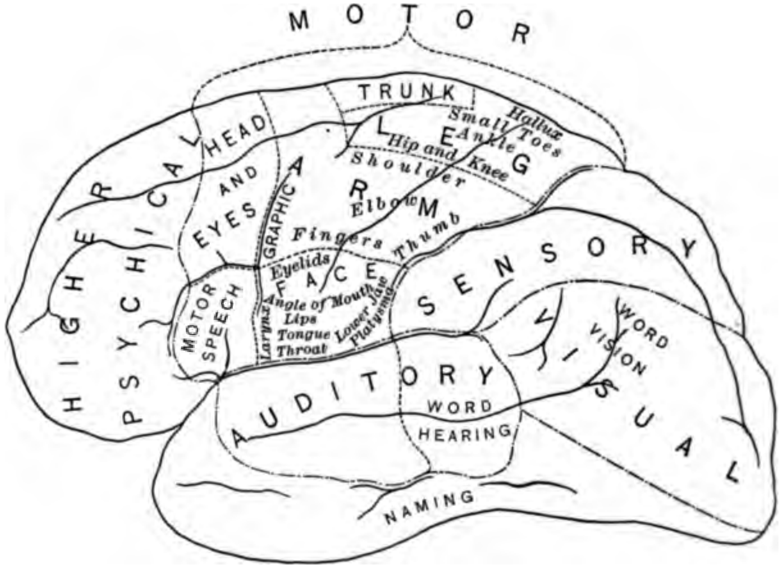
[1] Beevor and Horsley, op. cit.

[2] V. Horsley: American Journal of the Medical Sciences, April, 1887.

FIG. 104.



Cortical centres and areas of representation on the lateral aspect of the hemicerebrum.

The study of motor localization shows that some of the motor areas apparently overlap each other. Ferrier holds that the various centres of representation are isolated from each other, but, according to some authors, this overlapping is real. The views of Horsely[1] are given in the following quotation: "The general result has been to show that there is no hard and fast line limiting the representation of any given segment. I mean to say that there is no area of the cortex, over which any particular segment is equally represented throughout, but that in one spot especially the representation is concentrated and thence diminishes gradually; thus in the representation of the thumb, we find, for example, that there is a focus, but that the thumb is represented over a great deal of the upper-limb region, and that this representation diminishes in intensity gradually as we pass from the focus upward. To give one more example: the shoulder and the thumb form the two great 'purposive' parts of the upper limb, and we find that the shoulder is represented in exactly the opposite part of the upper-limb region to that of the representation of the thumb. I cannot do more now than thus indicate the mode in which we believe that the various segments of the body are represented. I have here a diagram (Fig. 99) showing the various foci of representation of all the small segments of the body. Professor Ferrier states that se areas are limited by hard and fast lines, that no one area trenches upon e other. I cannot follow my honored teacher in this belief. We are perectly sure that there are what may be called border centres, and indeed in e portion of the brain, Professor Ferrier has himself called attention to this

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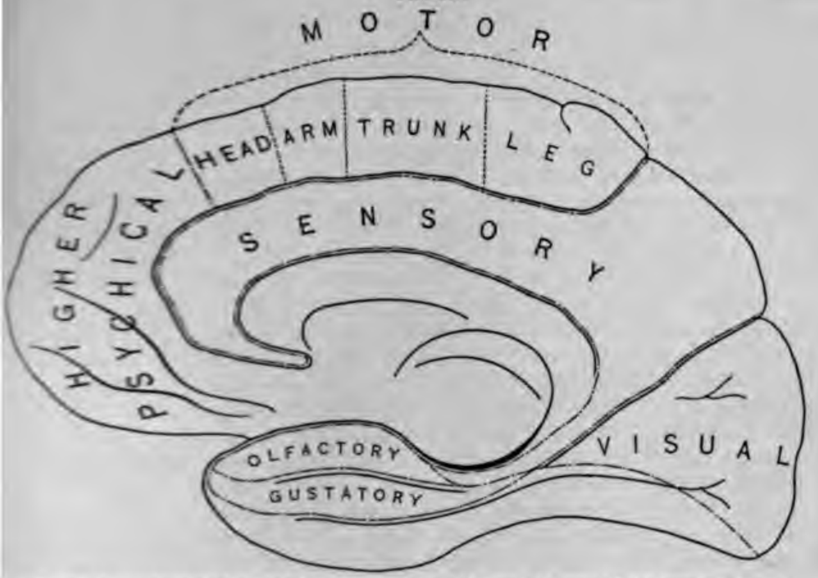
[1] V. Horsley: Trans. Congress Amer. Phys. and Surg., 1888, vol. i. p. 342-345.

FIG. 105.



Cortical centres and areas of representation on the mesal aspect of the hemicerebrum.

ct. There are border centres, and the area of representation of the face erges into that for the representation of the upper limb. If there was a cal lesion at that point you would have the movements of these two parts arting together. You have then two 'signal' symptoms, meaning that you ive two movements of two different segments."

SENSORY LOCALIZATION (Areas for Cutaneous and Muscular Sensations).

The remarks already made regarding the different theories as to motor nd sensorimotor localization indicate that the question of cortical representation of the cutaneous sensations of touch pain and temperature—and other -called senses embraced under the designation "common sensibility"—is ill under discussion. As we have already indicated, it is only necessary for actical purposes to admit that something occurs in each separate area of e cortex which gives rise to manifestations which must be referred to this rt. In order that a certain spot in the brain shall constitute a centre it is ly necessary, in the neurologist's sense, that its abolition shall cause certain enomena to disappear, and its excitation shall cause certain phenomena to present or to be intensified.

In 1888 I declared that the conclusion was warranted that there was in the, rebrum a region for general sensation, including touch, pain, temperature d possibly the senses of pressure and of the location of a limb, which could divided into special subareas for the various distinct portions of the body, and that these regions lay alongside of and had close relation with corresponding motor areas, but that they were not identical with them; also that no part of the brain was more likely to contain these differentiated areas for sensation than the gyrus fornicatus, the hippocampal gyre, the precuneus, and the postparietal convolutions. I see no reason to change the conclusions expressed in this paragraph, but the use of the term "centres" may, as just pointed out, be misleading in this connection. Too much stress is laid upon *motor cells* and *sensory cells*. The great function of all cells is trophic. Cells in sensory areas do not feel, nor do they originate sensation; neither do motor cells spontaneously generate motion. They are simply bodies placed in the great sensorimotor arc to administer to the nutritive functions of the fibres which convey sensory and motor impressions. In a certain practical sense we have sensory cells and motor cells, the former being small and having short neurons, the latter large with long axis-cylinder processes; but these cells do not fundamentally differ. They vary in size according to the length and bulk of the fibres which they nourish.

The great sensory tract or path is constituted of nerve cells and fibres which originate in the dorsal spinal ganglia and their cranial homologues. These ganglia send out prolongations which become the true peripheral nerve fibres and endings, and shorter processes which penetrate the spinal cord, and there bifurcate, a branch descending and another ascending. Some conflict still exists as to the course of sensory fibres in the spinal cord. Ferrier[1] holds that the evidence is in favor of this view—that all of the sensory paths pass up the opposite side of the spinal cord, and that they are not contained either in the postero-median column or in the direct cerebellar tract, or in the antero-lateral tract; and as the pyramidal tract may be entirely sclerosed without any affection of sensation—he is led by a process of exclusion to suppose that the sensory tracts ascend in immediate relation with the gray central matter. The view which at present has the most support is that decussations of the fibres which constitute the great sensory pathway take place both in the cord and oblongata, (1) by crossings in the spinal commissures at all levels, of fibres which convey pain and temperature impulses, which ascend in Gowers's antero-lateral tract of the opposite side; and (2) by crossings in block or mass of the tactile sensory tracts of the columns of Goll and Burdach, just above the cuneate and clavate nuclei at the decussations of the fillet, or upper sensory decussation. From this level all the fasciculi for common sensation pass cephalad together. According to this view the columns of Goll and Burdach constitute a direct path, conveying to the clavate and cuneate nuclei, by long axis-cylinder processes, the tactile impressions received from the same side of the body. The cells for these columns are chiefly tautomeral—cells whose axis cylinders pass into tracts on the side on which they originate. The antero-lateral tract of Gowers is the chief path for pain and temperature impressions, largely constituted by processes of heteromeral cells, whose neurons pass by the way of the anterior commissure to the white substance of the opposite side. These views have not yet received full acceptance. One of the strongest arguments in their favor is furnished by syringomyelia. In this disease destruction of the spinal decussations accounts for the impairment or abolition of the pain and temperature senses, the tactile sense remaining intact in most cases, because Goll's columns have not been invaded.

However views may differ with reference to the spinal sensory paths and the cortical representation of cutaneous sensations, they are in general accord with regard to the course of the great sensory tract in the fillet, or lemniscus

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[1] David Ferrier: The Croonian Lectures on Cerebral Localization, London, 1890, p. 90.

—in the oblongata, pons, and mid-brain—and its continuation in the posterior third of the posterior limb of the internal capsule.

As the sensory paths are differentiated up to the internal capsule, it is not probable that they at once begin to blend with the motor and other fibres of the alba immediately after emerging from the capsule. On the whole, the weight of evidence is in favor of a separation of the regions especially related to common sensibility from the motor and other areas of the brain. My own views on this subject have not changed, but have undergone some modification as to the method of expressing the facts regarding this localization. Indisputable facts of experiment and pathology show that the callosal gyre (gyrus fornicatus) and hypocampal region, and probably also the postero-parietal convolutions, are in some way especially concerned with the representation of sensation, or with some cerebral process of a sensory character in the great sensorimotor arc.

After sensory fibres have passed the internal capsule they soon begin to spread outward, but still hold an intermediate place between the motor areas and the areas for the special senses. As the sensory tract approaches the cortex some fibres pass to neighboring regions, but most of these fibres reach the postero-parietal region and the falciform lobe before sending their branches to other zones. *Sensory cells*—that is, cells solely concerned with nourishing fibres which are still responding by excitations which pertain to the afferent side of the nervous system—are found in the limbic lobe and post-parietal region, but their extensions (processes) must form various thicknesses and complications of fibres and cells all over the cerebrum.

Cases have been reported by Demeaux, by F. Müller, by the writer, and others in which autopsies have shown hemianæsthesia from lesion of the hinder third of the internal capsule or adjoining portions of the corona radiata. Veyssiere produced hemianæsthesia on the opposite side of the body by dividing this portion of the capsule in animals. The fibres of the sensory tract are probably separated from the motor in the centrum ovale until the cortex is reached or nearly reached. According to Flechsig, the third set of fibres of the projection system included those which lie just posterior to the motor tract, and which pass inward from the parietal convolutions, and these take a similar course to the motor tract, and fill up to a considerable extent the space between it and the radiation of the visual tract, toward the occipital lobe.

Ferrier in his earlier experiments found that lesions involving the horn of ammon and the hippocampal convolution caused impairment or abolition of tactile sensibility on the opposite side of the body, and located the cortical centres for this form of sensibility in the hippocampal region. In experiments on monkeys, with Professor Yeo, he established that tactile sensibility was in every case temporarily impaired or abolished, according to the amount of destruction of the hippocampal and temporo-sphenoidal regions.

Horsley and Schäfer found that hemianæsthesia, partial or complete and contralateral, resulted from destructive lesions of the limbic lobe. They found that any extensive lesion of the gyrus fornicatus was followed by hemianæsthesia more or less marked or persistent; sometimes the loss of sensation involved almost the whole of the opposite side of the body; sometimes it was localized either to the upper or lower limb, or to a particular part of the trunk. They did not, however, succeed in establishing the relations between special regions of the body and the parts of the convolutions which had been destroyed. Their experiments were frequently, but by no means in every case, complicated by the presence of a certain amount of motor paralysis, chiefly, if not entirely, affecting the muscles of the leg, and they believe that this condition was always due to a lesion (accidentally produced during the operation or subsequently as the result of interference with the circulation) in the leg area of the marginal convolution.

Some light has been thrown on the disputed question of the existence of sensory centres in the motor cortex by careful examinations of patients after operations, particularly when definitely determined gyral areas have been cleanly excised. In a case of Lloyd and Deaver, in which the facial and arm centres were carefully excised, I, on several occasions, with Dr. Lloyd, tested the conditions as to motor power and sensibility and the reflexes in this patient, with the results which have been reported by Dr. Lloyd. The patient blindfolded could instantly recognize the slightest touch on all points on the affected side; even light breathing upon his hand was at once detected; pain and temperature sense were normal, and he could discriminate between weights. If objects were placed in his paretic hand he often failed to recognize what they were, but apparently because he was not able to grasp and run his fingers over them and take in their form and bulk.

Bechterew[1], who believes in separate sensory areas, maintains that the loss of sensation in animals that have had the motor area of the convolutions destroyed is apparent and not real; that they cannot withdraw the irritated extremity, though they feel the pain, because they have not the control of the muscles. He also considers the loss of the muscle sense only apparent, because if the animal's paw be placed in an uncomfortable position its failure to be removed is due rather to the motor inability than to impaired muscle sense.

Starr,[2] from a series of American cases of cortical lesion of the brain, and also from a study of the sensory tracts, concluded that the various sensory areas lie about and coincide to some extent with the various motor areas for similar parts; that, in other words, the Rolandic region is a sensorimotor region, the sensory area, however, including to some extent the gyres of the adjacent postero-parietal lobe. Collections of cases such as these cannot, however, overcome positive evidence of decided destructive lesions of the cortical motor centres, without any disturbance of touch, pain, or temperature, or even of muscular sense.

Dana[3] collected 142 cases, including four personal observations, and concluded that the clinical and pathological evidence collected by him showed that the motor areas of the cortex contained also the representation for cutaneous sensations.

Both Ransom[4] and Dana,[5] in advocacy of the view that the Rolandic areas of the brain are centres for sensation as well as motion, have recently reported cases of epilepsy in which the brain was exposed through an opening made to relieve the epilepsy, and needle electrodes were stuck through a cocainized dura to the depth of half an inch; and in both cases subjective anæsthesia of the limb corresponding to the area experimented upon resulted. It is only necessary to recall a criticism already made on one of these cases, namely, that the subjective symptoms of an epileptic, confessedly of weakened intellect, are not sufficiently reliable to justify any conclusions whatever. *Subjective* perversion of sensation means nothing for the determination of this question. Albertoni and Brigatti report a case of epilepsy due to a tumor, which was removed from the mid-Rolandic region by operation. The epilepsy ceased as a result, and the motor paralysis greatly improved, but tactile, thermal, painful, and muscular sensations, which before the operation were intact, were afterward affected in an appreciable degree

[1] Bechterew: Neurol. Centralbl., Leipzig, 1883, ii. pp. 409-414.
[2] M. Allen Starr: Amer. Journ. Med. Sci., Phila., 1884, N. S., lxxxvii. pp. 366-391.
[3] Dana: Journ. Nerv. and Mental Dis., vol. xiii. No. 9, September, 1888, p. 650-681.
[4] Ransom: Brain, vol. xv. 1892, p. 437. [5] Dana: Medical Record, May 13, 1893.

over the entire opposite half of the body. This case, however, proves nothing conclusively, because of the large mass of brain substance removed, which probably included the cortical sensory tracts.

The cases reported by Savill[1] are confirmatory of the doctrine of separate sensory localization. The first was a case of anæsthesia and trophic changes consequent on lesions of the gyrus fornicatus. The lesion was strictly localized, and was situated precisely beneath the parts of the cortex corresponding to the callosal gyre (gyrus fornicatus), and part of the marginal convolution in their entire extent, and also beneath the anterior half of the quadrate lobule (precuneus). The cavity stretched from before backward over the roof the right lateral ventricle, from which it was separated in places only by the ependyma, without nerve tissue. Careful details are given of the autopsy and subsequent examination. This patient had completely lost the power of perceiving when the left side of the body was touched or pricked seven days after the hemorrhage which Savill believes cut off all communication between the right gyrus fornicatus, marginal convolution, and the anterior part of the quadrate lobule with the part beneath. Sensation was also imperfect in portions of the right arm, right leg, and trunk, which Savill believed was due to pressure on the corresponding convolutions of the left hemisphere. Other important symptoms were present.

Savill concludes, (1) that the case supports the experimental evidence that the gyrus fornicatus is the centre for common sensation on the opposite side of the body; (2) that this loss of sensation in man may be quickly recovered from, presumably by a process of substitution of centres, the other hemisphere taking on the functions of the parts destroyed; (3) that a destructive lesion of the gyrus fornicatus may produce loss of sensation without loss of voluntary motion, at any rate to any serious extent; (4) that a destructive lesion in the same position is also attended by vasomotor or trophic changes of a more permanent character than the sensory changes in the skin and subcutaneous tissues on the opposite side of the body; and, therefore, these convolutions may possibly be centres not only for sensation, but also for trophic influences transmitted to the opposite side of the body.

Savill's second case seems to indicate that the posterior part of the gyrus fornicatus is the cerebral centre for tactile sensation of the arm. The autopsy in this case revealed a tumor with a zone of softening, which corresponded to the paracentral and quadrate lobules, and the posterior third of the gyrus fornicatus, also projecting into the posterior and superior wall of the lateral ventricle. It did not appear on the vertex of the brain, but the posterior third of the hemisphere was congested. "The diffuse nature of the anterior and upper parts of the tumor prevented one from saying very precisely which of the white fibres of the corona radiata were destroyed or involved; but it was quite certain that the gray matter of the posterior third of the gyrus fornicatus, the greater part of the quadrate lobule, and the posterior extremity of the marginal convolution, was considerably disorganized, and its communication with the parts below cut off. Indeed the tumor fungated out on the median aspect of the first-named convolution, which was the chief one involved.

Savill refers to a case of hemianæsthesia with other symptoms published by Dr. Sharkey,[2] and believes that the extent of the lesion, judging from the illustration, was such as to involve the fibres coming from the gray matter on the median aspect of the hemisphere.

Luys, largely from anatomical studies, made the thalamus a centre of sensibility, subdividing it into four special centres, olfactory, optic, acoustic, and general sensibility. Ferrier regards the thalamus as in some way connected with sensation, but believes that differentiated areas for sensation also exist in the cortex. The experiments of Monakow led to the conclusion that different portions of the thalamus are related to cortical areas. Fournier believes that sensory fibres terminate in the thalamus. Crichton-Browne regards this ganglion as a great centre of general sensibility; but Flourens, Longet, Schiff, and Tamburini, among others, have assigned to it motor functions. The weight of evidence, both from experiment and disease, connects the thalamus with various forms of sensation, but it is not the sole cerebral region for sensibility.

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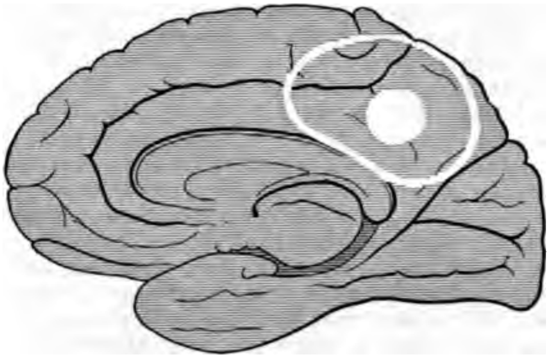
[1] Savill: Brain, vol. xiv. 1891, p. 274; and vol. xv. 1892, p. 450.
[2] Sharkey: Med. Chir. Trans., 1884, p. 265.

FIG. 106.



Savill's case of tumor with zone of softening, invading the paracentral and quadrate lobules, and the posterior third of the gyrus fornicatus. The white central spot indicates the position where the tumor protruded from the surface. The white line around indicates a zone of softening and congestion which projected above the level of the median surface.

VISUAL LOCALIZATION.

The visual area is represented in the two diagrams as taking in all the occipital lobe, and, on the lateral aspect, adjoining portions of both the temporal and parietal lobes, including the so-called angular gyre. As the motor zone has been subdivided into areas of representation, not only for the leg, trunk, and arm, face, speech, etc., but also into areas or centres for parts of the leg, arm, face, and speech, so efforts partly successful are now being made to subdivide the visual zone. The retina, so far as its connection with the central cortex is concerned, can be subdivided into segments probably of a somewhat irregular shape. At first, studies in hemiopia and hemianopsia seemed to show that the only definite connection was between halves of the retinæ and cortical centres, but the latest observations indicate that quadrants and probably even smaller portions of the retinal expansion are related to separate areas of the brain. The macular region certainly has its special cortical centre.

Ferrier has modified his earlier views in so far that he no longer localizes the visual centres in the angular gyre to the exclusion of the occipital lobe, but he believes now that the visual centres embrace not only the angular gyre, but also the occipital lobes, which together he terms the occipito-angular region. He believes that the angular gyre maintains relations with the retinal area of clear central vision, and especially with the macula luteæ.

From a long series of experiments upon the monkey's brain in which he was engaged with Horsley during more than two years, Schäfer found that extensive lesions, both of the occipital lobe and of the temporal lobe, were invariably followed by visual disturbances, taking the form, when the operation was confined to one side of the brain, of bilateral homonymous hemianopsia; but in nearly every case the hemianopsia was merely temporary. The most marked results of this kind were obtained when the occipital lobes were the seat of operation, extensive unilateral lesions producing amblyopia; but in neither case were the symptoms permanent. In conjunction with Sanger-Brown, Schäfer also experimented upon the angular gyre and the occipital lobes. Destroying one angular gyrus as completely as possible with the actual cautery, they could discover no defect of vision, no loss of the movement of the eye or eyelids, and no anæsthesia of the corneal conjunctiva. A week later the angular gyre of the opposite side was destroyed, also with negative results.

With reference to visual localization, the importance of confirming physiological experimentation by careful clinico-pathological observations cannot be overestimated; these observations are even here of more importance than in motor localization.

A few well-reported cases of hemianopsia with autopsies by Jastrowitz,[1] Haab,[2] Hueguenin,[3] Monakow,[4] Seguin,[5] Hun,[6] Féré, Keen and Thompson settled beyond doubt the connection of the cuneus.

Seguin collected forty cases with autopsies, and five traumatic cases without autopsies. Eleven of these were cases of hemianopsia due to lesions of the white substance of the occipital lobe. Sixteen were cases of cortical lesion, or of lesion limited to the cortex and the white substance immediately subjacent; and four of the sixteen (those of Haab, Hueguenin, Féré, and Seguin, referred to above) are what might be termed conclusive cases as to the question of the location of at least a portion of the cortical visual centre in man, as in them the lesion was circumscribed and occupied nearly the same place in the occipital lobe. In one of his tables Seguin has included five cases of traumatic hemianopsia due to injuries of the occipital region of the skull and lesion of the subjacent brain. One of the most important cases which has been recorded since the paper of Seguin is that of Hun, in which a defect in the fields of vision involving the lower left quadrant of each eye occurred with atrophy of the lower half of the right cuneus.

I[7] have recorded an account of the brain of a man who had been blind more than twenty-five years—how much longer could not be positively ascertained. Both occipital lobes were unquestionably small. The cuneus on each side was small, the first occipital convolution of Ecker (superior external *pli de passage* of Gratiolet and paroccipital of Wilder) showing lack or arrest of development. The second and third occipital convolutions of Ecker, especially on the left, presented a dwindled appearance. In another brain, that of an old woman, blind for at least thirty years, similar gross appearances of arrested development in the occipital region were present.

One of the latest reviews on the subject of visual localization is by Henschen.[8] His conclusions are based on observations of lesions of the visual paths and centres, with autopsies, and negative cases with absence of visual defects with lesions in their neighborhood, he having collected about 160 cases with post-mortems. He divides the occipital path into a frontal, middle, and occipital portion. The frontal includes the optic nerves, chiasm, and tracts; the middle is mainly the pregeniculum or external geniculate body; the occipital path is in the occipital lobe. Nothnagel is quoted as believing that lesions of the gemina need not give rise to visual disturbances. Henschen holds that destruction of the pulvinar is not always accompanied with hemianopsia; also, that a lesion of the posterior portion of the internal capsule does not cause hemianopsia. A recent observation of my own is conclusive in the affirmative to both of these propositions. In a case of large lesion of the thalamus and adjoining internal capsule, involving the pulvinar, but not the external geniculate body, hemianopsia was not present. The middle path of the optic fibres and their connection with the central ganglia has not, however, been thoroughly investigated, and it is necessary, as Henschen points out, to make a distinction between visual fibres, lesions of which produce defects in the visual field, and optic fibres which are concerned with reflexes. With reference to the occipital subdivision of the visual path an analysis of all cases shows that it is situated in the ventral portion of the optic radiation, and there forms a bundle less than a centimetre thick, which lies at the level of the second temporal gyrus and second temporal sulcus. The fibres for the dorsal retinal quadrant lie dorsally; and for the ventral retinal quadrant ventrally; the macular fibres have a more median situation.

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[1] Jastrowitz: Centralbl. für Prakt. Augenheilk. vol. i. Dec., 1879, p. 254.

[2] Haab: Klinischer Monatsblatter f. Augenheilk, xx. 141, 1882.

[3] Hueguenin, ibid.

[4] Monakow: Archiv. f. Psychiat. u. Nervenkrankheiten, vol. xvi. S 166.

[5] Seguin: Journ. Nerv. and Ment. Dis., vol. xiii. No. 1, Jan., 1886, 1-38.

[6] Hun: American Journal of the Medical Sciences, vol. xciii. Jan., 1887, 140-160.

[7] Mills: Univ. Med. Mag., Nov., 1889, vol. ii. p. 69.

[8] Henschen: Brain, Parts, lxi and lxii, 1893, vol. xvi. p. 170.

As to the exact position of the retinal centres for quadrant or half vision, Henschen is very definite. He holds that a lesion on the mesal surface causes hemianopsia only if the cortex of the calcarine fissure or the fibres derived from it are affected; also, that a lesion limited to the calcarine cortex can induce a complete hemianopsia, in proof of which he regards one of his own cases as most instructive. It was stationary, uncomplicated, and the clinical examination, as well as the post-mortem, was accurate. The lesion was limited to the cortex in the depth of the calcarine fissure, and the hemianopsia was complete and absolute.

Henschen believes the elements of both retinal halves are represented in the calcarine cortex by different cells, which lie beside each other. After destruction of both eyes in a patient suffering from leprosy, complete atrophy of the calcarine cortex ensued, but in a case of destruction of one eye only he found in that situation a number of pigment-charged cells alongside of perfectly normal ones. With reference to the position of the macular field Henschen believes it lies in the calcarine cortex, but with this I do not agree. According to Wilbrand, the macula is innervated by both hemispheres in a variable manner.

Vialet[1] has published a monograph on the cortical centres for vision, in which he reviews the work of previous writers and contributes a valuable microscopical research of several cases previously recorded by Dejerine. His conclusion is that the cortical centre for vision occupies the entire internal face of the occipital lobe. It is limited in front by the internal perpendicular (parieto-occiptal fissure), above by the superior border of the hemisphere, below by the inferior border of the third occipital convolution, and behind by the occipital pole. He believes that the calcarine fissure plays an important special part in this localization; that it certainly forms the centre of the visual sphere in man. Wilbrand has advanced the hypothesis of the existence of three distinct centres—for space, for light, and for color, believing that these centres or areas are superimposed in the visual zone. Vialet does not believe in the existence of these secondary centres, and the question can be regarded as still unsettled.

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[1] Vialet: Les Centres Cérébraux de la Vision et l'Appareil, 8vo. pp. 355, Paris, 1893.

AUDITORY LOCALIZATION.

The localization of the auditory sphere in the superior temporal gyres, clearly established by the experimental work of Ferrier, can be regarded as settled, although this view is still antagonized. The most important physiological experiments which seem to contradict this position are those of Schäfer and Sanger Brown, who in six monkeys more or less completely destroyed the supertemporal convolution on both sides, and claimed that in all hearing was not only not abolished, but was not permanently affected. In one the whole temporal lobe of one side was removed, and yet Schäfer holds that the animal responded to all impressions of all senses, but appeared to understand very imperfectly their meaning. Sounds even slight in intensity were heard. He believes that this case militated strongly against the view that auditory perception is localized in the temporal lobes. Ferrier,[1] however, was not satisfied that the evidence of the existence of auditory perception in these animals was unequivocal. His previous experiments had all pointed in another direction, but as an unaccountable discrepancy seemed to exist between Schäfer's results and those of Yeo and himself, he determined to reinvestigate the question. In one monkey on which he performed the operation of bilateral extirpation of the superior temporal gyre, with one month interval between the operations, the result was very striking. At first the animal failed absolutely to respond to any of the tests which invariably attracted the attention of normal monkeys. To the last, with the single exception of the door of the laboratory, from which it was always expecting something, it never realized the origin of sounds; it was altogether indifferent to sounds which formerly were full of significance for it, and all that could be said was that it was not insensible to sonorous vibrations, According to Munk, the auditory area is at a position corresponding in the monkey and in man to about the meditemporal gyre, considerably lower in the temporal lobe than Ferrier's localization. In the posterior portion of the supertemporal convolution, to which Ferrier gives the preference as the location of the auditory centre, Munk places the area for sensibility of the auricle, while Luciani includes in the auditory sphere the parts which correspond to the upper temporal gyres, and also a large part of the cortex which is concerned with other functions.

Confirmation of the localization of the auditory centre in the supertemporal gyre is afforded by cases of auditory discharges, or subjective auditory sensations in connection with irritative lesions implicating these gyri, of which a few illustrations have been recorded.

A few cases of prolonged deafness, with autopsies, have assisted in determining the position of the cerebral centres for hearing. I have recorded the case of a man deaf for thirty years,[2] whose brain showed atrophy of both supertemporal convolutions, and particularly of the left, and Broadbent reported a similar case. Manouvrier[3] has left a valuable contribution to the subject in his description of the brain of Bertillon, the statistician. This description was first published in the *Bullétin d'Anthropologie*, of Paris, in 1878, after which the author accidentally learned that Bertillon was so deaf in the left ear as to be incapable of hearing what was said to him on his left side. This deafness he had suffered since infancy. In the description of the brain attention has been drawn to the large size of the left supertemporal convolution as compared with the right; the difference both in size and in the amount of convolution present was remarkable, and much too great to be accidental, a fact which goes far to render it probable that in man hearing on one side is subserved by the supertemporal gyre of the opposite side, as Ferrier has shown to be the case in apes. The part of Broca's convolution contiguous to the supertemporal gyre was much better developed on the right than on the left. As Bertillon was left-handed in his youth, it is suggested that, so far as speech is concerned, he was right-brained, and the difference in the development of the auditory and speech centres on the right side is offered as an explanation of the difficulty which he found in expressing himself orally. The angular gyre was much more developed on the right side of the brain than on the left, and the explanation is offered that there was a hypertrophy of the part subserving the visual sense on that side to compensate for the auditory deficiency. The postcentral gyre on the left side was found to have quite an extraordinary size, which the author accounts for by the close connection which must exist between the sensorimotor incitations connected with the limbs and the sensory phenomena of auditory origin.

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[1] Ferrier: Brain, April, 1888, and Croonian Lectures, p. 80.

[2] Mills: Univ. Med. Mag., November, 1889, vol. ii. p. 69.

[3] Manouvrier: Bullétin de la Société de Psychologie Physiologique. T. 5. Abstract by James Taylor in Brain, 1890, vol. xiii. p. 429.

In order to have complete cerebral deafness, it is necessary that the lesions should be bilateral. Such bilateral lesions affecting the supertemporal gyres of both sides of the brain, and causing deafness in persons without peripheral disease, would afford conclusive evidence of the position of the auditory centres in man. Ferrier speaks of the extreme rarity of bilateral lesions of the superior temporal convolutions, but gives two important cases in which these double lesions simultaneously or successively occurred. One case, first recorded by Shaw,[1] is that of a woman who suddenly lost power in the right arm, with also loss of speech and word-deafness. The loss of power passed away, but she became incoherent and subject to delusions, and on testing her she was found to be deaf and blind. The post-mortem showed atrophy of both the angular and the two superior convolutions of both hemispheres. The other was a case of Wernicke and Friedlander,[2] being a woman who, after apoplexy, had right hemiplegia, aphasia, paraphasia, and word-deafness. A few months later she had a second attack, causing paresis of the left arm, and she became absolutely deaf. Extensive lesions were found in the superior temporal convolutions of both hemispheres.

A case has been reported by the writer,[3] which should be ranked as conclusive in the decision of this question of auditory localization. The specimens showed an isolated lesion in the two upper temporal gyres of the left hemisphere, which caused word-deafness, and also a later lesion of the two superior temporals of the other hemisphere, with a history of total deafness. The patient, a woman of forty-six years, fifteen years before her death had an apoplectic attack, which left her word-deaf, but not paralyzed. Prior to this first attack of apoplexy her hearing had been good, but after it she could not, by hearing, understand anything that was said to her. She could, however, hear music and sounds of various kinds; for instance, when an organ or a band had performed upon the street she at times called attention to the fact; and she had also come down from the second, and even from the third story to open the front door in answer to a knock. She could hear such sounds as a bell ringing or a clock ticking. These facts were elicited from her relatives through various statements made by them, chiefly spontaneously. When any one wished to communicate with her it was done by means of writing or signs, as she had fully preserved her vision, and was evidently not word-blind either for writing or printing. She often read the newspapers, and could do so with intelligence up to a few weeks before her death. Her sister-in-law said that several times she had heard her try to read the newspaper aloud, and in so doing she had seemed to understand what she read, "but made a tangle of her words." From the time of the first attack she had never been able to speak well, her words being "jumbled" or "tangled." From the description given of her manner of speech, the defect was evidently a serious form of paraphasia and paralexia. Her relatives spoke positively of her deafness as having been due to "stroke;" but the apoplectic attack, although it had at once caused this word-deafness and paraphasia, had not in any way, so far as could be ascertained, affected either motion or sensation. She could write, but "sometimes mixed up her words in writing." Nine years before her death she had another and more severe apoplexy, and her deafness increased for sounds, as well as for words, until it was almost total. The seizure left her with partial left hemiplegia, chiefly affecting the arm, and in this extremity, from the description, the paralysis was more marked below the elbow.

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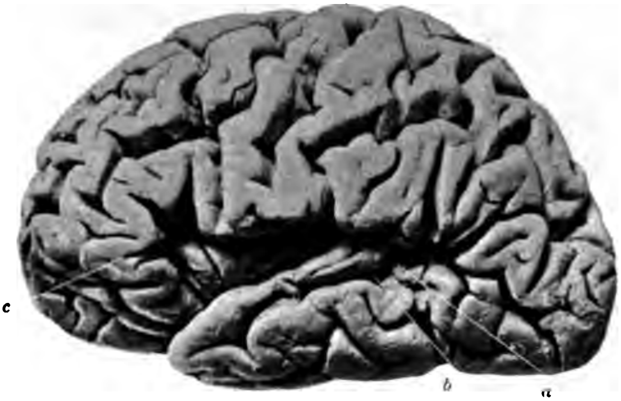
[1] Shaw: Archives of Medicine, February, 1882.

[2] Wernicke and Friedlander: Fortschritte der Medicin, Bd. I., No. 6, March 15, 1883; Brain, April, 1888, p. 19.

[3] Mills: Univ. Med. Mag., 1891, vol. iv. p. 105.

At the autopsy the left supertemporal convolution was found to be much smaller and thinner than usual, and at the posterior extremity of it and of the second temporal was a depression, covering a space about seven-eighths of an inch in diameter. The posterior two-thirds of the supertemporal convolution had shrunken to a thin strip. At a position corresponding to the posterior fourth of the second temporal convolution and the parallel fissure the brain presented a marked depression or cavity, at the bottom of which, when the specimen was in a fresh state, was a mass of yellow, shrivelled, puckered tissue. This was evidently the remains of an old embolic softening (Fig. 107). The second temporal (meditemporal) convolution was decidedly atrophied, and in its posterior fourth, or perhaps third, it had practically disappeared, and had been replaced by the cavity or cyst just described. The third, fourth, and fifth temporal convolutions were undoubtedly not involved. Around the ascending branch of the Sylvian fissure, and at the bases of the two central convolutions, much atrophy had evidently taken place. The

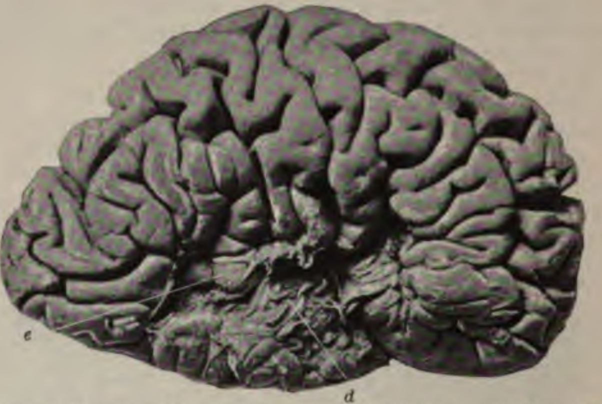
FIG. 107.



Lesion of the superior temporal convolutions. *a*. Cyst or cavity, the result of an old embolism. *b*. Atrophied first temporal, continuous with retroinsular convolution. *c*. Ascending Sylvian fissure.

hinder portion of the third frontal, and particularly the strip of convolution between the ascending and Sylvian, and precentral fissures, were markedly wasted. The posterior of the two retroinsular gyres was very small. The anterior retroinsular presented the appearance of being a continuation of the anterior half of the first temporal, and the posterior retroinsular was continuous with the posterior, much-shrunken half of the first temporal. In the right hemisphere an old and very extensive hemorrhagic cyst had completely destroyed the first, and almost completely the second, temporal gyre, the insula, the retroinsular gyres, the lower extremities of the central gyres, and a large extent—but exactly how much could not be determined—of the ganglia and capsules (Fig. 108). Examination from within showed that the caudatum and thalamus were largely preserved, and that the chief interior destruction was probably of the lenticula and the external capsule. A study of this case justifies the conclusion that the centre for word-hearing is situated in the hinder thirds of the first and second temporal convolutions, and that the third, fourth, and fifth temporal convolutions take no part in word-hearing. A lesion confined to the posterior thirds of the first and second temporal convolutions of the left hemisphere will produce complete, or almost complete, word-deafness, the corresponding regions of the other hemisphere remaining intact; but the field or sphere for all auditory memories covers a much larger cortical area than that for word-hearing, including at least the posterior two-thirds of the first and second temporal convolutions. The auditory field, and special auditory centres, have their highest development in the left hemisphere, but destruction of the auditory areas of the upper temporal convolutions of both hemispheres is necessary to complete brain-deafness.

FIG. 108.



Lesion of the superior temporal convolutions. *d.* Hemorrhagic cyst, destroying two upper temporal convolutions and other parts. *e.* Depressed lower extremity of inferior parietal convolution.

Ferguson[1] has reported the case of a young man who for eight years had suffered with chronic otitis media in the right ear. The hearing on this side toward the last was lost, when the vibration proceeded from without the ear, but was slightly retained to vibrations conducted through the solid media of the bones. For two years prior to the death of the patient there were symptoms of cerebral tumor situated in the right temporal region, such as convulsive movements with auditory aura, both being referred to the left side. Hearing on the left side became gradually lost, and for at least six months was entirely gone, though the auditory auræ were still present. The post-mortem revealed a large tumor in the first and second temporal convolutions on the right side, destroying the first entirely and the second slightly.

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[1] John Ferguson : Journ. Anat, and Physiol., vol. xxv. N. S., vol. v. London and Edinburgh, 1891, p. 292.

THE NAMING CENTRE.

An area which includes portions of the third and fourth temporal convolutions has been designated in the diagram (Fig. 104) as the naming centre. The setting apart of a special area with this designation is in accordance with the views of Broadbent, Kussmaul, and Charcot. According to Broadbent, the formation of an idea of any external object is the combination of the evidence respecting it received through all the senses; and for the employment of this idea in intellectual operations it must be associated with and symbolized by a name. The structural arrangement connected with this process he supposes to consist in the convergence from all the receptive centres of tracts which go to a convolutional area on the sensory side of the nervous system, which may be called the *naming centre.* Its correlative motor centre is the propositionizing centre, in which names or nouns are set in framework for outward expression, and in which a proposition is realized in consciousness or mentally rehearsed. The destruction of this centre would cause loss of the memory of names or nouns. As a provisional guess Broadbent placed this centre in an unnamed lobule situated on the under surface of the temporal lobe, near its junction with the occipital lobe, where he believed fibres from all the perceptive centres converge to and end in the cortex of this region. A careful study of the entire subject of speech disturbances, including an analysis of cases already reported, will be convincing as to the necessity of a higher area for thought and speech, intermediate between the sensory or receptive centres, and the motor or emissive[1].

Other names which have been applied to this centre or area are *idea centre* and *concept centre.* Some authorities, as Ross[1] and Bastian, consider that it is not necessary to have a particular region of the brain in which concepts are elaborated and symbolized by name; but even Ross and Bastian acknowledge a special development of the cortex for concepts and names, but would not restrict it to an isolated area.

Recently a case which seems to be convincing as to the existence and location of this naming or idea centre has fallen under my own observation, one which would seem not only to support the separate localization of such a region, but also to confirm Broadbent's speculation, made long ago, as to the exact position of this centre in the temporal lobe.

This patient was seen by me in consultation with Dr. Wilson Bowers, and an account of the case was recently presented to the Philadelphia Neurological Society by Dr. J. W. McConnell and the writer. The following is a condensed history of the case and of the most interesting autopsy:

M. R. W., white, married, aged about forty years, five years before her death, for the first time complained of numbness of the back of the neck and vertigo, and had an attack of excessive vomiting, lasting two days. Nothing occurred subsequently to this, or nothing could be learned of anything occurring again, until three years later, when it was noticed that she did things differently from her usual custom. Dressmaking, at which she had been successful, was poorly done. She hung upside down a certificate of membership in a society to which she belonged without realizing the error. Her appearance during the ensuing year also changed from that of a woman in her prime to one fast advancing in years; but nothing especial occurred until the morning of December 31, 1893, when she acted very strangely, was very nervous, imagined that she saw a light, could not read, and remarked that she felt "like killing her daughter." In the evening of that day she was seized with a convulsion, frothed at the mouth, and was unconscious. The next day she was very forgetful, but without apparent speech trouble. She was confined to bed for two weeks, during which time she aged very rapidly and commenced to complain of severe headache and of numbness in the neck. Soon after this verbal amnesia became very evident.

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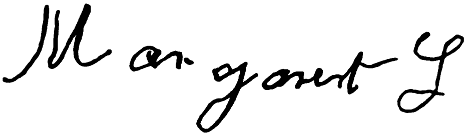
[1] This question and other matters connected with the mechanism of speech are fully discussed in my monograph on Cerebral Localization, and also in a paper on Aphasia, in the Review of Insanity and Nervous Disease for September and December, 1891.

[2] Ross: Aphasia; in Wood's Med. and Surg. Monographs, vol. vi. No. 1, April, 1890.

This patient was first seen by me in consultation with Dr. Wilson Bowers, July 16, 1894. In April she had a spell of vertigo, on rising in the morning, and toward noon a similar attack, and from this time on it had become almost impossible for her to name objects. Until the first of August she was seen by me five or six times, and careful examinations during this period showed neither anæsthesia nor paralysis. Ophthalmological examination showed no optic neuritis, but an irregular left lateral homonymous hemianopsia. The following is a fac simile of an effort to write her name:

FIG. 109.



Name written by patient with verbal amnesia and partial word-blindness from lesion of the naming centre and adjoining regions.

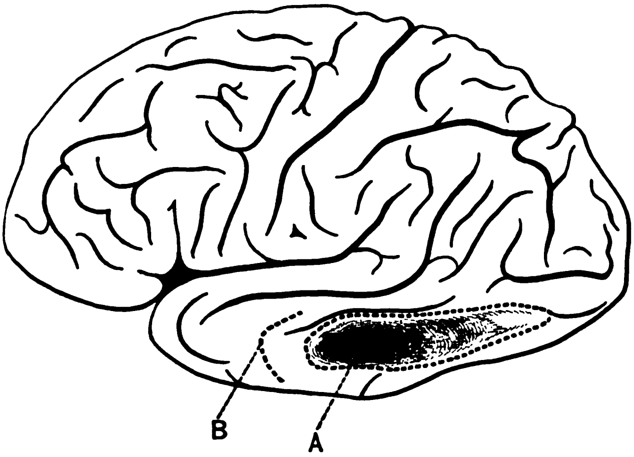
She was word-blind in large part, but not letter-blind; she could name single letters slowly.

She could not name objects either from sight or touch. When a pencil, pen, scissors, or purse was held before her, or when she was allowed to touch them, she could not give their names, although she understood what they were. On one occasion she called the scissors "what I sew with," and the purse "what I buy with." At times she became much worried and emotional because of this inability to name objects. When such objects were named to her she would promptly, and with evidences of satisfaction, indicate that the names were correct; and she could also, as a rule, repeat the names spoken before her, but not always quickly, and occasionally she had considerable difficulty in repeating them. When asked her first name, she said "Margaret" with facility, but she had great difficuty in mentioning her last name, unless it was repeated to her, although she sometimes succeeded without this prompting. She used "yes" and "no" properly, and in many ways indicated that she knew what objects were and their uses, but could not give their names. She talked spontaneously, but not freely, not using concrete nouns, or but rarely, and sometimes misplacing words. She was not seen by me after August 1st, but Dr. Bowers has kindly furnished me with notes of her condition from that date until her death.

August 1st she became unable to perform shoulder and elbow movements and leg and thigh movements on the right side, but retained power in the hand and fingers and foot and toes. Gradually the paralysis of the right limbs became complete, but without loss of sensation. The skin on the paralyzed side assumed a pinkish hue, and its temperature was increased. The pain in the head disappeared when the paralysis became complete, but returned to some extent a week before her death. Three weeks before her death she regained some power in the toes and fingers. During the last weeks of her life "yes" and "no" were the only expressions used by her. She became somnolent, and later stuporous; and she developed bedsores and lost control over the vesical and rectal sphincters. Death occurred September 10, 1894.

An autopsy was made by Dr. C. W. Burr, Dr. J. W. McConnell, Dr. W. Bowers, and the writer. In the course of the removal of the brain a small nodulated, half-disintegrated mass, about the size of a hickory nut, was pulled out of the brain surface at a position which corresponded to the posterior fourth of the third temporal convolution (at B, in Fig. 110). The surface of the third temporal in its posterior half, and to a much less extent of the second temporal in the same general region, and of the fourth temporal, presented a granular, slightly disintegrated appearance. On cutting into the temporal lobe a tumor, hard and yellowish-brown in color, was revealed; its hardest, and apparently oldest, part was at A, about the middle of the third temporal (meditemporal gyre), and passing slightly into the second temporal. The mass extended cephalad and caudad a short distance, almost entirely in the white matter of the third temporal gyre, but a soft, nodulated, more or less hemorrhagic condition reached caudad as far as the white matter of the middle of the occipital lobe, and cephalad to the junction of the first and middle thirds of the second and third temporal convolutions, as indicated by the dotted lines. The parts chiefly destroyed were the white matter of the third; to a less extent, of the second; and to a still less extent of the fourth temporal convolution. Internally the roof of the posterior horn presented a slightly granular appearance. The disease almost certainly started in the third temporal convolution, at a point in a line with the posterior extremity of the horizontal branch of the Sylvian fissure.

FIG. 110.



Tumor of the third temporal convolution, indicating the position of the naming centre. A, densest and probably oldest portion of the growth. B, anterior limit of the lesion beneath the cortex: the limits on the surface of the portion surrounding the lesion are indicated by the dotted lines.

OLFACTORY LOCALIZATION.

With such light as is furnished by histology, pathology, and physiology, the olfactory centre can be best located in the uncinate gyre, as indicated in the diagram, (Fig. 105). This position, as stated by Ferrier, is indicated by the evident anatomical connections of the region with the olfactory bulb. "The chief, and, in man, the only constant connection of the olfactory tract with the hemispheres is by the so-called external root, which passes outward across the anterior perforated space to the cortex of the hippocampal gyrus." Broca compares the connections of the olfactory tract with the falciform or limbic lobe, by means of its inner and outer root, to a tennis racquet, of which the circumference is formed by the lobe and the handle by the olfactory tract and bulb, the force of which comparison is seen on an examination of Fig. 105.

Zuckerkandl, of Gratz, in 1887, published an anatomical and physiological monograph, in which he claims that the entire limbic lobe is the seat of olfactory sensation. In this view he simply follows Broca, who divided all animals into osmatics and anosmatics, holding that the whole of the falciform lobe was the cerebral organ of smell; but a knowledge of the relatively large size of the gyrus fornicatus and hippocampal region in an animal like man, in whom the sense of smell plays so comparatively an unimportant part, is a weighty argument against the views of Broca. In osmatics, however, the the hippocampal lobule or region of the amygdala—the uncinate convolution, so-called—is very large, while in the anosmatics it is comparatively small; and Ferrier's view is, therefore, probably correct.

Luciani and Tamburini believe that in dogs the region in front of the auditory area is especially olfactory in function.

Electrical irritation of the hippocampal lobule or uncinate gyrus in monkeys, cats, dogs, and rabbits furnished Ferrier with significant indications of subjective olfactory sensations in the form of a peculiar torsion of the lip and nostril of the same side.

Ferrier refers to cases reported by W. Ogle, Fletcher and Ransom, of the occurrence of loss of smell in the left nostril with right hemiplegia and aphasia; and he also alludes to cases reported by Hughlings Jackson with lesions of the region of the hippocampal lobule. The connections of the olfactory tract are with the hemisphere on the same side. I have had several epileptic patients in whom the attacks were initiated by an odor, usually offensive; and Hughlings Jackson gives interesting histories of three such cases in which the attacks were ushered in by a crude sensation of smell, accompanied sometimes by other warnings, as epigastric sensations and the dreamy state. These cases are of great clinical interest, but are not accompanied by autopsies. He refers, however, to the necropsy of a woman who had paroxysms with the dreamy state and crude sensation-warnings of smell. She had left hemiplegia and double optic neuritis, and the autopsy showed a tumor in the right temporal lobe. McLane Hamilton[1] has reported a case of epilepsy with softening of the temporal lobes, in a woman of forty years, who always had as an aura a disagreeable odor, sometimes of smoke, sometimes of a fetid character, and quite uncomplicated by other sensory warnings. Worcester[2] has reported the case of a farmer, aged thirty, who had had epilepsy for two years before admission to the hospital. For several days hallucinations of smell—at first constant, afterward transitory—were present. Once he imagined the room was full of smoke. He fancied at times there was an odor like the vapor of alcohol passing quickly. He thought this took the place of a convulsion. The autopsy revealed a small spot of red softening at the most prominent part of the left gyrus uncinatus. The brain was not opened until it had been hardened in alcohol. A focus of softening existed in the white matter of the anterior part of the left temporal lobe, extending to the surface, externally, and internally involving the pes hippocampi in the floor of the descending cornu of the lateral ventricle. The portion of the hippocampus major not discolored was swollen and softened. A very small focus of softening, about the size of a large pea, was found in the white matter of the frontal lobe on the same side.

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[1] A. McLane Hamilton: Am. Journ. Med. Sci., April 1, 1884. Quoted by Starr.
[2] Worcester: Am. Journ. of Insanity, July, 1887.

GUSTATORY LOCALIZATION.

The sense of taste is closely related to that of smell, and in the diagram has been placed in the fourth temporal convolution. The experiments of Ferrier seemed to show that affections of both taste and smell were evidently connected with lesions of the hippocampal lobule and its neighborhood. He noted in connection with electrical irritation of the lower extremity of the temporo-sphenoidal convolutions in the monkey, and of the same region in the brain of a cat, that the movements of the lips, tongue, cheek, pouches, and jaws were occasionally induced—phenomena which he believed might be regarded as indications of the excitation of the gustatory sensation. Anderson[1] has recorded a case of epilepsy in which from symptoms, ocular and cerebral, detailed in his report, he correctly predicted tumor and its position. The patient's dreamy state was associated with a rough, bitter sensation in his mouth. It is the only case published, according to Jackson, in which a necropsy has been had revealing any morbid changes in a case of the variety of epilepsy mentioned. Gad concluded that biting, chewing, and swallowing are in the rabbit reflex acts dependent upon the brain stem, while the formation of the bolus and its backward movement are governed by the cortex. Schtcherback[2] attempted to discover whether these actions could be rendered impossible by the destruction of any definite part of the cortex, and what was the mechanism of the origin of these movements. In all cases in which the power of swallowing at will is lost, loss of taste was also evident. He found that injury to an area extending from two to three millimetres in front of and behind the line of the coronal suture, and vertically from the longitudinal fissure to the lower edges of the brain, caused loss of taste on the opposite side of the tongue, a loss which lasted for six days at the longest. "The localization," he says, "of the described centre presents no special difficulties, neither upon the skull nor upon the exposed brain. In the former case the coronal suture serves as a guiding line; in the latter case a line corresponding to it and passing at the base of the brain through the posterior border of the chiasm of the optic nerves. The gustatory centre extends two to three millimetres anterior and posterior to this line (somewhat more posterior than anterior), and lies upon the entire convex surface of the hemisphere, from the longitudinal fissure to the lower border in the region corresponding to the precentral lobe."

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[1] **Anderson**: Brain, October, 1886. Quoted by Hughlings Jackson in Brain, July, 1888.

[2] **Schtcherback**: Centralbl. f. Physiol., vol. ii. pp. 289–298, August 29, 1891. Abstract in Brain, vol. xiv. 1891, p. 574.

THE PREFRONTAL REGION.

When we speak of latent regions we simply indicate either our ignorance or the want of refinement of our knowledge, as every well-defined brain region must have its function. The prefrontal region is sometimes designated as a latent district, but this is an error even with our present light. The symptoms are largely psychical, and unfortunately the physician is not usually well trained to study such phenomena. Mental disturbances of a peculiar character occur, such as mental slowness and uncertainty, want of attention and control, and impairment of judgment and reason; closely studied, the inhibitory influence of the brain, both upon psychical and physical action, is found to be diminished. Memory is not seriously affected, although a continuous train of thought cannot well be followed, and complex intellectual processes cannot be thoroughly performed. The results of experiments upon lower animals have not been very helpful toward determining the existence of prefrontal lesions, because psychical phenomena cannot be studied with accuracy in animals below man. Ferrier, however, found, after removal of the prefrontal lobe, a decided alteration in the behavior of the animals, difficult precisely to describe. They had apparently lost the faculty of intelligent observation. Horsley and Schäfer, Hitzig, and Goltz have also observed apparent mental changes.

THE RIGHT TEMPORAL LOBE. The right temporal lobe is probably the most latent district of the brain; although if the regions for word-hearing or the general auditory sphere in the left temporal lobe is destroyed, subsequent destruction of the corresponding areas in the right temporal lobe will complete the cerebral deafness which has been partial as the result of the first lesion. With a large lesion of the right temporal lobe, as a tumor, hemorrhage, or abscess, the diagnosis is best made by a careful consideration of pressure and invasion symptoms, in addition to those which are strictly localizing in character.

APHASIA, AND THE CORTICAL MECHANISM OF SPEECH.

It is often most important for the practitioner of medicine to be able to quickly make the diagnosis of particular varieties of cerebral speech disturbance. To many physicians the subject has an unnecessarily difficult look. A knowledge of the special cortical centres already described, with a further knowledge of the tracts which relate them to each other and to the outside world, should enable the examiner to proceed by a thoroughly systematic method to the determination of the special character of a speech disturbance, and the site of the lesion or lesions to which it is due.

The centres already alluded to which have received consideration in the preceding pages are for word-hearing, word-vision, naming, and incidentally for propositionizing and articulation as well as for the motor centres which play their part in writing and pantomimic speech. Special terms in common use will need to be defined. It will be especially necessary to consider briefly certain anatomical and physiological facts with reference to the entering, associating, and emerging tracts for speech, in addition to the matters already discussed.

The word aphasia, which means loss of speech, is the general designation or the generic term applied to all the various forms of defects of the elements of speech due to disease of the cerebral hemispheres—usually lesion of the cortical centres concerned in the mechanism of speech. It is to be distinguished from the defects of speech that result from mechanical difficulty in articulation which are due to lesions of peripheral organs or nerves, and also from speech disturbances which are dependent upon the cranial nuclei, and the associating and co-ordinating tracts for speech in the cerebellum, pons, and oblongata.

Aphasia is therefore the loss, partial or complete, of the power of expression or comprehension of language. It may be either sensory or motor. The former may be termed receptive and the latter emissive.[1]

The subdivisions of the subject of aphasia which will be considered are as follows:

Motor aphasia:
  Aphemia (complete motor aphasia),
  Articulative ataxia,
  Articulative paresis or paralysis (?),
  Agraphia,
  Amimia.
Sensory aphasia:
  Auditory aphasia (word-deafness, music-deafness),
  Visual aphasia (word-blindness, alexia),
  Apraxia (mind-blindness, soul-blindness, object-blindness).
Conceptual aphasia:
  Complete verbal amnesia,
  Incomplete verbal amnesia (articulative amnesia),
  Literal amnesia (amnesia for written language),
  Symbolic amnesia (amnesia for signs other than letters or words).
Conduction aphasia:
  (Paraphasia, paragraphia, paramimia, paralexia, dyslexia.)
  Subcortical (infra-pictorial sensory aphasia),
  Transcortical (supra-pictorial sensory aphasia),
  Transcortical (supra-pictorial motor aphasia),
  Subcortical (infra-pictorial motor aphasia).

Wernicke has applied the principles which are illustrated in the above tabulation to written speech, and differentiates seven varieties of aphasic disturbance of written speech, all of which are indicated above. These are cortical alexia, cortical agraphia, subcortical alexia, transcortical alexia, transcortical agraphia, and subcortical agraphia.

Besides the subjects indicated, brief attention will be given to the methods of investigating patients suffering from cerebral speech disturbances.

Four of the forms of aphasia included in the above arrangement are usually considered as elementary, namely, aphemia or complete motor aphasia, agraphia, word-deafness or auditory aphasia, word-blindness or visual aphasia. I believe that to these elementary forms should be added verbal amnesia, or at least that variety of verbal amnesia which is due to lesion of the naming centre, and has been discussed in the last section. In the first place, the special forms of motor aphasia will be defined and described and the sites of the lesions causing them indicated.

The diagram (Fig. 111) indicates the position in the cerebral cortex of the centres concerned in the mechanism of speech according to the views held by the writer.

Motor aphasia or *aphemia*, as Broca, Ross, Wyllie, and others have preferred to call it, has been discussed and by some authorities subdivided differently. Physiological and clinico-pathological observations have shown that so far as speech is concerned this motor disturbance may assume two or perhaps three forms. In the first place, we have the true aphasia or aphemia of Broca and others, an affection of speech which is limited strictly to the foot of the left third frontal convolution, the so-called Broca's convolution. Broadbent, in a remarkable paper on the mechanism of speech, describes this region as a propositionizing centre, that is, the place by the functioning of which thoughts are set into a framework of words, but through which the utterance of such words is not consummated. Other cortical centres essential to motor speech must therefore be present, and these are found caudad of the convolution of Broca at the foot of the two central convolutions. Broadbent preferred to call this region the utterance centre, and whatever view may be taken of the nature of the process of emissive speech, such a subdivision into propositionizing and utterance centres has a practical value in the consideration of this subject. Other writers and observers, while recognizing the necessity of some such subdivision of the mechanism of speech on its emissive side, not only use different terms, but have different conceptions of the nature of the processes which take place, although their conclusions are for practical purposes in accord. According to one group of writers, for example, the foot of the left third frontal convolution is the centre or storehouse for the guiding psychomotor images or memories, and these are regarded as largely "sensory in constitution, being made up chiefly from memories of muscular and tactile sensations." (Wyllie). Some cases of limited lesion of this region would bear out either interpretation—that is, that the centre may be properly designated as propositionizing, or as a centre of psychomotor pictures or memories concerned in speech. Total destruction of Broca's convolution in right-handed people makes spoken speech impossible, at least for a time. As time progresses, particularly when the lesion occurs in the comparatively young, the hitherto untrained centre in the right third frontal enables the aphasic to acquire or regain in some degree the faculty of speech; although, in the majority of cases of such speech disturbance in association with lesions of the left cerebrum, the recovery is as much

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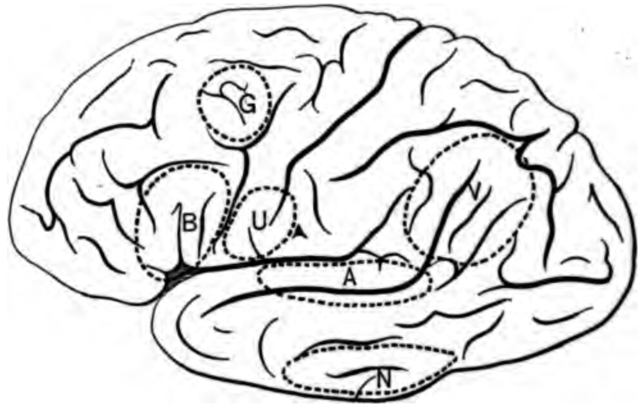
[1] In the preparation of this section, besides the writings of Hughlings Jackson, Wernicke, Lichtheim, Ross, Bateman, and the older authorities upon aphasia, frequent use has been made of the valuable series of papers on "The Disorders of Speech," by John Wyllie, M.D., F.R.C.P.Ed., in the Edinburgh Medical Journal from 1891 to 1894.

FIG. 111.



A, auditory centre (centre for word-hearing); V, visual centre (centre for word-seeing); N, naming centre (centre where percepts are given a name); B, motor speech centre in Broca's convolution); G, graphic centre; U, utterance centre.

or more due to the fact that some portion of the speech centre has not been destroyed.

It has been suggested by Moxon, Wyllie, and others that the reason that the vast majority of individuals are left-brained for movements of the limbs, and particularly of the arm, for speech, for word-hearing, and word-seeing, finds its best explanation in the fact that less expenditure of time and energy would be called for in the development of the powers of attention if one hemisphere rather than both was thoroughly trained. One hemisphere is much more highly developed than the other in obedience to the principle of economizing energy which is evident in the study of natural evolution.

Under the designation of *articulative ataxia*, Wyllie describes what is practically an incomplete motor aphasia from partial destruction of Broca's centre, or that presented by a patient who has partially regained speech after a more complete destruction of this centre and the subjacent cortex. It is simply the old *ataxic aphasia*, the *asynergia verbalis* of Lordat. Such a patient gradually improves in speech through recovery of the injured centre, or through the education of the previously uneducated centre of the right side; and as he does so he shows articulative disturbances of various kinds, such as "slurring, lalling, syllable stumbling, sometimes a little stammering, and often an utterance that is slow and laborious." According to Wyllie, in these same cases patients may show amnesia verbalis and a slight degree of paraphasia, although it seems to me questionable whether this verbal amnesia is not apparent rather than real.

Let me now recur to the subject of utterance in connection with motor aphasia so-called. In the act of utterance, or articulate speech, several processes involving movement are concerned. It includes vocalization, phonation, and the formation of sounds into elementary sounds, into words and phrases, processes which are performed by the larynx, tongue, jaw, cheeks, and lips. It will be seen by consulting several figures in preceding pages that the centres for the larynx, throat, jaws, lips, and mouth, and movements of the face in general, are situated backward from Broca's centre, chiefly at the lower extremities of the two central convolutions. If, now, cases are recorded in which limited destruction of this cortical region has caused impairment or loss of articulate speech, or the power of utterance, without destroying the patient's ability to recall those psychomotor images which are concerned in speech, while, on the other hand, destruction of the left third frontal makes all speech impossible, we should have corroborative evidence of the separation of the motor speech area into these two subdivisions. Such a case has been described by me, and referred to under the face area, the lesion present being shown in Fig. 101. This was a typical case of orolingual monoparesis, with distinct paralysis of the lower face on the same side, no interference with propositionizing being present. Instead of speaking of this general region as an utterance centre, Wyllie prefers to describe it as a region containing motor executive cells for speech, the place where speech begins to be locally exteriorized. Whatever names may be given to these different regions, many personal observations, as well as the study of the literature on this subject, have convinced me of the necessity of recognizing Broca's centre as separated in function from the utterance region, however closely the two may be allied in the ordinary processes of speech. The motor centres for utterance, in brief, are the cortical areas which innervate the muscles of the larynx, throat, tongue, mouth, and lower face. In the tabular analysis this variety of speech disturbance has been indicated as doubtfully belonging to motor aphasia, under the name of *articulative paresis* or *paralysis*.

Most cases of motor aphasia also suffer from agraphia, or the inability to write, although this loss or defect may also be due, as has been shown, to lesions of the centres for word-seeing. The motor centre for writing has been placed at the caudal extremity of the second or medifrontal convolution, and in close relationship with the upper or mesal boundary of the true speech centre. Destruction of this region, which is just in advance of the area for movements of the fingers, hand, and other parts concerned in writing, it is supposed, would cause a true agraphia, although no clinico-pathological case fully corroborative of this statement has been put on record. True agraphia, however, is an almost invariable result of destruction of Broca's centre, and opinion is returning to the views held by Trousseau and Gairdner. Wyllie, for example, cites and indorses the recent conclusions of Dejerine,[1] which are given in the following words: "Thus in proportion as we advance in the study of agraphia we find that Trousseau was right, when, speaking in his celebrated cliniques upon aphasia, of the trouble of writing in aphasics, he said, having in view certainly only the cortical motor aphasics, the only kind known at his epoch: 'Usually the aphasic is not more able to express his thoughts in speech than in writing; and, although he has retained the movements of his hands, and can employ them with as much intelligence as formerly, he is incapable of composing a word with the pen as he is of composing it for the purposes of speech.'"[2]

It is probable, as Wyllie concludes, that motor agraphia, at least so far as writing with the right hand is concerned, may result from a lesion of the graphic centre of the posterior portion of the second frontal convolution.

The study of pantomime is only second in importance to that of the study of spoken speech in the consideration of aphasia. Pantomime is, in fact, a part of speech, and its losses and defects are, on the one hand, most frequently associated with disorders of speech, while, on the other, the disturbances of speech from cerebral disease usually have pantomimic disorders as accompaniments. The whole subject of pantomime may be here dismissed, although the consideration belongs to both motor and sensory aphasia.

Amimia is loss or impairment of the power of expression by signs when this loss is dependent upon cerebral disease. *Paramimia* is the misuse of signs in efforts at the expression of thoughts. Amimia may be either sensory or motor, or it may be of mixed origin. Paramimia, like paraphasia for speech, and paralexia for reading, is usually dependent upon destruction of the commissures or associating tracts between sensory and motor centres. Many erroneous ideas with reference to pantomime are prevalent among physicians and those engaged in medico-legal work. It is supposed by many that persons who are aphasic can usually express themselves by means of signs; but, on the contrary, disorders of speech and pantomime in common go hand-in-hand. A patient may, however, regain pantomimic power sooner or in a greater degree than speech, and each case should be studied on its own merits. Loss of pantomime is frequently dependent upon destruction of the left third frontal convolution, but this power may be lost or greatly impaired by lesion of the naming or concept areas of the brain. It may accompany verbal amnesia, dependent upon disease of these centres, or of the tracts uniting them with other parts. Just as aphasics will sometimes say "yes" when they mean "no," so sometimes pantomime indicative of negation is used when affirmation is meant, or the same sign is used for both. Occasionally patients are unable to interpret the pantomime of others. The study of these disorders of pantomime can be undertaken from the same points of view, and pursued in the same anatomical directions as the studies of ordinary aphasia. The following quotation from another paper by the writer indicates the peculiarities of pantomime closely studied by him:

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[1] Comptes Rendus des Séances de la Société de Biologie, July, 1891.

[2] Clin. Med., p. 718.

"In nine cases of aphasia or pseudo-aphasia which were investigated by me notable differences and peculiarities in pantomime were presented by the patients. In one case of brachio-crural monoplegia, almost complete motor aphasia, with marked preservation of pantomime, was present; in a hemiplegic with convulsions, word-blindness, verbal amnesia, and motor aphasia, there were marked sensorimotor disturbances of pantomime; in a third case, one of right hemiplegia, and nearly complete aphasia, chiefly of the motor type, the pantomime was varied and uncertain; a fourth case was one of right hemiplegia, with marked contractures, complete aphasia of the mixed type, with a single recurring utterance, and almost complete amimia; a fifth was a case of rigid hemiplegia, paralysis of the face, almost total sensorimotor aphasia, and obstinate and energetic emotional gesticulation. In the sixth case, of marked hemiplegia of gradual development, with motor aphasia and anarthria, only a slight degree of loss of pantomime was shown; while case seven, one of right-sided pseudo-bulbar paralysis, with anarthria and preservation of writing ability with the left hand, exhibited also full preservation of pantomime. Case eight was an example of right-sided pseudobulbar paralysis and ophthalmoplegia, with anarthria, marked orolingual paresis, and full preservation of pantomime, but with considerable mental apathy. The ninth and last case recorded was one of double hemiplegia from successive lesions on the right and left side of the brain, with absolute abolition of speech and pantomime."

Sensory aphasia is usually considered as having at least three special forms, namely, *auditory aphasia or word-deafness*, *visual asphasia or word-blindness*, and *apraxia*, sometimes called *mind-blindness*, *soul-blindness*, or *object-blindness*. In the section on auditory localization, the locality of the centres for word-hearing, or for auditory images of words, has been thoroughly discussed, and, to some extent, the whole subject of word-deafness. From physiological investigations, and especially those of Ferrier, from the study of a few reported cases, and especially as the result of the findings in a case of word-deafness reported by me, in which lesions of the two superior temporal convolutions of both sides were present, the conclusion was reached that the centre for word-hearing is situated in the hinder thirds of the first and second temporal convolutions, the third, fourth, and fifth taking no part in the process. The chief part is probably played by the first or supertemporal convolution. The symptoms in case of a lesion of this region are, in the first place, word-deafness, although this may not be complete unless the corresponding region of the right cerebral hemisphere is also impaired or destroyed. Other symptoms present are inability to read aloud correctly, the patient not being able to verify what he reads by hearing. In complete word-deafness he cannot echo spoken words. Both paraphasia and paragraphia may also be present, and by some authorities verbal amnesia and articulative amnesia are also said to be present; but it is probable that in some of these cases at least, these amnesic phenomena are dependent upon involvement of the naming centre or of the tracts leading from the centres of word-hearing to the conceptual or motor centres. *Music-deafness* is sometimes associated with word-deafness, but the latter may be present and the former absent. It is probable that an innate musical faculty belongs to both hemispheres of the brain to a greater degree than does the faculty of hearing. As already stated, the sphere or field for all auditory memories covers a much larger region of the cortex than that for word-hearing alone.

Between the auditory centres at the base of the brain and the cortical auditory centres in the left temporal lobe are also entering tracts for hearing. A lesion of these entering tracts which probably go from both sides of the brain will give a form of word-deafness. Lichtheim has placed the entering auditory tract chiefly in the left temporal lobe, believing that the fibres from both acoustic nuclei and both primary acoustic centres come together in this lobe and pass to the centre for word-hearing. A lesion of this tract will cause pure word-deafness, as will also a lesion of the centre for word-hearing; but in the latter case paraphasia and paralexia will be present, as the patient will not be able to verify what he says or means through his centre for word-hearing, whereas, in the former case, he can do this. A case of lesion probably restricted to this entering auditory tract has been reported by Lichtheim. This patient preserved the power of volitional speaking and writing and of reading aloud, which is lost in deafness from lesion of the centre for auditory images. He had neither paraphasia nor paragraphia.

For a full comprehension of cortical auditory localization and auditory aphasia we should have an understanding of the entire auditory tract or path. Meynert held that the central acoustic fibres all passed through the cerebellum on their way to the cerebral hemispheres; but this, as Ferrier points out, is not consistent with the results of the destruction of the cerebellum, and an explanation is to be found in the fuller understanding which has been recently reached of the subdivisions and functions of the so-called eighth nerve, which, in reality, consists of two distinct portions, which might, perhaps, be better classed as two distinct nerves. These and their central paths have been studied experimentally by Baginski, and microscopically with great care by Flechsig, Bechterew, Bruce, and Hans Held, and particularly by the latter. A dorsal or posterior root becomes the cochlear nerve, the true nerve of hearing, while a ventral or anterior root becomes the vestibular nerve, which is probably a nerve of space or equilibration. The vestibular nerve, which arises in the ganglion of Scarpa, in the semicircular canals, has a portion of its central path by way of the cerebellum. Possibly a centre for equilibration or space may be present in the cerebral cortex, but of this we have not as yet any knowledge. In connection with the question of auditory localization, we need at present only to concern ourselves with the source and termination of the cochlear nerve. This cochlear or acoustic nerve, according to the latest researches, originates in the spiral ganglion or organ of Corti, which corresponds to a posterior spinal ganglia. It passes to the lateral tubercle and accessory nucleus of the same side, thence decussating by the trapezium and medullary striæ, its fibres enter the lateral layers of the fillet, passing by way of the superior olives, trapezoid nuclei, lateral nucleus, post-geminum and post-geniculum, and, eventually, by cerebral fibres to the cortex of the temporal lobe. The post-geminum or posterior quadrigeminal body, and post-geniculum or internal geniculate body, are probably primary acoustic centres, or central terminations of the reflex acoustic paths. It is altogether probable, as Luciani and Tamburini have suggested, that we have a semidecussation of the acoustic nerves similar to that of the optic nerves, and that therefore both ears are represented in each cerebral hemisphere. "This latter is undoubtedly the case; for unilateral extirpation never gives rise to permanent deafness of the one ear; but though I have had on many occasions, after extirpation of the auditory area in one hemisphere, observed loss or impairment of hearing in the opposite ear, I have never been able to detect the slightest impairment of hearing in the ear of the same side." (Ferrier).

Just as there are anatomical and physiological homologies between the optic and the acoustic paths, so there are doubtless clinical phenomena which are more or less fully comparable with those of simple amblyopia and hemianopsia; and the discovery and study of these will eventually assist in the subdivision of the auditory field.

Visual localization, like auditory localization, has already been considered at length. Although Henschen believes that the macular field lies in the calcarine cortex, I do not accord with this view. The cortical field for the macula is doubtless also the field wherein are stored the visual images of words, of letters, and, probably, of objects; and this is probably in the angulo-occipital region on the lateral surface of the hemisphere, practically where it was located by Ferrier. Limited destruction in this region will produce word-blindness, and letter-blindness. *Alexia*, or the inability to read, will also, of course, be produced by such a lesion, as will also agraphia, at least, so far as this is dependent upon sight. Patients who have been rendered word-blind and alexic by a lesion of the cortical area for word-seeing can sometimes write their names or a few simple words, or, in rare cases, a number of words, apparently doing this through touch or recognition of psychomotor images.

Between the primary optic centres at the base of the brain and the primary visual centres in the cortex of the occipital lobe, which are situated in the calcarine region, are certain entering tracts for vision, the optic radiations of Gratiolet. The primary cortical visual centres themselves are connected with the retinas of both eyes, but only with half of the retina of each eye, the half on the same side of the head as itself. From these primary cortical centres of both occipital lobes tracts pass to the angular region and its neighborhood in each hemisphere. This region is a centre of high function, one in which is stored the visual images for words and objects. A lesion which severs the tracts going from both occipital lobes to this higher visual centre will cause word-blindness but not agraphia, as the memory pictures can still be revived and made use of by the motor centres for writing. Commonly a lesion of this kind causing word-blindness will also cause hemianopsia, because the radiations of Gratiolet are usually involved.

Not infrequently associated with word-blindness is another disorder which has been variously called mind-blindness, soul-blindness, and object-blindness. The word apraxia is also used by Kussmaul in practically the same sense for the loss of memory of the uses of things and the understanding of the signs by which things are expressed. In testing for this condition, the physician observes whether the individual examined shows signs of recognition of objects of various kinds which are presented to him; he may not comprehend the use of the simplest things; and he also may not recognize persons with whom he is intimate. In such cases the person may recognize another when he speaks, or by touching him, even when he cannot by sight. Sometimes, however, a comparable form of mind or psychical-deafness is observed in which the patient cannot recognize another by a word used or by the familiar sound of the voice. The centre for the visual images of things may or may not be separate from that for the visual images of words, but both are in the same adjacent regions of the cerebral cortex. Both hemispheres of the cerebrum doubtless take part in the storage of such object images, and probably in nearly equal degree, whereas for words, the man is mainly left-brained as for many other of the higher faculties. It is true that a few cases of partial mind-blindness are on record in which unilateral lesions are present.

The mental percepts of objects and the mental percepts of names are the results of different processes of cerebration. Wyllie holds, with many others, that it is not necessary to have a special cortical centre set apart for either of these processes, that is, that special "ideational" centres and "naming" centres probably do not exist; but that the interaction of the entire cortex, or of certain of its layers, is concerned with ideation, and that names arise in consciousness through the action of the centres for percepts and the motor centres. Whatever may be true as regards the formation of ideas, my own experience and views as to cerebral action have led me to the opinion of Broadbent, that a naming centre at least exists in the cortex, and in the preceding pages I have recorded the details of a case which would seem to be corroborative of this position.

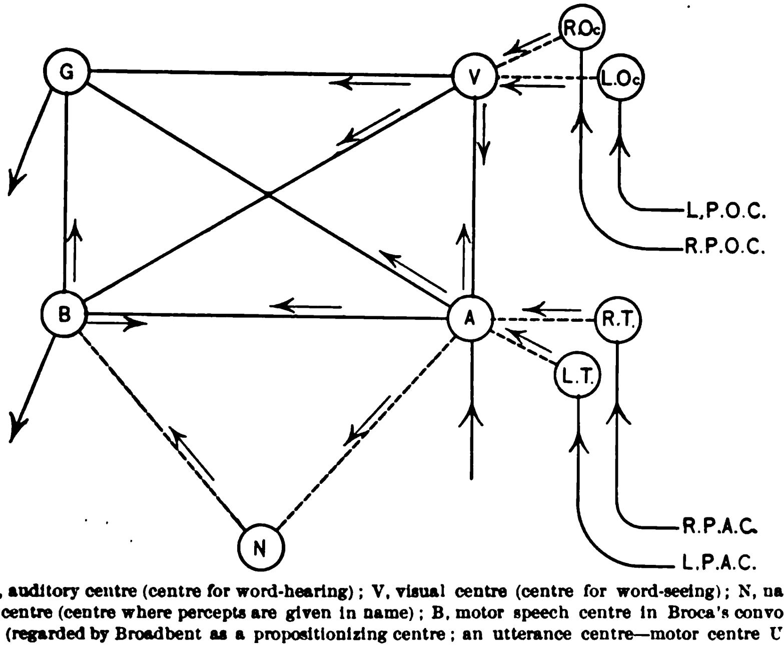
As shown in the preceding section on the naming centre, destruction of this area will cause loss of memory of names or nouns, in other words, one of the forms of *verbal amnesia*, but this may be considered as a conceptual aphasia, using the word conceptual in the sense already described in speaking of idea, concept or naming centres. A form of verbal amnesia of incomplete type, *incomplete articulative amnesia*, may be due to partial destruction of this region, or of the channels connecting it with its correlated centres on either the receptive or emissive side of the brain. It must not be forgotten that real or apparent verbal amnesia may, however, be due to lesions variously situated, in fact lesions in almost any area concerned with the mechanism of speech. *Amnesia literarum*, to which Wyllie refers, may be due to lesions similarly located to those causing verbal amnesia. This term is used "as indicative of a failure (on the productive side) to call up the images of letters and words in the mind when the effort to write is being made; in contrast to word-blindness, which implies a failure on the side of reception and interpretation."

Conduction aphasias are those forms of speech disturbance which are due to defect or destruction of the tracts which associate the different regions concerned in this mechanism of speech. Certain terms need to be defined in this connection. The general term for these conduction aphasias is *paraphasia*, which may have as many types as there are commissures. *Paragraphia* is the misuse of words in writing; *paramimia*, the misuse of signs or pantomime; and *paralexia*, the misuse in reading of either syllables or words; *dyslexia* refers to difficulty or fatigue shown in reading. These may all be regarded as forms of conduction aphasia.

The following, which is summarized from Wyllie, expresses the different varieties of speech which are present in the different varieties of conduction aphasia in connection with the special sites of different lesions. The first names given are those of Lichtheim and Wernicke, the designation given parenthetically being those used by Wyllie. These varieties of aphasia are (1) subcortical (infra-pictorial sensory aphasia); (2) transcortical (supra-pictorial sensory aphasia); (3) transcortical (supra-pictorial motor aphasia); (4) subcortical (infra-pictorial motor aphasia). Wyllie has suggested the name inter-pictorial aphasia for the conduction aphasias, applying the term pictorial also to both the sensory and motor aphasias which we have already considered.

In subcortical, or infra-pictorial sensory aphasia, simple word-deafness, without verbal amnesia or other associated symptoms of speech disturbance, is characteristic. The auditory centre is intact, but is cut off from incoming words which usually reach it from both ears. Speaking, reading, and writing are not affected, the patient is word-deaf, he hears the words as sounds, but does not understand what is said. In transcortical or supra-pictorial sensory aphasia, incoming audible speech is not understood, nor, according to Lichtheim and Wernicke, is incoming visual speech, as the path between the auditory centre "and the centre where the idea or meaning to be expressed, or to be evoked is located"—what I would call the naming or concept centre—is cut across, so that the auditory word-pictures revived in the auditory centre do not call up the ideas or meanings in this higher centre. What is heard or read is not understood, and yet the word-images in the auditory centre are intact and can be revived from without. Whatever is heard can be repeated, and the patient can easily read aloud, but he understands neither what he hears nor what he reads. He can write to dictation and he can copy from print or from writing. A true echolalia is the most characteristic symptom, which has its equivalent in his written speech, in his power of copying or of writing to dictation, which he retains without any understanding of the words so written or copied. Amnesia of nouns, paraphasia, and in consequence of the latter, paragraphia, are also shown. The patient cannot call up from the centre, where the meaning to be expressed is located, the auditory images at the auditory centre for motor memories of speech. In transcortical or supra-pictorial motor aphasia, the path between the centre where the idea or meaning to be expressed is located, and the centre for motor memories of speech in Broca's convolution, has been cut across. The patient cannot express his thoughts when he tries to speak volitionally. The failure is the same as in an ordinary case of motor aphasia, when the centre for the motor memories of speech is destroyed. According to Lichtheim, he cannot express his thoughts any better in writing than in volitional speech. Everything else is normal. What he reads and what is said to him are both understood, and he can repeat with correct articulation words that are spoken to him, understanding them in this variety. He can also write to dictation and copy writing with ease and correctness, understanding in both cases what he writes.

FIG. 112.



A, auditory centre (centre for word-hearing); V, visual centre (centre for word-seeing); N, naming centre (centre where percepts are given in name); B, motor speech centre in Broca's convolution (regarded by Broadbent as a propositionizing centre; an utterance centre—motor centre U in Fig. 111 is also required to complete the motor side of the speech process; if the view is accepted); G, graphic centre; R. Oc., primary cortical visual centre in the right occipital lobe; L. Oc., primary cortical visual centre in the left occipital lobe; R. P. O. C., optic centres at the base of the brain, right side; L. P. O. C., optic centres at the base of the brain, left side; R. T., primary cortical auditory centres in the right temporal lobe; L. T., primary cortical auditory centres in the left temporal lobe; R. P. A. C., auditory centres at the base of the brain, right side; L. P. A. C., auditory centres at the base of the brain, left side.

In subcortical or infra-pictorial motor aphasia the lesion cuts across only those fibres which connect the centre for the motor memories of speech in Broca's convolution with the centres for the nerves of speech in the oblongata. The connecting fibres between the centre of motor memories of speech and the other speech centres, and the fibres between it and the centre, where the idea or meaning to be expressed is located, are all intact. If this lesion exists, spoken speech, both volitional and on attempted repetition of the words heard, would be disabled. "Otherwise, everything would be normal; there would be no amnesia verbalis, no difficulty in volitional writing, writing to dictation or copying, and no word-deafness." If paralysis prevents writing with the right hand it can still be done with the left. In some cases sensory centres, motor centres and associating fibres, may all be involved at the same time, giving forms of *combined* or *mixed* aphasia, or it may be what is termed *total* aphasia.

The diagram (Fig. 112), which has been modified from Lichtheim, indicates in accordance with the views expressed in this and the preceding sections the various centres concerned in speech, and of the entering, associating and emerging tracts.

Wyllie makes the following suggestions for the systematic examination of a patient supposed to be suffering from one of the various forms of aphasia. He takes each of the two forms of speech, the spoken and the written, separately. If there is danger of over-tasking the impaired brain of the patient, the examination should be made gradually.

SPOKEN SPEECH. 1. *How is it received and interpreted?* Is the hearing good? The hearing being good, is there any difficulty in interpreting the meaning of words heard (word-deafness)? The patient's replies to questions, even if these replies are made only by gesture, will show whether or not he understands what is said. Ask him to put out his tongue, to shut his eyes, to give his hand, etc. He should also be tested specially as to his power of interpreting nouns and verbs. Ask him to touch, one by one, as the parts are named, his nose, ear, eye, chin, etc. Ask him to try to whistle, to shut his eyes, to smile, etc. If the examiner carefully refrains from giving the patient hints, by gesture or expression of countenance, of what is wanted, the recorded answers to such questions will indicate whether word-deafness is present, and in what degree.

2. *How is it produced?* When there is pronounced motor aphasia, and speech is reduced to a recurring utterance, one or two familiar words, and, upon occasion, an emotional or conventional expression, the whole vocabulary of the patient should be carefully noted down. When, in either motor or auditory aphasia there is considerable vocabulary we should—

a. Record words or phrases of the patient showing evidence of defective powers of articulation. As to his utterance, for example, we should note any specimens of lalling, stammering, syllable-stumbling, or slurring.

b. Look for evidences of amnesia verbalis, and its companion symptoms, articulative amnesia and paraphasia. In doing so we should show the patient common objects and ask him to name them; and, in recording his answers, we should make note of difficulties in remembering nouns, etc., as well as of mistakes in the use of them. When the patient is practically dumb we should test him for amnesia verbalis by asking him to write answers to questions; if he cannot write, by asking him, after Lichtheim's method, to indicate with his fingers the number of syllables in the names of the common objects shown him. If there is paraphasia, we should note whether the patient is immediately conscious of the errors he makes in the use of nouns, etc., or, on the other hand, is quite unconscious of them.

3. *How is it repeated or echoed?* The patient should be got to attempt the

repetition or echoing of words spoken to him. If he is word-deaf, and cannot understand a mere verbal request, to attempt this the examiner must try to get him to understand what is wanted by putting the request into the language of gesture, and repeating the word or phrases over and over again. If the patient is able to echo words or phrase the attempt should be made to ascertain whether, in doing so, he understands what he is saying.

WRITTEN SPEECH. 1. *How is it received and interpreted?* Is the sight good? Is hemianopsia present? Is the patient able to understand questions put before him in print or writing? If he is speechless, and cannot by *speech* indicate how much he understands, show him in writing or print such requests as "Put out your tongue," "Give me your left hand," etc.; and note how he complies with them. If there is some power of interpreting written or printed words, try to ascertain how much there is by asking the patient silently a sentence in a book or newspaper, and then questioning him about it in such a manner that he will not be able to answer them by gesture or by pantomime.

2. *How is it produced?* Ask the patient to write his name. If he succeeds, put simple questions to him, and ask him to answer them in writing. If he writes with comparative ease, ask him to write, at his leisure, the history of his illness. Note in his performance evidence of (1) paragraphia, which is usually only the written translation of paraphasia; (2) intoxication of the mind with a letter or word, and (3) faults of spelling or syntax. If the right hand is paralyzed, let the patient try to write with the left. If the right hand is not paralyzed, but is agraphic, let him furnish examples of the performance in writing of both the right hand and the left.

3. *How does the patient write to dictation and from copy?* Try him in both ways, and if he is able to do either, or both, try to ascertain if he understands the meaning of what he writes.

ASSOCIATED PHENOMENA. 1. *Gesture language (Pantomime).* Does the patient understand the gesture language of the examiner, and does he himself employ gesture language intelligently? Or, on the other hand, are there evidences of amimia or of paramimia?

2. *Extra-graphic Symbols.* How does he understand and employ such graphic symbols as numbers, algebraic signs, if previously shown to him; musical notation, if he was previously a musician, etc.?

3. *Is there any evidence of Mind-blindness (Psychical Blindness)* as shown by inability to recognize common objects shown to him, and to indicate their uses; or by inability to recognize intimate friends at sight?

4. *The Emotional and Intellectual Faculties.* Are the emotions lively and their expression vivid, or are they dull? Is self-control well maintained, or does the patient frequently exhibit emotional disturbance?

What is the condition of the intelligence as exhibited in expressive actions and in capability in engaging in games of skill, of conducting business affairs, etc.

5. *The Motor and Sensory Functions.* All symptoms of motor or sensory paralysis should, of course, be carefully described.

CHAPTER XV.

FOCAL DISEASES OF THE BRAIN.

By CHARLES L. DANA, M.D.

APOPLEXY.

INTRODUCTORY. By apoplexy, as I shall use the term, is meant a sudden shock due to the bursting of an intra-cranial bloodvessel or to a stoppage of the same, with consequent laceration or softening of tissue. Apoplexy is a term which has been used more especially in connection with intra-cranial hemorrhage, but since it is often impossible in an apoplectic attack to say whether the condition is due to rupture of a vessel or to obstruction of it, with consequent local softening, the term must be used to include both conditions. Apoplexy is so often associated with hemiplegia that clinically we often speak of apoplexy as the beginning and hemiplegia as the later condition; but there may be an attack of apoplexy without hemiplegia, and there may also be a hemiplegia which is not due to any apoplectic insult. The later and secondary effects that follow an intra-cranial hemorrhage resemble very closely those of an acute softening, so that it would be in many respects advantageous to describe the two conditions together. This, however, is not possible, for the reason that the causes of intra-cranial hemorrhage are different from those of intra-cranial acute softening, and the same may be said of the mode of onset, of the immediately subsequent symptoms, of the prognosis, and of the treatment. I shall therefore have to describe, first of all, the intra-cranial hemorrhages and the secondary effects from them. In describing the acute softenings I shall be able, in going over the later symptoms, simply to refer to many of the facts given under the head of the chronic stage of cerebral hemorrhage.

There are some general facts which may be stated here with regard to the two pathological conditions which I am about to describe. Apoplexy from intra-cranial hemorrhage is a condition which occurs most often a little after middle life, and much oftener in men than in women. It, however, is found with considerable frequency in the first year of life, while it hardly ever occurs in the period of advanced senility. Acute softenings practically never occur in infancy. They are met with just before the middle period of life, and then again in life's last decade. Acute softenings stand much more often in relation to syphilis, while hemorrhages are much more closely related to chronic alcoholism. Hemorrhages are more dangerous to life and are not so frequently repeated as are the acute softenings. These are some of the general facts which the student may bear in mind before proceeding to more particular inquiry into the nature of the two disorders.

CEREBRAL HEMORRHAGE. CEREBELLAR, PONTILE, AND BULBAR HEMORRHAGES.

Hemorrhages in the brain occur in various localities. The most frequent are those of the cerebrum, and they are called cerebral hemorrhages. Very often the term cerebral hemorrhage is used loosely to indicate a hemorrhage in any part of the brain.

For ordinary purposes a simple anatomical classification of hemorrhage may be used such as I have indicated at the head of the section. But owing to peculiarities in the distribution of the cranial arteries it is often convenient to make another division. Thus hemorrhages of the central arteries of the brain which supply the great ganglionic deposits are specially frequent and clinically distinct, so that we speak of central hemorrhages in the same way we speak of cortical or *pial* hemorrhages and of *dural* or pachymeningeal hemorrhages, because different groups of arteries, as well as different parts of the brain, are involved. The forms of intra-cranial hemorrhage, therefore, from the standpoint of arterial distribution and anatomical structure, are the central, cortical (or pial), dural, or pachymeningeal, cerebellar, and pons-medulla hemorrhages.

Finally, it is somewhat unfortunate that the hemorrhages of infancy have been studied and described separately, and we have as a result the term infantile cerebral hemorrhage spoken of.

THE BLOOD SUPPLY OF THE BRAIN. In order to understand the symptoms and pathology of apoplexy it is necessary to know the anatomy of the blood supply of the brain and its membranes. While I do not propose to go into many details, I shall give a description sufficient to remind the reader of the general facts regarding the subject. The external carotids furnish the blood supply to the scalp, the skull, and dura mater; the internal carotids and vertebrals furnish the blood supply to the brain proper, the pia mater, and the eye. It follows that most of the meningeal hemorrhages, in fact, nearly all those that are due to injuries of the skull and dura mater, are the result of ruptures of some branches of the external carotid. The hemorrhages into the brain substance, however, in all its parts, as well as hemorrhages of the vessels of the pia mater, are due to ruptures of some branches of the internal carotids or vertebrals. Practically the internal carotid arteries are those whose branches are most affected in non-traumatic intracranial hemorrhages.

The Brain Arteries. The internal carotid passes up through the carotid foramen into the cavity of the skull, and reaching the base of the brain gives off arteries which unite with the branches from the vertebrals and basilar to form the circle of Willis. The most important arteries that this latter gives off are the anterior and middle cerebral, the posterior communicating, and the central arteries. The vertebral arteries give off the inferior cerebellar and then unite into a single basilar trunk which give off the posterior cerebral, and this, in turn, gives off the posterior communicating, which unites with the middle cerebral to form the circle of Willis. It also gives off also central arteries. This circle is completed by the anterior communicating, which unites the two anterior cerebrals. (Fig. 113.)

The Central Arteries. From the circle of Willis and the beginnings of the anterior, middle, and posterior cerebrals several groups of vessels, six in all, are given off; they supply the great basal ganglia and adjacent white matter and are called the central arteries of the brain. They are the vessels usually affected in cerebral hemorrhages of adult life.

Of the groups of central arteries which are sent off from the circle of Willis and the three large cerebral vessels the most important are those given off from the middle cerebral. These consist of two groups, one a proximal, the other a distal. The proximal group consists of small arteries, called lenticular arteries by Duret, which pass directly up into the internal and middle segments of the lenticular nucleus and the internal capsule. The distal group consists of the lenticulo-striate arteries, which send branches directly to the outer segment of the lenticular nucleus, external capsule, and caudate nucleus, and the lenticulo-optic arteries which pass to the outer and posterior part of the lenticular nucleus and the outer part of the optic thalamus. One of the lenticulo-striate arteries, larger than the rest, is considered to be an especially frequent seat of cerebral hemorrhage. It passes outward and upward at the junction of the anterior and middle thirds of the lenticular nucleus, between the latter and the external capsule, and perforates the internal capsule to end in the caudate nucleus. It is called by Charcot the artery of cerebral hemorrhage. The lenticular-striate and the lenticulo-optic sets of arteries, as well as the proximal set of arteries, do not anastomose, consequently when one of them is plugged softening inevitably occurs in the area of brain substance which it supplies.

FIG. 118.

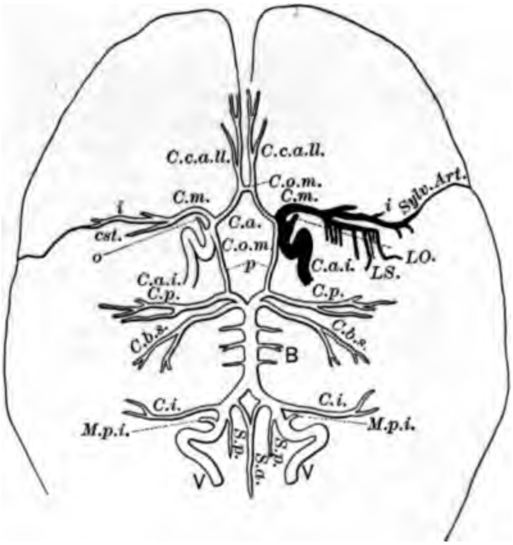
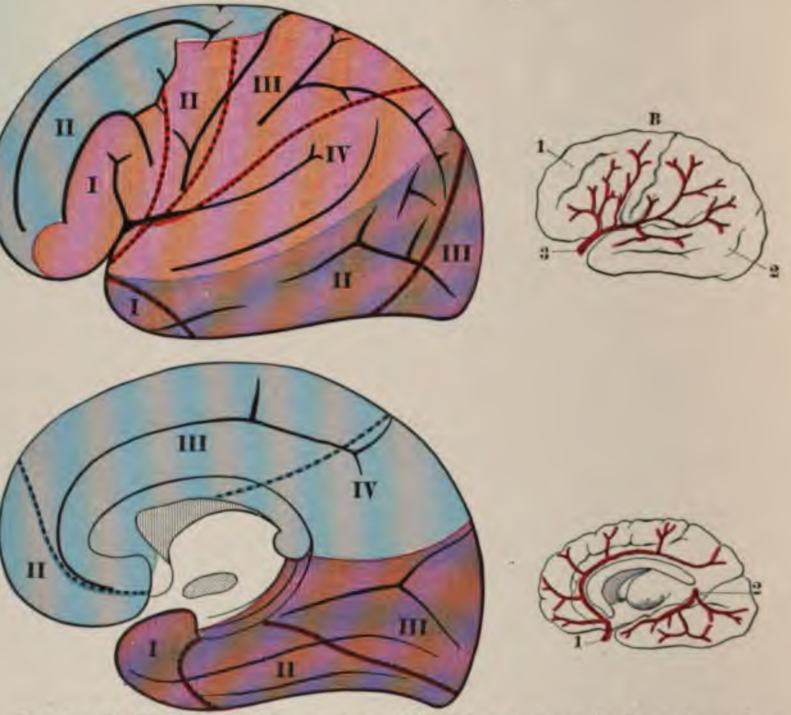


Diagram of the arteries of the base of the brain, showing *LO.* the lenticular-optic and *LS.* lenticular-striate sets of arteries. One of the latter is called the artery of cerebral hemorrhage. *V.* A. Vertebralis. *S.a.* Spinalis ant. *S.p.* Spinalis posterior. *B.* A. Basilaris with median branches. *C.b.s.* A. Cerebralis superior. *c.i.* A. Cerebelli inferior. *C.p.* A. Cerebralis posterior (profunda cerebri). *Com. p.* A. Communicantes posteriores. *C.a.i.* Carotis interna. *o.* A. Opthalmica. *C. m.* A. Cerebralis media. (A. fossæ Sylvii). *i.* A. Insularis. *cst.* A. Corp. striati. *C.a.* A. Cerebralis anterior. *Com.* A. Communicans anterior. *C. c.a.ll.* A. Corp. callosi.

While the most important of the central arteries are those which we have just mentioned as being given off at the beginning of the middle cerebral, there are two other sets of central arteries that deserve notice, they being furnished by the posterior cerebral. These are known as the postero-mesial central arteries and the postero-lateral central arteries. The former group ascend through the posterior perforated space and supply the inner part of the crus cerebri, partof the optic thalamus, and the wall of the third ventricle. The second group supplies the corpora quadrigemina and the hinder part of the optic thalamus, together with a few twigs which join the posterior choroid arteries and go to the velum interpositum. The anterior cerebral artery sends off also a few sets of central arteries, which have, however, a very limited dis-

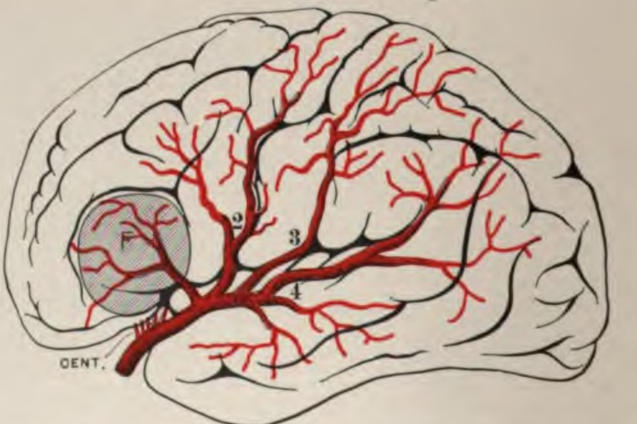
PLATE I.

Fig. 1.



Showing the distribution of the anterior, middle and posterior cerebral arteries on the surface of the brain. The numbers I, II, III, IV indicate the areas supplied by the different branches; the dotted lines indicate the main trunks. In the smaller figures, 1 indicates the anterior cerebral; 2, the posterior; 3, the middle cerebral. (Modified from Merkel and Debierre.)

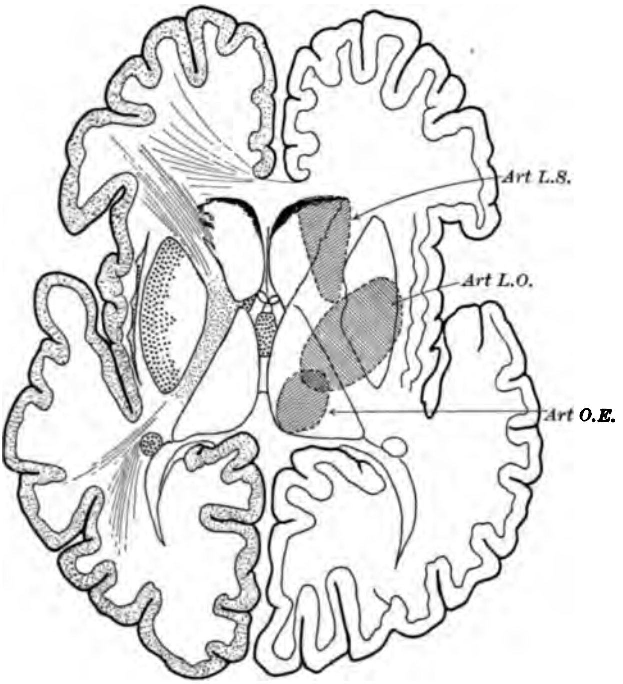
Fig. 2.



F. Softening or rupture of left inferior external frontal artery, Motor Aphasia. Softening of central convolutions occurs mainly from involvement of (3) median parietal artery, (Duret). Hemiplegia. Softening of inferior parietal and 1st temporal, from involvement of (4) posterior parietal artery, Sensory Aphasia.

tribution. They pierce the inner part of the anterior perforated space and supply the anterior extremity of the caudate nucleus.

FIG. 114.



Showing common location of hemorrhages and softenings. Areas supplied by: *Art L.S.* Lenticular striate. *Art L.O.* Lenticular-optic arteries. *Art O.E.* Postero-central arteries. In hemorrhage these limits are often exceeded; in softening not so often. In softening or hemorrhage of the thalamus alone, the arteria optic externa or the perforating arteries from the choroid plexus may be involved.

The Cortical Arteries. (Plate I.) The three great cerebral arteries above referred to, besides giving off these groups of central branches, send larger branches over the surface of the entire cerebral hemispheres. The vessels are distributed in the pia mater, and from this membrane they send down arteries into the cortex and white matter of the brain. These are called the cortical arteries. They are practically the terminal branches of the great cerebral arteries. These vessels have each a somewhat distinct area of the cortex which they supply with blood. Thus the anterior cerebrals supply the larger part of the frontal lobe; the middle cerebral supplies the central convolutions and island of Reil, and some of the temporal and parietal lobe; the posterior cerebral supplies the occipital and part of the temporal lobe. The three great arteries do not anastomose with each other in their cortical distribution to any great extent, but a degree of anastomosis does exist, so that when, for example, some of the branches of the anterior cerebral are plugged a certain amount of blood may be furnished vicariously by the middle cerebral. The cortical arteries leave the pia and pass directly into the convolutions at nearly a right angle with the membrane from which they start. There are two sets of these cortical arteries—one small and short, which supplies the gray matter of the cortex, and known as the cortical arteries proper; the arteries of the other set are longer, pass deep into the white matter of the hemispheres, and are known as the long or medullary arteries. The short or cortical arteries proper are smaller, measuring from 0.005 to 0.003 mm. in diameter; the long arteries or medullary arteries measure from 0.08 to 0.14 mm. and are from 3 to 4 cm. in length. One of these often passes down along the axis of the convolution. Both kinds of arteries anastomose freely in their finer branches.

From the anatomical arrangement it is believed that the intra-vascular pressure is less in the gray than in the white substance. The blood from the arteries of the pia mater leaves the capillaries, enters small veins, and passes for the most part upward in veins that lie in the pia. Toward the longitudinal sinus most of the vessels enter the posterior portion of the sinus and in a direction forward and upward, that is, against the current in the sinus. The course of the blood current is, therefore, opposed both to gravitation and to venous flow. The veins as they leave the pia and arachnoid in the neighborhood of the longitudinal sinus pass through the arachnoid cavity, and in many cases enter the substance of the dura and run through that a short distance before emptying into the longitudinal sinus. It is thought that at this point where the veins pass from the pia-arachnoid to the dura the venous trunks are subjected to an extra amount of strain, owing to the fact that the brain is movable while the dura mater is immovable; consequently, at this point, the veins can be more easily ruptured.

The Circle of Willis. The arteries at the base of the brain (see Plate II.) form, as I have stated, the circle of Willis. This, in its typical form, is made up of the anterior communicating, the anterior cerebrals, the middle cerebral or two carotids, the two posterior communicating, and the posterior cerebrals. This arrangement occurs in over three-fourths of the cases. The trunk of the basilar artery averages very nearly in size that of one internal carotoid, that is to say, it furnishes, supposing the pressure to be equal, one-third of the blood supply to the brain (Ehrmann).

We thus see that the cerebral hemispheres are supplied in the main by three large arteries—the anterior, middle, and posterior cerebrals; that these give off at their origin various groups of small arteries which pass directly up into the centre of the brain, supplying the basal ganglia and adjacent parts, and known as the central arteries. We see also that these three great arteries send the most of their blood to the cortical substance of the hemispheres, being distributed in the pia mater covering these parts, and that each one of the three great arteries has its own special area of distribution.

Hind-Brain Arteries. The pons-medulla and cerebellum, constituting the hind-brain, are supplied by the branches of the vertebral arteries. These vessels enter the skull through the foramen magnum, and bending from the side toward the front of the medulla oblongata unite in the middle line at the lower border of the pons-varolii to form the basilar artery. This runs forward along the median line and extends from the lower to the upper border of the pons, along the median groove in which it lies under cover of the arachnoid. At the upper border of the pons it divides into the two posterior cerebral arteries. The vertebral arteries within the skull send off a small artery to the dura mater known as the posterior meningeal. They send off also spinal arteries and numerous small arteries which are known as bulbar branches, which supply the medulla oblongata. The vertebral arteries also give off the inferior cerebellar artery which supplies the inferior surface of the cerebellum. The basilar artery gives off transverse arteries on each side which pass outward, supplying the pons, and it gives off also the anterior and the superior cerebellar arteries.

Veins. I have already stated that the blood from the convexity and me-

PLATE II.

Fig. 1.

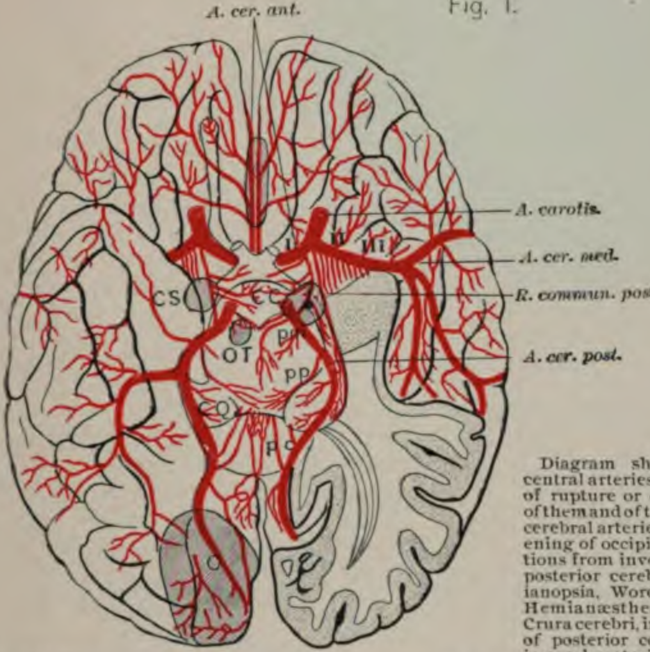
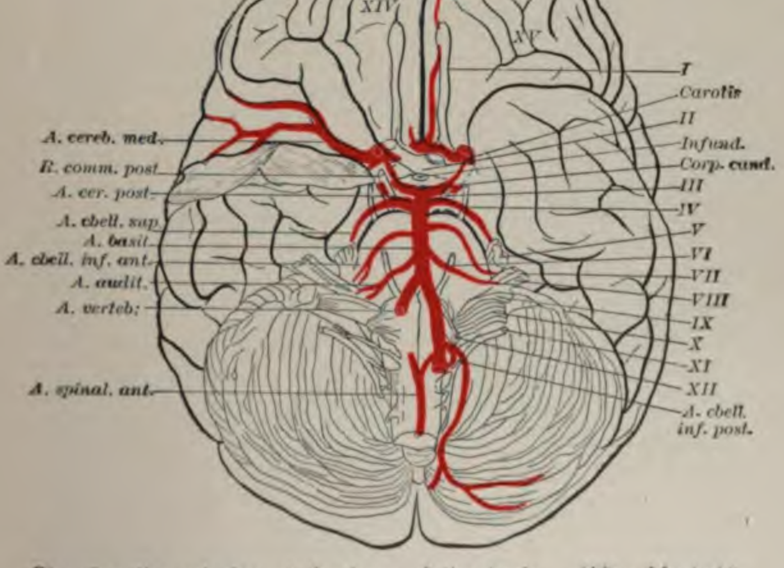


Diagram showing the central arteries and effects of rupture or obliteration of them and of the posterior cerebral arteries. O. Softening of occipital convolutions from involvement of posterior cerebral; Hemianopsia, Word-blindness, Hemianæsthesia? C C. Crura cerebri, involvement of posterior communicating and posterior cerebral. O T. Optic thalamus, mainly supplied by posterior cerebral and post-communicating. C Q. Corpora quadrigemina, mainly supplied by posterior cerebral. C. Corpus callosum, mainly supplied by anterior cerebral. C S. Corpora striata, supplied by lenticular-striate arteries.

Fig. 2.



Showing the arteries at the base of the brain. (After Merkel.)

dian surface of the cerebral hemispheres passed into veins which had a direction upward and forward, and which finally emptied into the longitudinal sinus. These veins are known as the super-cerebral veins. The blood from the inferior and lower surfaces of the hemispheres enters veins which pass to the cavernous, superior petrosal, and lateral sinuses. One of these vessels is particularly large, and is known as the middle cerebral or Sylvian vein; it runs along the fissure of Sylvius to end in the cavernous sinus, and collects blood from the frontal, parietal, and temporal lobes. The blood from the central arteries may be said in general to pass into veins which are in connection with the veins of Galen, or the Galenic system, and which end in the straight sinus. The blood from the cerebellum passes into the superior cerebellar veins which run to the straight sinus and the veins of Galen in part, and in part to the superior petrosal and lateral sinuses. On the under surface of the cerebellum the blood passes into the inferior cerebellar veins, which, together with the veins from the medulla oblongata and pons, carry blood into the inferior petrosal, lateral, and occipital sinuses.

General Facts Regarding the Circulation in the Brain. The size of the different trunks of the brain arteries is a matter which has been studied by various authors. Bevan Lewis, investigating the average diameters in 45 cases of the insane, found that the right vertebral had an average diameter of 3.147 mm.; the left, 3.42 mm. The diameter of the basilar was 3.82 mm.; of the right carotid, 3.951 mm.; of the left, 4.002 mm.; the right middle cerebral, 3.133 mm.; and the left, 3.55 mm. The diameter of the internal carotids is given by Gerhardt as 4 mm.; that of the vertebrals, as 3.5 mm. Löwenfeld has found that often the width of the brain vessels rises and falls with the width of the aorta, and that not rarely abnormally small brain arteries occur in otherwise well-developed arterial systems. He finds also that the left carotid is usually wider than the right.

The pressure in the internal carotid arteries is estimated by Gerhardt to be about 150 mm. Mendel finds experimentally that the pressure in the central or striate arteries is not much less than that in the carotids, while the pressure in the cortical arteries is very materially less. Mendel's investigations, however, were made with an artificial system of vessels, and it is doubtful if they can be strictly applied to the living subject.

Etiology. *Age.* It is generally conceded that at the time of birth and during the first two years of life there is a considerable predisposition toward intra-cranial hemorrhages. Gowers states that among 1000 persons living in the first decade of life 1.8 will have an intra-cranial hemorrhage. In a table showing the annual number of deaths from apoplexy, including hemorrhage and acute softening, in the city of New York from 1866 to 1893 inclusive—a table which was kindly furnished me by the New York City Board of Health—I find that in the first year of life the deaths from apoplexy range from 15 to 20 in a population of one and a half millions. For example, in 1868, among a total of 356 cases of apoplexy, 15 occurred during the first year of life. In 1886 there were 20 cases in a total of 762, and in 1893, 18 cases in a total of 1171. This mortality-rate drops very rapidly after the first year, so that by the fourth year there were often no cases of apoplexy reported, and practically none began to occur until after the fifteenth year. The number of deaths from apoplexy, then, begins to rise very rapidly after the twenty-fifth year, and reaches its maximum in the decade between forty-five and fifty-five. It then gradually sinks to the eighty-fifth year, after which very few cases occur. The statistical tables obtained from boards of health show pretty accurately the number of deaths from intra-cranial hemorrhage in the first year of life; but after that time they are of less value, because distinctions between hemorrhage and softening are not made. In a statistical table of forty-five cases of cerebral hemorrhage, prepared by myself, I found that the greatest number of cases occurred between the ages of thirty-one and fifty, and these may be considered as the apoplectic decades. My own table includes only hemorrhages of the central arteries; the cases of meningeal hemorrhage are excluded for the reason that many of them are traumatic, and cannot be fairly introduced:

CENTRAL HEMORRHAGES, INCLUDING MID- AND HIND-BRAIN.

Age.	*Cases.*
10 to 20	1
21 " 30	6
31 " 40	10
41 " 50	9
51 " 60	6
64 " 70	8
71 " 80	5
Total	45

The age-curve of cerebral hemorrhage, then, starts rather high, drops during the first year of life rapidly, sinks almost to a normal line until the age of fifteen, gradually rises to the age of twenty-five, and reaches its height at the age of forty-five to fifty-five, then sinks gradually to the age of seventy-five, after which time cerebral hemorrhages rarely occur. This age-curve differs somewhat from that given by the French observer, Gintrac, and places the maximum period somewhat earlier than that given by Gowers; it is correct, however, at least for the locality of New York. It is possible that in rural and more temperate neighborhoods hemorrhages occur somewhat later in life.

Sex. Hemorrhage occurs oftener in men than in women, and the proportion of males in my experience is greater than that given by other observers, namely, about two to one.

As to *race*, I do not know that any statistics have been collected which will have a very wide range of value. In a statistical report of the Mutual Life Insurance Company, prepared by Dr. Winston, and covering a long period of years, it was shown that diseases of the heart and bloodvessels and apoplexy occurred rather more frequently in the English race. In this city it occurs more frequently in the Irish race, but this is doubtless due to the preponderance of that nationality in our drinking population.

Occupation. It is frequently stated that occupations calling for excessive muscular strain tend to predispose to apoplexy; this, however, is not in accordance with my experience. Apoplexies occur, to be sure, in coachmen and drivers, and sometimes in laboring men; but, on the whole, they are found more frequently among mechanics and artisans, salesmen, and those who lead a more or less in-door life, accompanied with, if anything, only moderate exertion.

Climate. Cerebral hemorrhage is more frequent in temperate than in tropic climes, more common in winter than in summer.

Hereditary Influence. Undoubtedly, there is a hereditary influence in a small proportion of cases. This influence is shown in a tendency to arterial and renal diseases, rather than in a distinct inheritance of apoplectic attacks.

Alcoholism. Of all the single predisposing causes to hemorrhage chronic alcoholism takes the first rank. The excessive indulgence in alcohol for a number of years produces arterial disease, thickening of the meninges, and atrophy of the brain, with, in many instances, a terminal condition of cerebral hemorrhage. The moderate indulgence in alcohol in persons not especially predisposed to arterial disease has no particular influence in leading to apoplexy.

The use of *tobacco* I have found in two cases to have acted as an apparent predisposing cause, but no general statement can be made as to the specific effect of tobacco as a factor in predisposing to cerebral hemorrhage; on the contrary, its moderate use would perhaps have the opposite effect, on account of its tendency to relax the vasomotor tonus. Gout is a predisposing cause, but rheumatism is not.

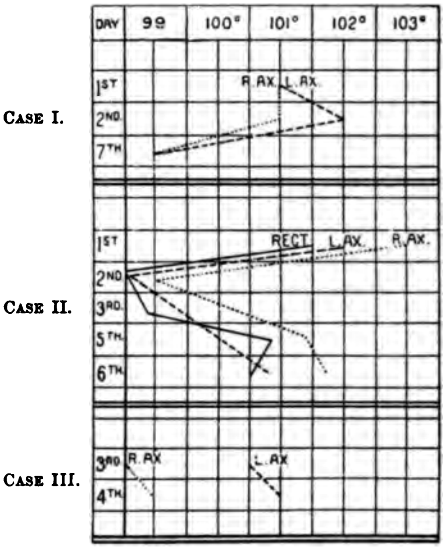
Next to alcohol, *syphilis* is perhaps the most potent factor in predisposing to cerebral hemorrhage, although it much more often leads to thromboses and softening than to rupture of the vessels. Among 179 cases of apoplexy I found sypnilis to be present in about one-fourth of the cases. Among these, however, there was a large number of acute softenings, so that I estimate syphilis to be the cause of less than one-sixth of the total number of cases of cerebral hemorrhage.

The bloodvessels of the brain sometimes rupture as a consequence of the fatty changes produced in the walls by acute infectious fevers, so that we may have intra-cranial hemorrhage following scarlatina, measles, whooping-cough, etc., though these are rather rare sequelæ. Cerebral hemorrhage also sometimes complicates attacks of pupura hemorrhagica and scurvy.

The *exciting causes* of intra-cranial hemorrhage are sometimes violent exertion, such as lifting a heavy weight, straining at stool, or it occurs as the result of great mental excitement, and in coitus; still, the great majority of cases are not brought on in this way. The vessels often rupture while a person is engaged in eating, or talking, or walking quietly. In my experience, bloodvessels do not rupture during sleep, as is often supposed, the attacks of apoplexy occurring during sleep being almost always due to thrombosis and softening. One of the most frequent exciting causes is indulgence in alcohol, and it is a common experience in hospitals to have patients brought in who are both drunk and hemiplegic, the hemiplegia being the termination of some debauch. Falls and blows on the head may also be considered as, at times, causes of intra-cranial hemorrhage, although in most instances the hemorrhage is in these cases due to rupture of meningeal vessels.

Symptoms. The immediate symptoms of an attack of apoplexy vary somewhat in accordance with the extent and location of the hemorrhage. In an ordinary case of cerebral hemorrhage the phenomena are somewhat as follows: The patient is often feeling, at the time of the occurrence, quite well; in some instances, however, his attack is preceded by sensations of fulness in the head, perhaps slight nose-bleed, sometimes a somewhat persistent headache. He may have had insomnia for a few nights, or disturbed sleep and bad dreams, and may have suffered from feelings of numbness and prickling in one side of the body. These premonitory symptoms, however, are much more frequently associated with thrombosis and softening than they are with intra-cranial hemorrhage. With or without such premonitory symptoms, the patient suddenly experiences a feeling of fulness and dizziness in the head, and then falls down unconscious. He is lifted into the bed, and then found to be suffering with a paralysis of one side of the body; this paralysis involving the arm and leg, and to some extent the face. The eyes are partly closed; the face is flushed; the heart beats slowly (50 to 60 per minute); the pulse feels hard and full; the carotids can be distinctly felt, and perhaps seen throbbing in the neck; the respiration is slow and stertorous. On the paralyzed side the cheek is blown out with each expiration. On lifting the paralyzed arm it falls helpless to the side, and if the leg is drawn up it also drops down limply upon the bed again. On pricking or pinching the patient some manifestation of consciousness is shown; the leg may be jerked up slowly, but the arm often remains helpless, and the patient simply carries the sound arm over and tries to push away the hand of the person who is producing the painful irritation. The pupils of the eyes are generally contracted and rather immobile, and one pupil may be, and often is, a little larger than the other, the pupil being more contracted on the sound side than on that which is paralyzed. On taking the temperature in the rectum it may be found that it has fallen two or three degrees, being 96° or 97° F., though this is not always the case; in four or five hours it will be found about normal, or a degree higher than normal, and it continues thus for twelve or twenty-four hours after the onset of the disease. It will be about one degree higher on the paralyzed side than on the sound side. (See Fig. 115.) The patient may continue in

FIG. 115.



Showing unilateral temperature in cerebral hemorrhage. The first patient had a difference of 2° F. on second day; the second a nearly similar difference on third, fourth, and fifth days.

this comatose or semi-comatose condition for several hours or a day; he gradually, however, becomes more conscious, recognizes friends, answers simple questions, begins to swallow food, and shows a general improvement in symptoms. At the end of twenty-four hours he may be able to move the leg a little, and perhaps the arm. The breathing is less stertorous, and the pulse has become more rapid, and regular and softer. Within three days, if the patient is going to recover, he will have regained largely his consciousness, and with it some slight mobility on the affected side. With returning consciousness it may be found that there is loss of sensation upon the paralyzed side, but this is rarely complete unless the hemorrhage is a most severe one. By the third day the apoplectic patient begins to show evidences either of a recovery from the attack or a fatal issue. In the former case there is a subsidence of the tendency on the part of the temperature to rise, both in the rectum and on the paralyzed side; in other words, the unilateral disturbance of temperature becomes less, consciousness gradually returns, and the patient is able to speak, to describe his condition, and to understand the conversation addressed to him. The paralysis improves slightly, although it still remains very considerable, and by this time there may be a slight increase in the deep reflexes of the affected side. The pulse becomes normal, the pupils even, the patient swallows and digests his food. There is a steady improvement in all symptoms, until at the end of from four to six weeks he has reached a very considerable degree of recovery. At this period he enters upon what might be called the chronic stage, which I will describe later. In cases in which the lesion is in the left hemisphere the patient has a disturbance in the faculty of language, known as aphasia; he is unable to express his ideas, although he understands what is said to him; or, on the other hand, he may be able to talk a little, but he skips various words and puts them together wrongly, and is in a condition which is known as paraphasia; or, finally, he may be able to talk intelligently, and express his wants by writing, but he is unable to understand what is said to him, and is unable to read. This condition, which is known as sensory aphasia, is often associated with some degree of anæsthesia on the paralyzed side, and with some hemianopsia. (See article on Aphasia, page 432.)

If the patient at the end of the third day does not begin to improve, new symptoms develop. His temperature begins to rise again until it may reach in a few days 102° or 103°, being still, as a rule, a little higher on the paralyzed side. He becomes unconscious again, or lies in a state of muttering delirium, being restless, throwing his sound limbs about, pressing his hand to his head, apparently suffering from pain. He will often in this condition pass his feces involuntarily, the mouth becomes foul with saliva, and deglutition is often difficult. The restlessness and delirium may pass into a condition of coma, and in this state the patient dies within four or five days or a week. Sometimes the patient lingers on a week to ten days, but rarely over this latter period of time. In many instances, a pneumonia develops which seriously complicates the condition and hastens the end.

The phenomena of an attack of apoplexy vary, as I have already stated, in accordance with the extent and location of the hemorrhage; but when the hemorrhage is extensive it usually breaks into the ventricles, and then is almost uniformly fatal, and the symptoms, when this occurs, are those which I have just described as being characteristic of an ordinary fatal attack of hemorrhagic apoplexy.

Special Symptoms in Accordance with Location of Hemorrhage. When the lesion is in the *frontal lobe* there are at first the usual symptoms of an attack of apoplexy, but without very much hemiplegia. The patient gradually recovers from unconsciousness, and is found to have only a moderate degree of paralysis. This rapidly improves, and if then the hemorrhage does not extend he may gradually come out of the attack with comparatively slight motor disturbance. In many cases, however, the hemorrhage in the frontal lobe gradually extends back, so that in a day or two hemiplegia does come on, or the blood breaks into a lateral ventricle, producing a severer apoplectic state than existed at first. Frontal lobe hemorrhages may therefore belong to that class of cases in which patients have what is first considered a mild apoplectic attack with a later severe and fatal recurrence of it.

Sometimes the hemorrhage occurs in the *occipital* and back part of the *parietal lobes*. In that case the hemiplegia is not very marked, and the leg is more affected than the arm or face. There is with this condition also a good deal of hemianæsthesia, and, perhaps, hemiataxia. If the lesion is on the left side there is a disturbance of the mechanism of language, causing aphasia. A test of the field of vision, if it can be made, often shows a hemianopsia. Hemorrhages of this kind are rare; the pathological condition being usually thrombosis.

In some cases hemorrhages occurring in any part of the cerebrum burst through the *cortex*, and the blood is poured out into the subarachnoid and arachnoid cavities. If this occurs convulsions almost invariably result, and these convulsions involve chiefly the side opposite the lesion. A case of apoplexy, therefore, which is complicated in a day or two by an attack of unilateral convulsions is probably one in which the hemorrhage has burst through the cortex.

Sometimes the hemorrhage attacks especially the *posterior basal ganglia*, destroying the *optic thalami* and parts of the tubercula quadrigemina. If this is the case, and particularly if the quadrigemina are affected, there will be paralysis of some of the eye-muscles, with ptosis and contraction or dilatation of the pupils; also hemianæsthesia and some hemiplegia. Such cases are usually promptly fatal.

Ingravescent Apoplexy. There is a form of cerebral hemorrhage which has been described as having a special symptomatology, and is known as ingravescent or progressive apoplexy. Dr. Broadbent first reported six case of this type, and later, cases have been reported by Puesch, Mills, MacBride, and myself. In this form of apoplexy the patient is siezed with sudden headache and vertigo, sometimes with vomiting, but without loss of consciousness. Complete hemiplegia with hemianæsthesia rapidly sets in; in the course of twenty-four hours the patient becomes somnolent and stupid, and, finally, comatose. Death occurs at the end of three to five days, with characteristic disturbances of respiration and with rise of temperature. In this form of apoplexy it has been found that the vessel ruptured is one of the branches of the external lenticular artery and that the hemorrhage lies mainly at first in the external capsule, cleaving forward and backward through the white matter, but, finally, breaking into the lateral ventricle. The special characteristic of these cases is that they begin without loss of consciousness, and that they steadily progress, and the special importance of recognizing them lies in the fact that if it be possible by treatment to stop the hemorrhage the patient may be saved, for the cases would get well if the hemorrhage did not finally break into the lateral ventricles.

Cortical Hemorrhage. Primary hemorrhages into the substance of the cortex of the brain are very rare unless they are the result of trauma, and if this factor is present the location of the trouble is more easily determined. The symptoms, when the hemorrhage is in the cortex, are almost always those of localized convulsions, for hemorrhages that occur primarily in the cortex are always small. Thus, a hemorrhage occurring into the motor area, involving the arm, will be associated with convulsive movements of that extremity. Besides these local convulsions, however, the patient's consciousness is usually seriously disturbed; he may, in fact, be semi-comatose most of the time, this condition being interrupted by frequent local spasms.

Pons Hemorrhage. Hemorrhages into the *pons* are very rare and usually small. They are accompanied with initial loss of consciousness and with spasmodic jerking movements of the limbs, more particularly of the legs. The pupils are often contracted and the respiration slow. There may be some disturbance in sensation and some hemiplegia; there is almost always a rise of temperature from 102° to 103° or 104°. The hemorrhages are usually fatal.

While hemorrhage into the pons does occasionally occur, hemorrhage in the *medulla oblongata* is very much more rare, acute lesions of this part being, as a rule, due to obliteration of the arteries and softening. If the hemorrhage in these parts is in anywise considerable, death rapidly ensues; if only moderate in amount, there is even then serious disturbance in the functions of the cranial nerves, producing paralysis of the throat and tongue muscles and disturbances in circulation and respiration. Hemiplegia and hemianæsthesia also occur.

Hemorrhage into the cerebellum is not very uncommon, b niti is difficult. Loss of consciousness occurs almost invariably, a re r is hemiplegia, but this is not always the case. Very often patic simply lie in a state of profound coma, with stertorous respirat and c tracted pupils. Vomiting occurs in, it is said, half the cases, out I h never observed it, nor has it been noted in any of the patients brought Bellevue Hospital. Cerebellar hemorrhages are apt to burst into the four ventricle and produce death.

Meningeal hemorrhages are considered elsewhere. (See page 362.)

The relative frequency with which different parts of the brain are affec by hemorrhage, as shown by my statistics, is as follows: First, hemorrhages involving the basal ganglia and breaking into the ventricles. Next in order. hemorrhages involving the corpus striatum and vicinity. Next, hemorrhag of the meninges; then hemorrhages into the cerebellum, and last, in abc equal order of frequency, hem he optic thalamus, corpo quadrigemina, and pons varolii. of intra-cranial hemorrhages the various localities were invol ving frequency:

Meningeal:		
Pachymeningeal	7	
Pial and cortical	7	
	—	14
Central:		
Ventricular	23	
Corpus striatum and vicin	7	
Optic thalamus	1	
Corpora quadrigemina	1	
	—	32
Pons		1
Cerebellum		3
Tc		50

Symptoms of the Chronic Stage— a. About two-thirds of the persons who are attacked with cereb rrhage recover from the immediate effects. After passing through the symptoms that are connected with the attack they gradually improve, and at the end of from four to eight weeks enter upon what is termed the chronic stage of the disease. The patient is then commonly spoken of as a sufferer from hemiplegia or a "hemiplegic," since hemiplegia is the striking and important symptom in the case. The paralysis which had at first affected the whole of one side, so that the patient was, perhaps, barely able to move the arm and leg, has now become so much diminished that he can walk and use this arm somewhat, while the face seems almost entirely well. The paralysis, in almost all cases, disappears to the greatest extent from the face, so that it can only be detected by a close inspection of the facial movements, there being, perhaps, a slight diminution in the innervation of the lower muscles of the face, and the angle of the mouth on the affected side being a little lower than that on the sound side. Protrusion of the tongue will sometimes show a deviation slightly toward the affected side. The control of the orbicularis palpebrarum and muscles of the forehead is never much disturbed, and now shows no disturbance at all. In very old cases a certain amount of contracture may set in, so that the angle of the mouth on the affected side is drawn up, and when the patient laughs the face is pulled over toward the paralyzed side. This phenomenon is particularly frequent in hemiplegia occurring in children. The paralysis improves next in amount in the leg; the patient is able to stand upon the once paralyzed limb; he can easily extend the foot, but flexes it with more difficulty. Flexion and extension of the leg are less perfect, while the control of the thigh muscles is most limited of all, not because there is a great degree of paralysis, but on account of the stiffness of the part. The arm recovers least. The patient is able to swing the arm as a whole forward and backward, but he can raise it but slightly; he can flex the forearm and extend it with a fair degree of strength. The movements of the fingers and hand are imperfect. Flexion is fairly well preserved, and the patient can grasp things, holding a cane or a table utensil often with a certain degree of ease; extension of the fingers, however, is very weak and so also is supination, pronation being fairly well retained. It will be seen in examin-

FIG. 116.

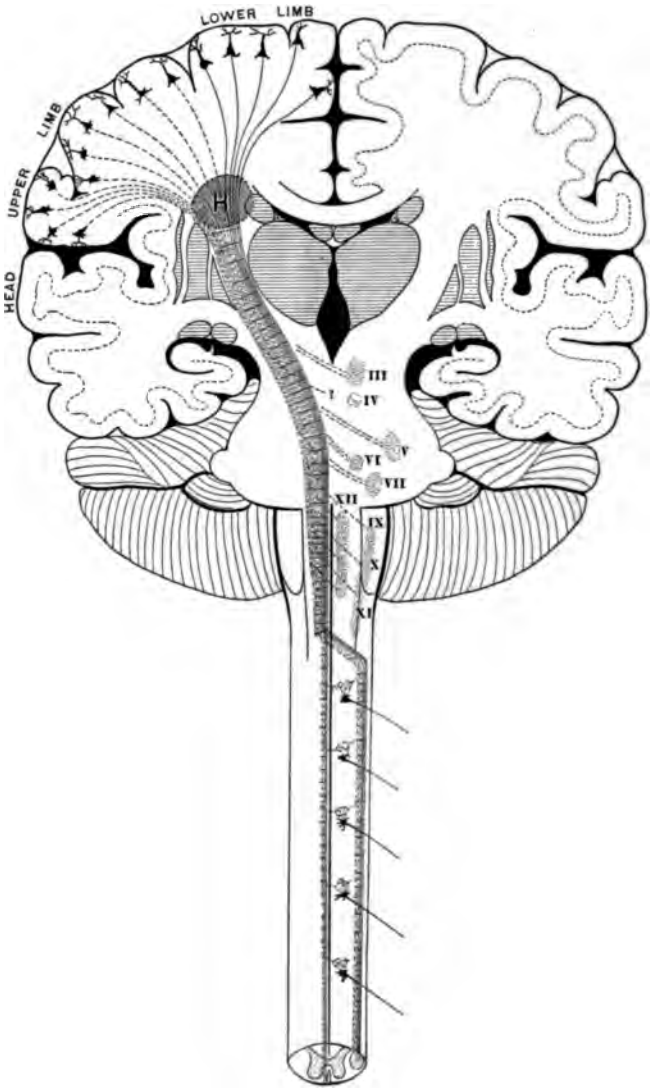
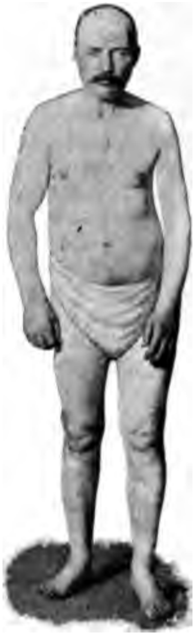


Diagram showing the secondary degeneration which occurs, a cerebral hemorrhage or softening, and which follows the course of the motor tracts into the spinal cord. H, site of lesion. The continuous lines are fibres going to the legs, the dotted are those going to arms and motor cranial nerves. (Modified from VAN GEHUCHTEN.)

ing cases of chronic hemiplegia that it is the extensor muscles of the toes and leg and thigh, also the extensors of the fingers, hand, arm, and upper arm which are more involved.

The paralysis in both arm and leg is made very much more marked by the supervention of what is known as secondary rigidity and contractures. Both the rigidity of the muscles and the contractures are due to the gradual degeneration of the voluntary motor tract in the brain and of the direct and crossed pyramidal tracts in the cord (Fig. 116). The effect of this degeneration is to lessen the inhibitory activities of the higher cerebral centres, and as a result, an excessive amount of action is brought to play upon the flexor groups. As time goes on, what was at first simply stiffness and rigidity in the affected parts becomes a fixed condition of contraction, or, as it is more commonly called, contracture. These contractures affect, as I have said, the flexor groups, causing the toes to be drawn down, the heel to be elevated, and the foot extended, the leg to be flexed slightly upon the thigh and the thigh slightly upon the trunk. In the arm the same process causes a tendency of the fingers to close upon the palms, of the wrist and forearm to be flexed, and of the upper arm to be held down next to the trunk. There is finally produced a condition of paralysis and contracture which gives to the attitude and gait of the patient a characteristic appearance (see Fig. 117). He walks slowly, swinging the toe of the paralyzed side out, so that it scrapes the floor, forming the arc of a circle as he goes along. The arm is held to the side, the hands are closed, the body is also slightly bent forward, and the position may be considered as one of very greatly exaggerated senility. The muscles of the trunk are very slightly affected in hemiplegia, although in the early stages some disturbance may be noticed. This is due to the physiological law that *all muscles which act synchronously upon symmetrical parts are innervated by each side of the brain.* Thus the respiratory muscles of the chest and abdomen are innervated on each side by centres in each half of the brain, and when the centre in one cerebral hemisphere is destroyed the function is assumed by the centre on the other side. It has been shown, however, by recent researches, that the pyramidal or motor tracts do not supply the opposite half of the body exclusively, but that some fibres go to the muscles of the same side. As the result of this there is always in hemiplegia a certain amount of impairment of power even upon the sound side.

FIG. 117.



Left hemiplegia involving face, arm, and leg.

Along with the paralysis, rigidity and contractures which I have described, there develops the condition of exaggeration of the deep reflexes. The patella tendon reflex, the triceps reflex, and the Achilles tendon reflex are exaggerated. Ankle clonus can be obtained in most cases. During the acute stage of apoplexy the superficial or skin reflexes are often abolished on the paralyzed side, but in the later stages they reappear, and become exaggerated also. Scratching the sole of the foot causes quick, jerking movements of the leg. Scratching the inner surface of the thigh produces a contraction of the cremaster, and so on.

In a good many cases there is a slight amount of hemianæsthesia during the early stage of cerebral apoplexies, but this almost invariably disappears in a few days, or weeks, and it is rare that any anæsthesia of the cutaneous or muscular senses is observed. Sometimes patients with hemiplegia suffer from pains in the affected parts. These pains are at times associated with cramp-like contractions of the arm or leg. In other cases they are burning, tearing, or neuralgic pains, and are due to the fact that the lesion irritates some part of the sensory tract or sensory centre in the brain. In hemiplegia of adults the affected side does not waste; the muscles, though but little used, do not become smaller to any notable extent. This is because the trophic centres for these muscles lie in the spinal cord—a part that is not affected in the disease. Once in a while, however, we find marked muscular atrophy in cerebral hemiplegia, though the cases are so rare that they may be considered freaks. When atrophy does occur it is, I think, usually due to the fact that the motor cortex is involved. In very bad cases of hemiplegia, where the patient remains so paralyzed that he is for the most part bed-ridden, some atrophy takes place, though this affects, as a rule, both sides. In these cases also there may develop arthropathies, that is to say, a knee-joint, or an ankle, or an elbow may become enlarged and show evidences of exudation and of proliferation of the osseous and connective tissues.

The patient's general bodily functions are usually carried on in a fairly normal manner. There is no great disturbance in digestion or in the activity of the kidneys. The sphincters of the bladder and rectum also perform their functions normally, although constipation is a frequent symptom, owing to the enforced inactivity of the patient. There is no doubt that the patient's general bodily vitality is somewhat lowered, and he is made somewhat more succeptible to the onset of infectious diseases or to the effects of renal, hepatic, or pulmonary troubles, which he may have had before the disease came on.

The mental condition of hemiplegic patients is a somewhat characteristic one. There is almost always a slight degree of mental impairment. This is shown in a weakness of memory and in an increase of emotionality, so that he laughs or cries easily, and becomes easily excited and irritable, or easily depressed. He may suffer from insomnia. In many cases, however, the patient is able to pursue his former vocations, if they do not greatly tax his mental or physical powers.

A certain proportion of hemiplegics are affected in the beginning with disturbances of speech which are known as aphasia. This aphasia is always associated with right hemiplegia in right-handed persons, and is due to lesions affecting the centres concerned in the faculty of language. It is not my purpose here to describe the different forms of aphasia. (See page 432.)

Among the rarer symptoms of the chronic stage of hemiplegia are certain spasmodic movements of the affected side of the body. These spasmodic disturbances occur much more frequently in connection with hemiplegia in children than in hemiplegia in adults, but they are occasionally seen in the latter class of patients. The most common of these disturbances is a cramp-like contraction of the fingers, hand, and arm, and also of the lower extremity. These contractions occur in a slow, clonic spasm, accompanied with much pain; they are in fact muscular cramps due to the excessive irritability of the motor nerves and muscles of the paralyzed part. Tremor is occasionally seen in the arm and leg. When present it is a tremor of the coarse type; that is to say, the rhythmical movements occur at the rate of five or six per minute, and the tremor is one that is usually increased upon voluntary movement of the part. It rarely affects the face, but at times there is a certain amount of twitching of the tongue and facial muscles. Inco-ordinated movements of the hemiplegic side are also observed; the patient, on attempting to place the hand in a certain position, or to move it in a certain definite manner, goes through awkward and irregular ataxic-like motions.

The condition is really an ataxia of the disordered limbs, and is due to an injury of the centres connected with the muscular memories. It is very rare indeed to find in adult hemiplegics anything in the nature of choreic movements. These, however, occur in infantile hemiplegia and even in hemiplegia occurring in youth; after the age of twenty, however, the hemiplegia is practically never associated with chorea. The same statements may be made with regard to the condition known as athetosis (*vide* Athetosis). Associated movements are sometimes observed in hemiplegics. By this is meant that when the patient tries to write, or makes some definite movements with the paralyzed arm or leg, there is at the same time a movement in the arm or leg of the sound side. The associated movements of the sound side resemble, in a measure, those of the affected side. The electrical irritability of the muscles in hemiplegia is at first somewhat increased; later it is diminished, but there are no qualitative changes in the ordinary types of the disease.

Mirror writing is a phenomenon sometimes seen in hemiplegics, though more common in young children. A right-handed person who has right hemiplegia attempts to write with the left hand and finds that he almost unconsciously writes from right to left, and reverses the letters so that in order to read them the sheet must be held before a mirror.

DIFFERENT TYPES OF HEMIPLEGIA. *Leg Sensory Type.* In the preceding remarks I have described the common type of hemiplegia following an intra-cranial hemorrhage. There are certain forms which differ from this type, and which merit some special attention. In one form the paralysis affects the leg much more than the arm or face. The patient in these cases, after the attack, gradually recovers the use of the arm to a very considerable extent, and the face becomes entirely free from any trace of paralysis. The leg, however, remains very much weakened, so that the patient is unable to walk, except with slowness and difficulty. Rigidity and contractures make the leg additionally clumsy and useless. In these cases there is often a hemiataxia with or without a considerable anæsthesia of the skin of the affected leg, and even of the arm, and sometimes there is also a hemianopsia and aphasia. Hemiplegias of this leg type are due to the fact that the lesion is situated more posteriorly, involving especially the leg fibres of the internal capsule, and some of the sensory tract. It is a comparatively rare type, and is due to a lesion of the lenticulo-optic arteries.

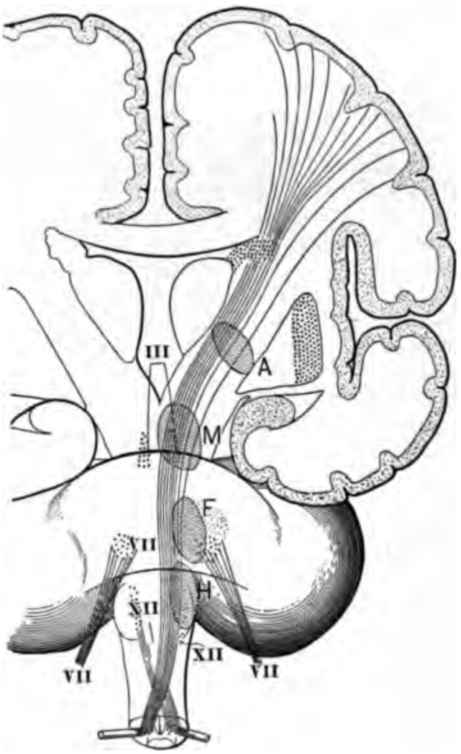
Arm Type. In another form of hemiplegia one sees the arm especially affected. In these cases the patient walks fairly well, but the arm is almost entirely powerless, the fingers are tightly flexed in the palm, and can with difficulty be extended. The face is perhaps somewhat affected also, and if the hemiplegia is upon the right side a motor aphasia, often very complete, accompanies the condition. Here the lesion involves the inferior anterior frontal artery.

Hemiataxic-aphasic Type. In another class of cases the hemiplegia is very slight, and can perhaps be detected, after a few weeks, only by careful examination, but there is with it a disturbance of the faculty of language, if the lesion is on the left side. There is often a considerable degree of hemiataxia and hemianæsthesia, and there may be also hemianopsia. The patients in these cases are usually unable to read, or perhaps unable to understand spoken language; they are blind on one half of each eye, on the side corresponding to the paralysis, and they use their arms and legs in a clumsy and awkward manner, due to the anæsthesia of the muscular sense.

Alternate or Crossed Hemiplegia. This name is given to cases in which the paralysis affects the arm and leg on one side and a cranial nerve or nerves on the opposite side. Alternate hemiplegia is due usually to acute softening, and this in turn to syphilitic disease at the base of the brain. (See also Chapter XXIV.) The lesion is situated in the crus, pons or medulla.

When the paralysis involves the arm and leg on one side and the third cranial nerve on the other, the lesion is in the crus cerebri, near its junction with the pons. This is the most common type. When there is paralysis of the fifth (trigeminal) nerve on one side, with hemiplegia on the other, the lesion is in the pons on one side, and near the ventral portion. When there is paralysis of the seventh (facial) nerve on one side, with hemiplegia of the other, the lesion lies at the junction of the pons and medulla; usually, the sixth and eighth cranial nerves are also involved.

FIG. 118.



Showing the mechanism of alternate hemiplegias. A lesion at A causes complete hemiplegia by destroying the motor tract. One at M causes paralysis of third cranial nerve (motor oculi) by destroying its nucleus or root on same side, and paralysis of arm and leg on opposite side. A lesion at F causes facial palsy on same side, hemiplegia on opposite side. In a lesion at H the hypoglossus would be affected on one side, with hemiplegia on the other. (Modified from EDINGER).

The mechanism of this hemiplegia is shown in the accompanying diagram. (Fig. 118.)

Pathology and Pathological Anatomy. The morbid condition underlying intra-cranial hemorrhages which are not due to trauma is invariably disease of the bloodvessels of the brain, and in most cases disease of the arteries. It is impossible to rupture a healthy artery by any ordinary physiological or pathological disturbance; consequently, the fundamental facts concerning the pathology of cerebral hemorrhage are those which pertain to arterial disease. An immense amount of study and laborious investigation have been put upon this subject, but it has not yet been entirely and satisfactorily cleared up. It is generally admitted that the cerebral arteries are subjected to three different morbid conditions: one of these is that known as fatty degeneration; the second is that known as a degenerative arteritis, and the third that of atheroma—a condition which really should be classed along with degenerative arteritis. In addition to this, there probably also exists at times certain congenital weaknesses or deficiencies in the coats of the arteries or in their size which lead to their occlusion or rupture. The fatty degeneration of the arteries of the brain affects, in an important way, at least, only the small arterioles. It causes a weakening of the walls at certain points, and in consequence of this a rupture occurs. Such fatty degeneration takes place in marasmic conditions, in leucocythæmia, scurvy, purpura, and it probably is caused also by the infectious fevers. The toxins developed by these diseases circulating in the blood appear at times to cause a fatty degeneration and softening of certain parts of the arterial walls. In septic conditions also the microbe itself may be carried into the brain and deposited in some small arteriole, causing there acute inflammatory destruction of the vessel. Hence we find that cerebral hemorrhages occur in connection with all the conditions which I have just mentioned. Atheromatous degeneration affects only the larger vessels, and chiefly those at the base of the brain; it is found in from one-eighth to one-fifth of the cases of cerebral hemorrhage. The atheromatous process lessens the elasticity of the arterial walls, and probably indirectly leads to cerebral hemorrhage. It is not, however, uniformly present, and an extensive atheroma of the cerebral arteries is known to be compatible with a long life. Arteritis in its various forms is the most frequent cause of the so-called idiopathic hemorrhages of the brain. There is still much discussion as to the exact nature of this process. While some look upon it as being originally a peri-arteritis, others as being originally an endarteritis, still others think that the process begins in the middle coats of the vessels. My own conviction, from a study of the subject and from an examination of specimens, is that the original process, in most cases, at least, is located in the intima, that this coat of the vessel is destroyed and the arterial wall is thus weakened. In consequence of the strain and bulging, there is a proliferation of connective tissue of the outer coat and an apparent peri-arteritis. Before this peri-arteritis occurs, however, the vessel undergoes a certain amount of fusiform or saccular dilatation in some cases, and these dilatations are known as miliary aneurisms. Their importance in connection with cerebral hemorrhage was first demonstrated by Charcot and Bouchard in 1872. It must be confessed that there have been very few thorough and systematic investigations subsequent to those of these authors, and that their results have been, perhaps, too uniformly accepted. While miliary aneurism undoubtedly is a factor, and an important one, in cerebral hemorrhage, I feel sure that it is not always the direct cause of it, and that sometimes vessels rupture without any notable preliminary dilatation. The small, or miliary aneurisms, range in size from one-fifth to one millimetre in diameter. They are usually not very numerous, but there may be as many as one hundred in the brain. They occur almost exclusively during the degenerative period of life.

Along with the causes of intra-cranial hemorrhage that I have already mentioned there are certain other rarer conditions which should be alluded to. Thus it may happen that in the case of a tumor of the brain, especially a sarcoma, bloodvessels rupture and hemorrhage occur. In abscess of the brain a cerebral artery or vein is sometimes opened and the patient dies from a hemorrhage into the cavity of the abscess.[1] Hemorrhages also occur sometimes secondarily to embolism or thrombosis. The sudden blocking up of an artery causes a necrosis of the parts supplied by it. There follows a collateral hyperæmia, then a congestion of the parts about the necrosed area, small hemorrhages take place or a large vessel may rupture.

In syphilitic inflammation of the arteries of the brain there may be so much arterial disease that one of the vessels is weakened and is ruptured. More commonly in syphilitic disease of the brain and of its arteries thrombosis is the result.

The large majority of hemorrhages of the cortical (pial) arteries are due to trauma. Idiopathic hemorrhages, if they occur, are always small and of slight importance, while traumatic hemorrhages are also apt to be small and accompanied with lacerations of the tissue, which are more important than the hemorrhage. Hemorrhages into the arachnoid cavity, which result in hematoma of the dura, are considered elsewhere. (See p. 351).

Cerebral hemorrhages occur with very different frequency in different parts of the brain. I have already dwelt upon this point, however, in connection with the symptomatology, and I showed there that the parts most frequently involved were those supplied by the central arteries given off by the middle cerebral. The order as ordinarily given of the parts affected is: the central ganglia, then the centrum ovale, then the cortex, cerebellum, and pons. The medulla oblongata, crura cerebri, and corpus callosum are almost always spared. In traumatic hemorrhages the most frequent part is that which is most exposed to injury, namely the convexity and the under surfaces of the brain. Cortical hemorrhages are, as already stated, generally small, and may be confined to the subarachnoid cavity or break through into the arachnoid. Ventricular hemorrhages are almost always secondary to a rupture in the neighborhood, in the basal ganglia, and it is the lateral ventricles that first and oftenest receive the effusion. Pons hemorrhages usually occur in the median line, and are, as a rule, small. Cerebellar hemorrhages are oftenest due to rupture of the superior cerebellar artery. They are apt to be large and to cleave their way externally, breaking into the fourth ventricle.

After a hemorrhage takes place, if life is preserved, certain reparatory changes occur. These consist of coagulation of the blood, which in a few days becomes softened, and in a week or two is absorbed. Then there is the formation of a fibrinous wall about the sac, and this occurs from the seventh to the ninth day. After this the fibrinous wall becomes organized, and a cyst with transparent fluid contents is developed. Sometimes fibrous trabeculæ run through it. This cyst may be formed by the twentieth to the thirtieth day. Contraction of the cyst wall takes place by the fortieth day, and, finally, if the hemorrhage was small, the cyst may close, leaving nothing but a cicatrix. This latter, however, occurs only in quite small hemorrhages. Finally, a very important pathological change takes place in connection with the destruction of brain tissue by the clot, and this is known as secondary degeneration. Owing to the fact that hemorrhages so frequently occur in the neighborhood of the internal capsule, the voluntary motor tract which carries impulses from the cortex of the brain to the motor cells of the spinal cord is more or less completely cut in two. This motor tract is really made up of the nerve-cell processes (neuraxons), starting in the cortical motor area, and when these processes are cut off their peripheral portions die throughout their whole extent; consequently, within a few days (from the tenth to the fourteenth) it is found that the whole of the motor fibres below the lesion, extending down into the spinal cord, begin to undergo a degenerative soften-

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[1] A case of this kind was reported by the writer, Journ. of Nervous and Mental Diseases, July, 1889.

ing. The fibres ultimately are destroyed and disappear, and in their place connective tissue is developed. Hence, from the seat of the lesion, passing down through the crura cerebri and the pons and medulla and lateral and anterior median columns of the cord there runs a strip of connective tissue instead of normal nerve fibres. This process is called a secondary degeneration, and its existence is the cause of many of the serious symptoms that are seen in the chronic stage following the apoplectic stroke.

Prognosis. The prognosis of intra-cranial hemorrhage depends a great deal on the seat and size of the lesion. Taking intra-cranial hemorrhages as a whole it is my experience that one-half to two-thirds recover from the first attack of hemorrhage, less than one-half of the remainder recover from a second attack, and very few, indeed, recover from a third; although I have known persons to have as many as five successive attacks, dying in the last. Apoplexies in which there is a very profound loss of consciousness, lasting for at least three days, are usually fatal. If the temperature, instead of falling on the second or third day, begins to rise, the outlook is uniformly unfavorable. The appearance of Cheyne-Stokes respiration is an extremely bad sign, although I have seen a case in which recovery took place after a free venesection. When any symptoms develop during the attack which indicate involvement of the medulla the prognosis is most unfavorable, and the same may be said of hemorrhages involving the tubercula quadrigemina and causing ocular paralysis. When a case of apoplexy which is apparently doing well suddenly develops coma or convulsions, the outlook is most unfavorable, for it indicates the extension of the hemorrhage. Cases in which the hemiplegia is very complete, so that the patient can move neither hand nor foot, are most serious, though not necessarily fatal. On the other hand, when the attack is not associated with loss of consciousness nor rise of temperature to any marked degree, the outlook is very good. The presence of Bright's disease is of bad omen, and if pneumonia develops the patient dies. As for the final issue of the disease, it is difficult to lay down positive rules. In many cases, cerebral apoplexy seems to put a check upon the activity of the patient, enforcing a quiet and regular life, and tending even to prolong existence. It may be thus considered almost a conservative process. Many cases of hemiplegia live for five or ten or more years. The prognosis as to the recovery from the paralysis also varies very much. As a rule, recovery begins to take place within a fortnight, and it continues up to the end of a year. Rapid improvement, however, ceases at the end of a couple of months, and after that time progress, if it takes place, is very slow.

Diagnosis Attacks of intra-cranial hemorrhage must be distinguished from acute softening, from coma due to alcohol, opium, chloral, uræmia, diabetes, the conditions of syncope and shock, the result of injury, and finally from epileptic coma. The diagnosis of intra-cranial hemorrhage from acute softening due to thrombosis or embolism will be discussed later. Alcoholic coma is distinguished by the history of the case, the odor of alcohol in the breath, the equality of the pupils, the absence of any evidence of hemiplegia, and the absence of a slow tense pulse, suffused face, and a puffing respiration. In alcoholic coma there is no unilateral disturbance of temperature, and there is no fall of temperature. Coma from opium poisoning is distinguished by the pin-point contraction of the pupils, the slow respiration, the congested condition of the skin, and the even bilateral temperature; also by the history of the case and the examination of the stomach-contents when that is possible. In uræmic coma there is usually a history of Bright's disease and often a history of preliminary uræmic twitchings or convulsions; there is the peculiar physiognomy of the patient with chronic Bright's disease, the thickened arteries, the presence of albumin and casts in the urine.

The pupils are usually even, moderately contracted, respiration is not as a rule stertorous, and the patient's coma is usually not extremely profound. In epilepsy there is a history of preliminary convulsions, there is often a condition of rigidity, there is also frequently a frothy red mucus in the mouth due to a bitten tongue, the pupils are dilated, and there is an absence of any evidence of paralysis. Sometimes apoplexy begins with convulsions, and then the apoplectic coma cannot be distinguished easily from epileptic coma except by the evidence of the paralysis.

When a person is recognized as having an apoplectic seizure, the diagnosis between hemorrhage or plugging of an artery has to be made.

In attempting to solve this problem, which is often one of extreme importance, many factors have to be taken into consideration, and a positive conclusion cannot always be reached. Still if one sees the patient during or soon after the attack I believe that one need rarely make a mistake.

Practically the distinction lies most frequently between acute softening from thrombosis and cerebral hemorrhage. Cerebral hemorrhage occurs most frequently in the middle periods of life, between the ages of thirty and fifty. After the age of sixty-five or seventy and before the age of thirty and thirty-five we should expect softenings from senile or syphilitic arteritis. There is in hemorrhage often a history of arterial disease, that is to say, of gout or chronic alcoholism, or Bright's disease. We have also sometimes a hereditary history of cerebral hemorrhage which may help us in diagnosis. Hemorrhage has, as a rule, few marked premonitory symptoms; the patient may even be feeling unusually well at the time of his attack, and a history of this condition gives rise to a strong presumption of vascular rupture. Cerebral hemorrhage is sometimes, though not always, the result of a sudden exertion, whereas thrombosis rarely occurs under such a condition. Hemorrhages occur more often in the morning or evening, and they do not often occur during sleep. When, therefore, a person wakes up in the morning and finds that he has a hemiplegia we may pretty safely assume that it is due to an acute softening. Cerebral hemorrhages come on suddenly and usually with loss of consciousness. The flushed face, the slow hard pulse, the throbbing carotids, and other evidences of intense cerebral congestion and overaction of the heart, all point toward hemorrhage. On the other hand, in thin, anæmic and weakened individuals the occurrence of a sudden hemiplegia would be more likely to suggest an acute softening. In cerebral hemorrhage consciousness is more frequently and profoundly lost, while in acute softening the patient may retain consciousness or be only semi-comatose. An inequality of the pupils, indicating an unequal pressure in the brain, suggests hemorrhage rather than softening. Vomiting indicates hemorrhage rather than softening; but convulsions may occur in either state.

In cerebral hemorrhage, if it is at all large, and there is decided shock, with loss of consciousness, the temperature in the rectum within a few hours after the attack sometimes falls two or three degrees. After twelve or twenty-four hours the temperature in cerebral hemorrhage is apt to be a little elevated, perhaps about one degree, and usually it is a degree higher on the paralyzed side; on the other hand, in acute softenings the temperature of the body is very slightly affected; it is equal or nearly so on the two sides, and practically normal in the rectum. In hemorrhages also the temperature is more apt to rise a little on the second or third day, while in acute softening such change does not occur unless the softening is very great, or affects the pons, or is associated with some septic process. In cerebral hemorrhage one finds more evidences, as a rule, of arterial disease than in embolism, but rather less than in thrombosis, unless the patient be a syphilitic. A history of syphilis, especially of an infection occurring within three or four years, suggests a thrombosis and softening rather than a hemorrhage, although sometimes a cerebral hemorrhage does occur as a result of syphilis.

Finally a quicker return of consciousness and a more rapid progress toward recovery indicate softening rather than hemorrhage. Hemorrhages are more serious and more fatal than softenings. Embolism practically never occurs without a serious and generally an active form of endocarditis being present. It does, however, occur after pregnancy and in certain cases of sepsis. When, therefore, a person has an abscess in any part of the body or an acute endocarditis the possibility of embolism may be considered great. The age of the patient is also a help. Embolism rarely occurs in the aged, and is most frequent in the earlier periods of life, at least before the degenerative changes have set in. The history of syphilis would lead one to make a diagnosis of thrombosis or hemorrhage rather than embolism. Embolism occurs more often in women, and is rare in children. A profound anæmia would lead one to expect an embolism rather than thrombosis, though this factor has not a very great weight. In embolism one rarely gets a history of any premonitory symptoms, while in thrombosis a previous history of headaches, mental confusion, vertigo, paræsthesia, and transitory paralysis is often found. The onset in embolism is always sudden; it begins often with some motor symptoms, that is, a slight convulsive twitching or a slight hemiplegia followed by complete paralysis. In thrombosis the symptoms come on gradually.

Treatment. The treatment of the condition depends naturally very much upon the fact whether or not the physician has been able to make a positive diagnosis of intra-cranial hemorrhage. If he is satisfied upon this point prompt and vigorous treatment may be of great use. The patient, if seen early, should be placed in bed, with the head and chest well raised, and the clothes loosened, so that the circulation from the head is not impeded. The extremities should be swathed in hot cloths wrung out in mustard water; an ice-bag should be placed upon the head; a drop or two of croton oil mixed with a little sweet oil should be placed upon the tongue. I believe that it is a wise practice in cerebral hemorrhage to attempt compression of the carotid upon the affected side, provided the patient is seen within two or three hours of the attack and the symptoms do not show positively that there is a rupture into the lateral ventricles. If this has occurred, carotid compression can avail little. Compression should be continued for three-quarters to an hour. The suggestion which has been made to tie the carotid is not to be recommended. To relieve the stertor, turn the patient on the paralyzed side and see that the tongue is drawn forward. In plethoric patients with a strong heart-action, congestion of the face and plain evidences of great cerebral hyperæmia and over-action of the heart, bleeding to the amount of ten or twelve ounces is advisable, and I have seen some desperate cases apparently brought up by this measure. Contrary to what might be expected, bleeding, if not done to great excess, does not necessarily injure a person with thrombosis. I base this statement on cases in which thrombosis has occurred, bleeding has been performed, and symptoms of improvement have appeared. I would not have it understood, however, that I consider it at all advisable to bleed in such conditions, but rather the contrary. If the patient's condition is such that one does not feel justified in bleeding, a somewhat similar effect can be produced by giving two or three drops of tincture of aconite every half hour until the evidences of lowered arterial tension and weaker heart-action are obtained. The tincture of veratrum viride is recommended for the same purpose. Duquesnel's aconitia in doses of $\frac{1}{200}$ of a grain, repeated in three hours, may be used instead of tincture of aconite, which is not always a trustworthy preparation. The administration of ergot has been advised, but I do not believe in its utility, nor have I seen any results from bromides. The preparations of hydrastis are recommended strongly as useful in hemorrhage; but their value in cerebral hemorrhage has, so far as I know, not yet been determined. In some cases of cerebral hemorrhage the shock and the weak condition of the patient are such that there is evidence of heart failure, and if this be present then we must not hesitate to use small amounts of alcohol, digitalis, and strychnine.

After the first few hours any attempt to control the hemorrhage is futile; it has by this time done its work. There may, however, sometimes be a second rupture of an artery or a breakage into the ventricles, and in order to avoid this the greatest care must be taken to keep the patient extremely quiet. If he is restless and delirious, bromide and chloral, or morphine, should be given, preferably the former. On the second or third day it is advisable, in suspected syphilis, to give small doses of iodide of potassium; that is to say, doses of two or three grains every two hours. If the patient gradually recovers consciousness all medication may be suspended except the iodide of potash and such measures as may be indicated in accordance with the development of the symptoms. At the end of ten days or a fortnight, if the patient has satisfactorily improved, one may begin cautiously to apply the faradic current to the paralyzed limbs. If the paralysis is slight this need not be done so soon; but if complete, there is some benefit in beginning electrical applications early, provided they are made very short and very mild; that is to say, each group of muscles should be made to contract three or four times by means of the current. If the patient does not progress favorably, if he continues in a state of partial unconsciousness and develops a slight fever, there is little to be done except to keep the emunctories open; the skin should be bathed with warm water, the kidneys should be acted upon by sweet spirits of nitre and iodide of potassium, and the bowels should be kept regular. A milk diet is to be recommended during this time. Special care should be taken lest pneumonia should develop. The mouth should be kept thoroughly disinfected, and the patient should be prevented from lying in the same position. Thorough cleanliness is necessary in order to prevent the development of bed-sores, for in some cases the patients void the urine and feces in the bed.

When the chronic stage has been reached the medical treatment will have to vary very much in accordance with the general health of the patient. In some cases where there is evidence of syphilis large doses of iodide of potash, with occasional courses of mercury, must be given. If the patient is gouty and has some renal complications the use of tincture of iron and of the iodides, or the acetate of potash and digitalis, may be indicated. Strychnine is of some use, not because it directly affects the paralyzed limbs, but because it strengthens the heart and is a good general tonic.

For the first year after his stroke the patient should have courses of electrical treatment, massage, hydrotherapy, and mechanical treatment of various kinds. An electrical treatment should not last more than six weeks, and, as a rule, three applications a week are sufficient. The faradic battery with the current of high tension is a useful one, but it seems from my experience that the long sparks of the static machine give the most successful results. I do not know that any advantage is to be gained from the use of galvanism. After a course of electrical treatment the patient may rest a week, and then have a course of massage daily for a month. He should rest then, and may afterward begin the electricity again. In some instances lukewarm baths are useful auxiliaries. The patient should be placed in a bath of a temperature of 95°, and should be made to exercise his muscles while there for a period of about ten minutes daily. He should afterward be taken out and rubbed well with cool water and alcohol. In some patients the contractures of the affected limbs are very great, and the deformities resulting render the limbs almost useless. The fingers of the hand are particularly apt to be affected in this way. I have, in some instances, had the arm and the foot hyperextended and placed in plaster-of-Paris with fairly good results. It has seemed to me that if a patient began his treatment by steady attention to the prevention of the excessive contracture a good deal of it might be avoided. Some improvement in the hemiplegia may be expected for over a year; after that not much can be done, still in cases which have been not thoroughly treated in the first year, or in cases in which there has been neglect of mechanical treatment, help may be given even late in the disease.

ACUTE SOFTENING OF THE BRAIN. EMBOLISM, THROMBOSIS.

Acute softening of the brain is a condition caused by the plugging of a blood-vessel with an embolus or thrombus, and is characterized by a more or less sudden apoplectic seizure; the symptoms in the later stage resembling those that follow intra-cranial hemorrhage.

Etiology. Embolism occurs rather more often in women, thrombosis in men. Embolism is rare in children; it occurs oftenest between the ages of twenty and fifty, thrombosis between the ages of fifty and seventy. The most important predisposing factors in embolism are acute or recurrent endocarditis, infectious fevers, profound anæmia, pregnancy, and blood dyscrasiæ; in thrombosis, syphilitic, lead, or gouty arteritis, fatty heart, and blood dyscrasiæ. The same causes which lead to the arterial disease which produces cerebral hemorrhage also predispose to thrombosis, though in the latter condition atheroma plays the important part.

Symptoms. In embolism there are rarely any premonitory symptoms; the onset is sudden; it may begin with some convulsive twitchings, then follow hemiplegia and temporary loss of consciousness. Coma, however, is rarer than in hemorrhage, and if present is usually shorter. There is rarely vomiting, nor do we find the hard, pulsating arteries, flushed face, and severely stertorous breathing. The initial temperature changes are slight, but in a few days fever may develop.

In thrombosis premonitory symptoms are frequent. In syphilitic cases there are headaches and cranial nerve palsies. In other cases vertigo, temporary aphasia, transient hemiplegia, numbness of the hand and foot, and drowsiness may be present. The onset is more gradual; the hemiplegia slowly develops, taking several hours, perhaps, for its completion; meanwhile the patient gradually becomes comatose. The attack sometimes is rather sudden, with no loss of consciousness, and it may occur in sleep. The temperature often has a slight initial fall, followed by a rise, just as in hemorrhage. In both embolism and thrombosis the hemiplegia tends to improve very much in a few days or weeks unless the vessel obliterated is a large one.

The right side of the body is affected slightly more than the left, owing to the fact that the left middle cerebral is rather more easily reached by an embolus.

Acute softening may kill within twenty-four hours; but, as a rule, the patient survives the onset, and if he dies it is not for several weeks. After the acute stage is over the patient passes into the chronic stage, which resembles in nearly all respects that of hemorrhage. (Vide Hemorrhage.) After an acute softening, however, it is believed that there are more spastic symptoms and a greater tendency to mobile spasm. In embolism, owing to the youth and freedom from arterial disease, the mind is less affected; while in thrombosis the contrary is the case.

Prognosis. The prognosis as regards the attack is somewhat better than in hemorrhages as a rule. In embolism it is good as regards recurrence; in thrombosis, bad. The mental condition is better in embolism; usually worse in thrombosis. The recovery from attack is more complete in acute softening. After the chronic stage is reached, however, the prognosis is about the same in all forms.

Treatment. The treatment of the attacks consists essentially in rest and such attention to the bowels, kidneys, and heart as may be indicated. In thrombosis it is important to give heart stimulants and arterial depressants, and for this purpose I advise the use of alcohol, digitalis, or strophanthus with nitroglycerin. Iodide of potassium and mercury ought to be given if there is the slightest suspicion of syphilis. Later one should prescribe courses of the iodides and mercury and of strophanthus, nitroglycerin, strychnine, and such tonics as may be indicated. The symptomatic treatment of the chronic stage is the same as in hemorrhage:

Softening in the region of the frontal convolutions on the inner surface of the hemispheres as far as the calloso-marginal sulcus—due to obliteration of the trunk of the anterior cerebral artery.

Total softening of the territory of the artery of the fissure of Sylvius, including the corpus striatum—due to obliteration of the first two centimetres of this artery. Softening is found in all the convolutions named hereafter.

Total softening of the cortical areas supplied by the artery of the fissure of Sylvius, the corpus striatum excluded—due to occlusion of the artery just beyond the giving off of the arteries of the corpus striatum.

Partial softening in the area supplied by the arteries of the fissure of Sylvius—due to embolism of the inferior external frontal artery. The result is a softening of the island of Reil and of the third frontal convolution. If this is on the left side, aphasia results.

Softening in the posterior part of the second frontal convolution and in the first central convolution—obliteration of the anterior parietal artery.

Softening of both central convolutions and the Rolandic fissure, the anterior part of the first parietal convolution, and the island of Reil—due to closure of the median parietal artery.

Softening of the lower parietal convolution and the first temporal convolution as well as of the island of Reil—due to obliteration of the posterior parietal artery.

Softening in the area of the posterior cerebral artery, rarely complete, involves the inferior part of the occipital lobe and the tip of the occipital lobe.

The secondary changes after thrombosis resemble those after embolism; a thrombus, however, may lead to supplementary embolism through breaking off of a clot, and both conditions may cause a complicating cerebral hemorrhage.

Total softening of the corpus striatum, including the capsule, the lenticular and caudate nucleus and the anterior third of the thalamus—seat of thrombus in the beginning of the artery of the fissure of Sylvius.

Partial softening in the form of a cone whose apex lies in the anterior part of the lenticular nucleus, while the base, directed forward, involves the anterior two-thirds of the corpus striatum; the cone is formed by the anterior part of the nucleus caudatus, the internal capsule, and the third segment of the lenticular nucleus—the seat of the occlusion here lies in the lenticular striate artery.

Partial softening lying more posteriorly than the preceding, involving the postero-external part of the lenticular nucleus, the internal capsule, the anterior part of the thalamus, and the tail of the corpus striatum—the lesion involves the lenticular optic artery.

Partial softening of the thalamus opticus, the lesion ranges in size from a pea to a hazelnut—the vessels involved are the perforating arteries of the choroid plexus.

Softening of the frontal, parietal, and sphenoidal lobes—due to embolism at the point of bifurcation of the internal carotid, extension of the thrombus into the anterior cerebral artery as far as the anterior.

Pathology. The embolus or thrombus cuts off the blood supply from a certain area of brain tissue. In twenty-four hours this begins to soften. If the area is in the cortex it becomes red (red softening); if in the white and less vascular part, it is usually white with a few red punctate spots. The red softening gradually becomes yellow (yellow softening). The dead tissue softens and is absorbed, leaving a cicatrix or cyst. If the embolus contains infective microbes there may be a local encephalitis and abscess.

In thrombosis there are usually evidences of extensive atheroma or of syphilitic arteritis. In those instances in which the thrombosis is caused by the blood state, as in scurvy or after wasting fevers and a weak heart, little arterial change occurs. Atheroma affects chiefly the internal carotids and the large arteries at the base, viz., the middle, anterior, and posterior cerebrals and the basilar and vertebrals.

Thrombotic softenings occur oftenest in the corpora striata and optic thalamus, next in the pons and medulla. Thrombosis affects the vertebrals, basilar and the posterior cerebral arteries much oftener than do hemorrhages or embolism. An embolism may, however, plug up the basilar or the vertebral at its junction with the basilar. Embolism usually affects the middle cerebrals (75 per cent. of 79 cases, Pitt), and the two hemispheres are almost equally involved, there being only a small preponderance in favor of the left side. The cerebellum is practically not affected by embolism or thrombosis.

CHAPTER XVI.

FOCAL DISEASES OF THE BRAIN.
(CONTINUED.)

BY M. ALLEN STARR, M.D.

TUMOR OF THE BRAIN.

GENERAL CONSIDERATIONS. This subject is an important one to the neurologist, both because of the relative frequency of this disease as compared with other organic nervous diseases, not only in adults, but also in children, and also because of the possibility of its treatment by surgical means. Collections of cases of brain tumors have been published during the past fifteen years by Bernhardt[1], Steffan[2], Bramwell,[3] Mary Putnam Jacobi,[4] Mills and Lloyd,[5] Knapp,[6] and myself.[7] It is possible by the study of these collections of cases to make a fair estimate of the relative frequency of different varieties of tumor and of the various situations in the brain in which tumors grow, and also to estimate the probabilities of success in finding and removing a tumor. Without attaching too great importance to statistics on this subject and realizing that a complete study of a single case is of more value than many carelessly reported cases, I have selected as the basis of this article, from the collections already named, 600 tumors. These are tabulated in Table I, in which tumors in individuals below the age of twenty, chiefly children, are separated from those in adults, for the purpose of contrast. In this table no tumors are included which have been removed or in which surgical interference has been attempted. Yet during the past six years over 130 tumors have been operated upon.

The percentage of cases open to operation has been variously estimated by different authors, and their results are demonstrated in Table II.

From this table it is evident that about 7 per cent. of tumors can be removed. The various authors cited have made collections of cases with post-mortem records, and from a study of the condition found at autopsy have estimated the probability of success in the removal of the tumor found.

Etiology. Little is known regarding the cause of brain tumor, though in a certain small percentage of cases a history of a blow or fall on the head has been obtained. It is known that males are more subject to brain tumor than females, and persons of all ages are liable to brain tumor.

It is not my purpose in this chapter to cite special cases of brain tumor or to recount histories. But anyone interested in this subject is referred to the articles and works already cited, from which the general conclusions which are here combined have been drawn.

[1] Beiträge zur Symptomatologie und Diagnostik der Hirngeschwülste, 1881. Also yearly summary in Virchow's Jahresbericht, 1881 to 1895.

[2] Die Krankheiten des Gehirns im Kindesalter. Gerhardt's Handb. der Kinderkrankheiten, 1880, vol. v.

[3] Intra-cranial Tumours, 1888. Edinburgh Medical Journal, Dec., 1894.

[4] Reference Handbook of Medical Sciences, 1888.

[5] Pepper's System of Medicine, vol. v.

[6] Intra-cranial Growths, 1891.

[7] Medical News, Jan. 12, 1889 ; Intra-cranial Tumors, Keating's Cyclopædia of Children's Diseases, 1890, and Brain Surgery, 1893.

TABLE I.

(The first column are children's tumors; the second column adults' tumors.)

Situation.	Tuber-culous.		Sarco-matous.		Glioma-tous.		Glio-sar-comatous.		Cystic.		Carcino-matous.		Gumma-tous.		Other va-rieties.		Total.	
I. Cortex cerebri	13	9	1	46	6	19	...	8	...	...	1	19	...	13	...	12	21	127
II. Centrum ovale	6	2	5	7	1	11	1	4	15	...	1	8	1	...	5	4	35	51
III. Cerebral axis:																		
1. Basal ganglia and lateral ventricles	14	3	5	8	3	9	...	...	1	1	1	2	...	1	3	5	27	34
2. Corpora quadrigemina and crura cerebri	16	1	3	2	1	2	...	5	...	...	...	1	...	...	1	7	21	14
3 Pons	19	11	5	1	10	...	2	1	1	...	...	2	...	3	1	...	38	17
4. Medulla	2	...	...	...	...	1	...	...	1	...	2	...	...	...	1	...	6	2
5. Base	...	3	1	3	...	2	1	1	1	...	1	...	...	...	4	1	8	9
6. Fourth ventricle	1	...	1	1	1	...	...	...	...	...	1	2	...	...	1	1	5	4
IV. Cerebellum	47	8	10	13	15	8	1	6	9	...	3	...	...	...	11	10	96	45
V. Multiple tumors	34	4	3	5	...	2	...	...	2	1	...	2	1	3	3	1	43	17
	152	41	34	86	37	54	5	25	30	2	10	33	2	20	30	41	300	300

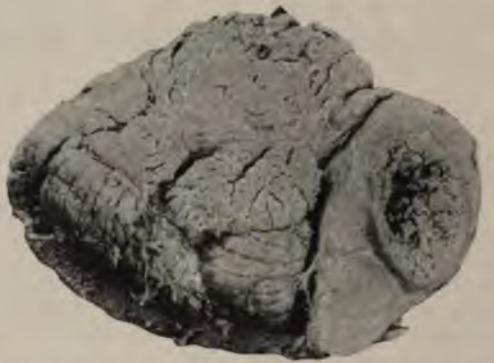
TABLE II.—PERCENTAGE OF BRAIN TUMORS REMOVABLE.

Author.	No. cases.	Operable.	Reference.
1. Mills and Lloyd	100	10	Pepper's System, 1886, vol. v.
2. Hale White	100	10	Guy's Hospital Rep , 1888.
3. Starr	300	16	Med. News, Jan. 12, 1889 (children).
4. Knapp	40	2	Intra-cranial Growths, 1891.
5. Gray	53	4	Sajous' Annual, 1891.
6. Gray	49	6	" " 1892.
7. Seydel	100	3	Verhand. Deut. Gesell. f. Chir., 1892.
8. Dana	29	5	Trans. N. Y. Acad. Med., Jan. 1893.
9. Starr	300	21	Brain Surgery, 1893 (adults).
10. Byrom Bramwell	50	3	Edinburgh Med. Journ., June, 1894.
	1121	80	

Pathology. *The Varieties of Brain Tumor.* Almost every variety of tumor known has been found within the brain. There are certain tumors, however, which are quite common, and others which are very rare. In the first class are tubercular, sarcomatous, gliomatous, glio-sarcomatous, cystic, carcinomatous, and gummy tumors. In the second class are fibromatous, angiomatous, myxomatous, osteomatous, and liptomatous tumors, also psammoma, cholesteatoma, and echinococcus cysts.

Tubercular tumors of the brain are the most common of all the forms of tumor. They occur with greatest frequency in childhood, being sometimes primary, but usually secondary to tuberculosis of other organs. In 20 per cent. of the cases recorded, tubercular tumors are multiple. This fact should not be forgotton in studying the symptoms of brain tumor and in diagnosticating the location of the tumor, for if the tumor is probably tubercular, and if the symptoms are not clearly explicable upon the theory of its location in one part of the brain, it is to be remembered that two or more different tumors in different locations may give rise to a great variety of symptoms. Tubercular tumors vary in size from a small collection of miliary tubercules lying in a mass of thickened pia mater up to a large solid circumscribed mass, with hard, cheesy, or broken-down granular centre and a distinct capsule. (See Figs. 119, 120, and 121). Not infrequently the tumor is surrounded by a tubercular infiltration both in the brain and its membranes. Sometimes irregularly-shaped deposits of tubercular tissues are found upon the base of the brain, in the meshes of the pia mater, compressing the cranial nerves.

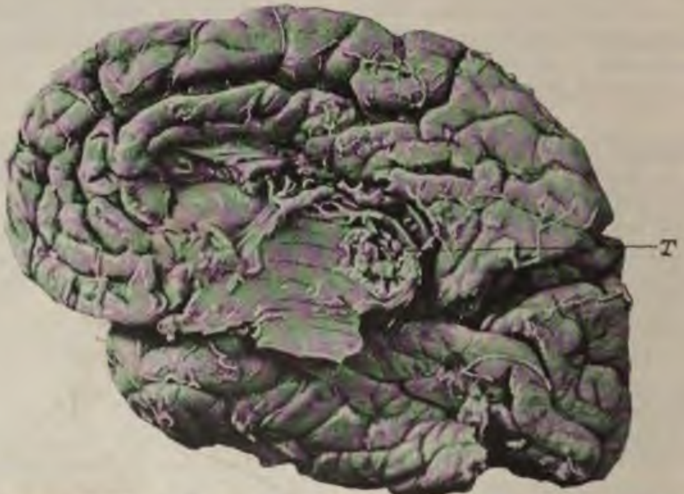
FIG. 119.



Tubercular tumor in the pons varolii with granular mass lying in a capsule. Sagittal section through pons and cerebellum.

Tubercular tumors may be found within the cerebral tissue at some distance from the surface, and no part of the brain can be said to be free from a liability to tubercular deposits. But the large majority of this variety of tumors is found in connection with the meninges and about the large vessels of the brain, so that they are more commonly discovered upon the surface and about the base than lying deep within the organ. When the tumor arises in the meninges meningeal thickening by tubercular deposits is quite

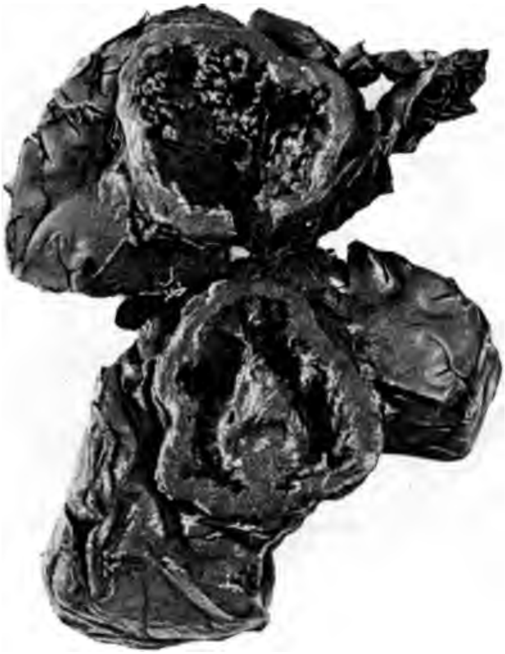
FIG. 120.



Tubercular tumor in right optic thalamus. Tubercular meningitis of the median surface of the hemisphere.

common. In a few cases the cranial bones have been eroded in the growth of the tumor. Tubercular tumors are sometimes found in the brain after death when no cerebral symptoms have been present during life.[1]

FIG. 121.



Section through cerebrum showing a large tubercular tumor with thick capsule and granular contents lying beneath the cortex in the centrum ovale of the frontal lobe.

For an account of the finer patholology of tumors the reader is referred to the text-books of pathology.

The important facts to be considered in making the diagnosis of tubercular tumors are (*a*) hereditary tendencies of the individual to tuberculosis; (*b*), exposure to such influences as are known to favor the development of the disease; (*c*), the history of symptoms of primary pulmonary, or intestinal, or joint, or glandular affections; and (*d*), the presence of local signs or general evidences of tubercular infection.

When cerebral symptoms develop in an individual with tubercular disease the possibility both of tubercular tumor and of tubercular meningitis should not be overlooked.

The question of operative interference when a tubercular tumor is diagnosticated has given rise to some discussion; von Bergmann[2] being opposed to such operations, and Horsley[3] being in their favor. It must be admitted that a permanent cure by operation is less likely to be achieved in the case of a tubercular tumor than in the case of a non-tubercular tumor, there being not only the danger of recurrence, but also the possibility of the existence of undetected tumors elsewhere in the brain, and the danger of the development of tubercular meningitis subsequently to the operation. An operation is capable of prolonging life, and hence should not be wholly condemned.

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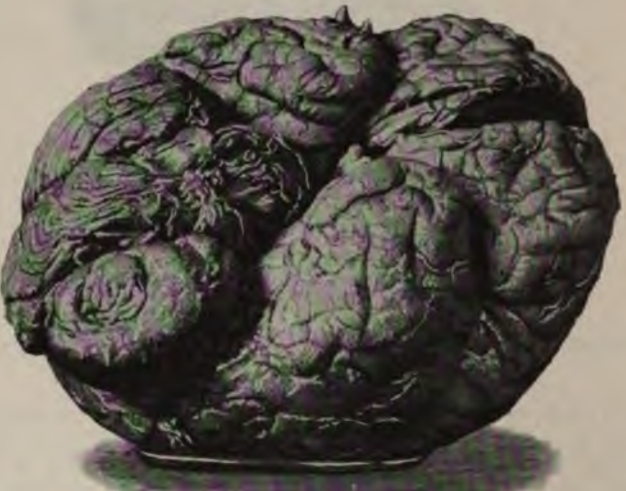
[1] Edinburgh Hospital Reports, vol. i. p. 420.
[2] Von Bergmann: Die Chirurgische Behandlung von Hirnkrankheiten, Berlin, 1881.
[3] Horsley: Trans. Internat. Med. Cong., 1890.

Sarcomatous tumors are next in frequency to tubercular. They are rarely secondary to sarcoma in other parts of the body, and they are only multiple when they appear in the form of melanotic nodules, which are rare in the brain. (Figs. 122 and 123.)

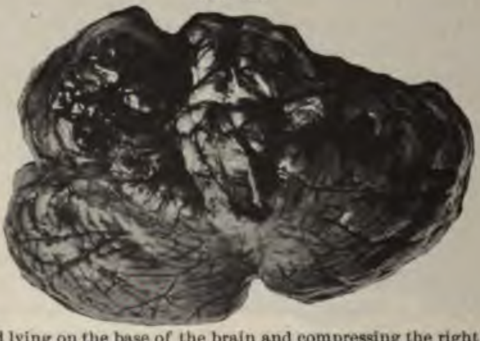
FIG. 122.



Sarcoma, encapsulated, with pedicle attached to the dura, lying in the posterior cranial fossa, and compressing the left hemisphere of the cerebellum.

Sarcoma is usually encapsulated, round or oval in shape, hard, approaching fibroma in consistency, and is easily separable from the brain tissue, which it compresses and indents more frequently than it infiltrates. Fig. 124 shows a sarcoma removed from the brain by McBurney.[1] Round-cell and spindle-

FIG. 123.



Sarcoma encapsulated lying on the base of the brain and compressing the right side of the pons and right hemisphere of the cerebellum.

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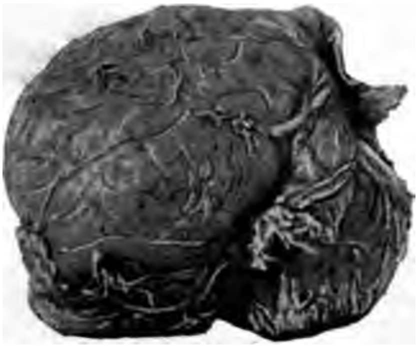
[1] Amer. Journ. Med. Sci., April, 1893.

cell sarcomata are more frequently met with than myxo-sarcomata or glio-sarcomata. These tumors grow rapidly and produce well-marked symptoms which do not vary very greatly in their intensity. They are not vascular, and hence the occurrence of apoplectic attacks during their growth is infrequent. It is not to be forgotten that a sarcomatous deposit in the meninges may occur, producing a thick layer of new tissue over an extensive surface of the cortex or base, and in such a case operative treatment is necessarily unsuccessful.[1] Occasionally a sarcoma has no defined limits, and infiltrates the tissue of the brain. Hence, sarcoma is not uniformly favorably for operation.

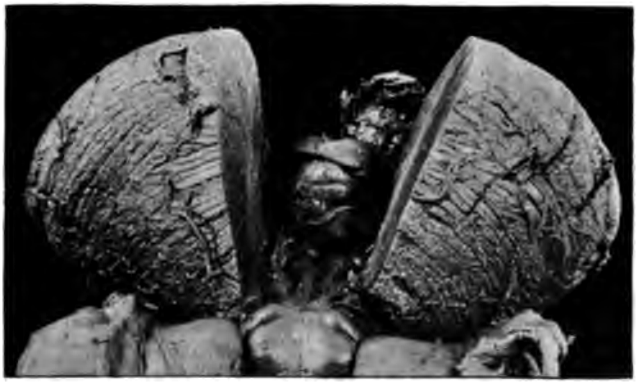
FIG. 124.



Sarcoma removed from the cortex of the frontal lobe. The capsule contained many vessels.

From the study of Table I it is evident that sarcoma may be found in any part of the brain, but it is more frequent in the cortex and in the cerebellum than in any other localities. It is the form of tumor most easily extracted from the skull, and the majority of the successful cases of removal of brain tumor have been sarcomata. (See Fig. 122, which shows a sarcoma lying on the cerebellum, which could have been removed very easily.)

FIG. 125.



A glioma of the pons and medulla infiltrating the tissues, but not destroying the tracts. The symptoms were chiefly cerebellar.

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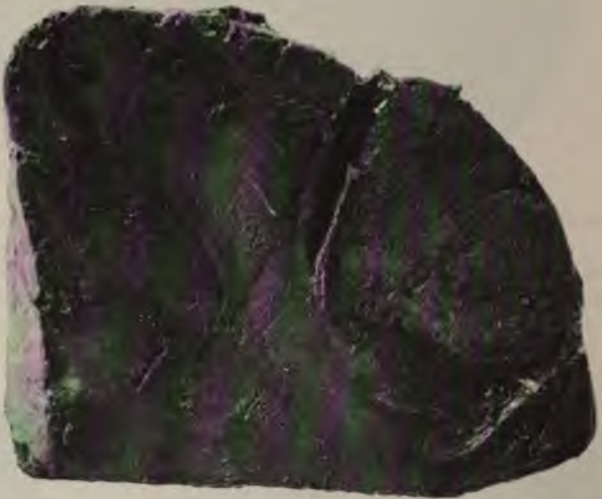
[1] Eskridge: Journ. Amer. Med. Assoc., Sept. 30, 1893.

Gliomatous tumors come third in the list. This tumor is a product of the neuroglia and presents the appearance of a connective tissue fibrillary network containing a greater or less number of large branching cells and many small nucleated cells.

Glioma of the brain is usually primary, but occasionally may develop secondarily to glioma of the retina.

Gliomata vary in density, but are usually softer than sarcomata, and are rarely well defined or separable from the brain tissue, even those which appear to have a definite limit, being found, on microscopic study, to be surrounded by a zone of gliomatous infiltration in the brain. The usual consistency of a glioma is about that of the brain substance. There is a tendency in all gliomatous tissues to undergo fatty degeneration, to break down and liquefy, so that cavities filled with clear fluid of a straw color or more sharply-defined cysts are very frequently found in and about a gliomatous tumor, and, in fact, some authors have maintained that all cysts found in the brain, not the relics of previous hemorrhages or of embolic softening, are due to gliomatous formations. A similar tendency to the formation of cavities within gliomatous tissue is manifested in the spinal cord in the disease syringomyelia.

FIG. 126.



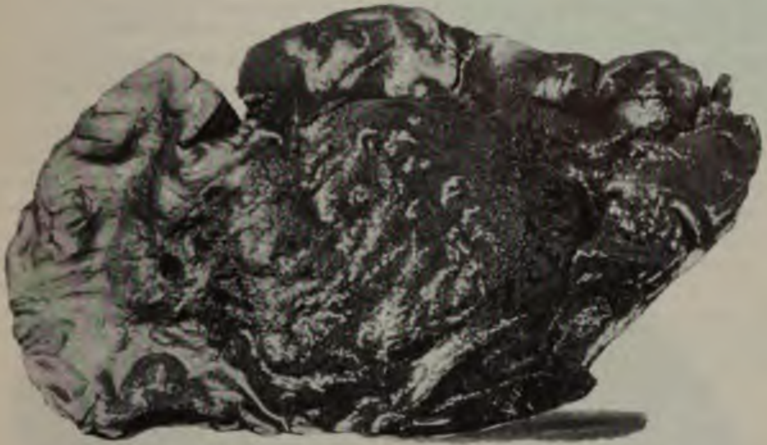
Glioma of the motor area infiltrating the cortex and centrum ovale, without distinct capsule, excepting at one part.

Glioma may appear in any part of the brain, but is somewhat more frequent in the white substance of the brain than in the gray matter. As a glioma grows it destroys the brain tissue, its branching cells surrounding and annihilating both nerve-cells and nerve-fibres. It does not compress the brain as a sarcoma does.

Glioma is very vascular, and hence the symptoms which it produces are somewhat variable in intensity, and, in case the delicate vessels within it rupture, apoplectiform attacks occur.

Glio-sarcoma is a variety of tumor not very frequent, partaking of the nature both of glioma and of sarcoma, commonly accompanied by cystic cavities. Like glioma, the tumor cells are infiltrated through the brain tissues; but, like sarcoma, its constituents are spindle and round cells, and its density is considerable. It is rarely encapsulated, and, like glioma, is much more difficult of removal than sarcoma.

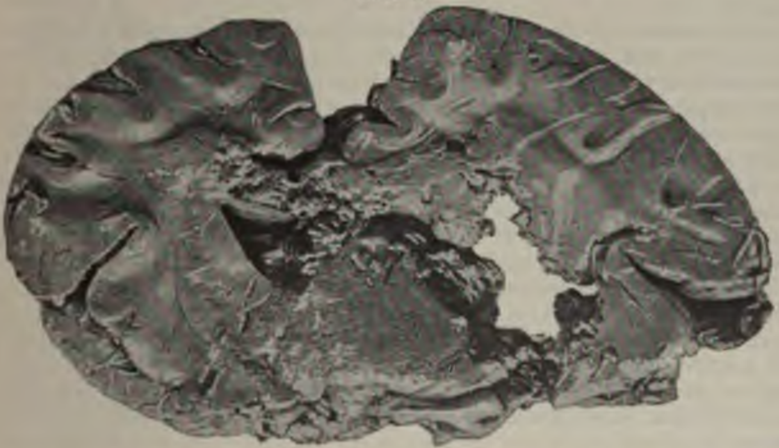
FIG. 127.



Glio-sarcoma of frontal lobes and corpus callosum. (FRANCIS.)

It is questionable whether the origin of a glio-sarcoma is the development of sarcomatous cells infiltrating the brain, and causing an irritation and proliferation of the neuroglia, or whether there is a development of hard, round connective tissue elements, sarcomatous in character, in the midst of the original glioma.

FIG. 128.



Glio-sarcoma of corpus callosum, invading the frontal lobes chiefly on the right side, where disintegration of the centrum ovale was produced. (FRANCIS.)

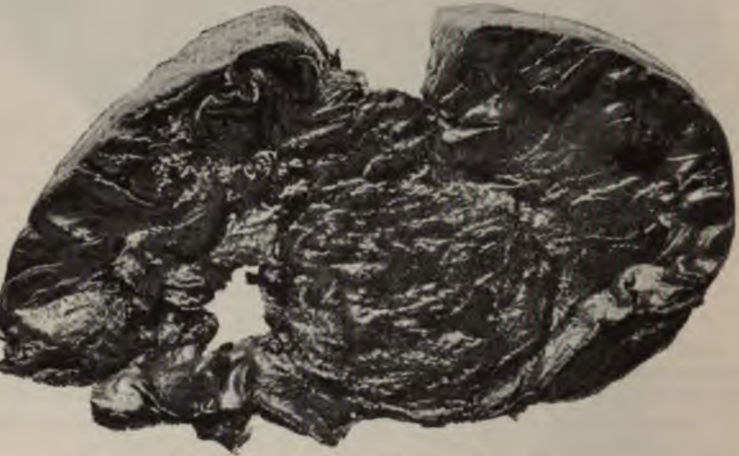
The symptoms are more closely like those of glioma than of sarcoma, the same variability of intensity being observed.

Carcinoma of the brain is usually secondary to carcinoma in other parts of the body, either of the orbit or retina, or of the internal organs, or of the breast.

Carcinoma is a variety seldom found in children, and usually develops in adults above the age of fifty. In cases in which it has occurred in children it has been from direct extension of the disease from the orbit. It may occur as the primary development of carcinoma in the body, giving rise to secondary infection elsewhere, but this also is rare.

Carcinoma grows rapidly in the brain, is not sharply defined, rarely has a capsule, is decidedly vascular, and, of all brain tumors, the one least open to operative treatment.

FIG. 129.



Glio-sarcoma of corpus callosum. (FRANCIS.)

Cystic tumors of the brain may arise either in connection with glioma and glio-sarcoma, as already described, or independently, as the result of parasitic infection. Hydatid cysts, echinococcus, and cysticercus are much more frequently recorded in German and Australian medical journals than in this country.

Kuchenmeister has made a collection of 88 cases of this variety, in all but 13 of which multiple cysts were found. Of these 49 involved the membranes, 41 were in the cortex, 19 in the white substance, 19 in the cerebellum, 18 in the ventricles, 17 in the basal ganglia, and a few in the corpora quadrigemina, pons, and medulla. These are not included in the tables, as it would give an appearance of undue frequency of this form of tumor. These parasitic cysts grow slowly, and produce compression of the brain tissue, but do not destroy it. The symptoms which they produce are very rarely so well marked as to enable a diagnosis to be reached, but in cases in which a diagnosis is possible these tumors may be easily removed, as the cyst-wall is separable from the brain tissue. Several such cysts have been removed in Germany.

Gummata of the brain, though very rare in childhood, and never the result of inherited syphilis, are very frequent in adult life, and may develop within a year of the original infection. They may also appear as the only apparent evidence of tertiary syphilis even twenty years after the disease was acquired.

Gumma may occur as a soft gelatinous exudation upon the base of the brain, its favorite position, or anywhere in the membranes, or as a harder and more circumscribed tumor in the meninges, especially of the cortex and cerebellum. It grows rapidly, but also subsides rapidly, under specific treatment. In one case under my care, where many symptoms, including double optic neuritis, were present, the patient was perfectly well in nine months. The occurrence of nocturnal headache and insomnia of an obstinate character in any case presenting the symptoms of brain tumor, will suggest the probability of a gumma; and, in fact, in every case in which the diagnosis of brain tumor is made it is well to subject the patient to a course of treatment by mercury and iodide for a period of at least two months, in order to eliminate the possibility of gumma. Yet it must not be hastily argued that the subsidence of symptoms under this treatment is positive evidence that the tumor is syphilitic. In a case under my observation, which autopsy proved to be a small sarcoma with a large cyst, the wall of which was sarcomatous tissue, all symptoms, including optic neuritis, subsided under this treatment, and the patient remained well for a period of five months, and then sudden death in coma occurred unexpectedly.

Other varieties of brain tumor, such as fibroma, angioma, myxoma, psammoma, osteoma, cholesteatoma, lipoma, teratoma, which occur in the brain, are of great rarity. They occur both in children and in adults, and their diagnosis cannot be made from other forms of tumor during life.

Psammoma is usually the form of tumor occurring in the situation of the pineal gland, and gives rise to symptoms similar to those produced by tumors of the corpora quadrigemina.

Tumors of the pituitary body are not uncommon, and are usually of the nature of fibroma or myxoma. It is maintained by some that the disease acromegalia is the result of such tumors. It is not true, however, that a tumor of the pituitary body always produces symptoms of acromegalia, and in one case of acromegalia under my observation the autopsy failed to show any change in the pituitary body.

The appearance of a brain in which a tumor has developed is usually characteristic. The membranes are tense, the convolutions are flattened by pressure, the ventricles are distended by serous fluid, the brain tissue is wet, the weight of the brain is increased, and frequently about the tumor there is a zone of softening of greater or less extent, according to the variety of the tumor. Encapsulated tumors are less likely to be surrounded by a softened tissue.

Intra-cranial aneurisms present the same features as tumor of the brain. They are rarely large in size, they appear upon the larger arteries at the base of the brain and upon the Sylvian arteries. They are usually fusiform, occasionally round. They increase in size more rapidly than ordinary brain tumors, or than aneurisms elsewhere, and usually rupture before attaining any great size. They produce symptoms by their pressure, and are occasionally to be diagnosticated by pulsating headache or the sensation which they produce, or by a distinct murmur audible to the stethoscope on the head. Such a murmur is not, however, pathognomonic, as I have heard such a loud double murmur over the Sylvian region in a case which proved at the operation to be one of extensive softening—no aneurism being found.[1] In a case recently seen at St. Luke's Hospital the aneurism developed rapidly after a blow on the head, producing, first, left-third nerve paralysis, and one month later right hemiplegia. The patient died three months after the injury, having had all the general symptoms of brain tumor. An aneurism of the left internal carotid artery at the point of union with the posterior communicating artery was found, which had ruptured. As the arteries were not diseased, it seemed probable that the bruise of the artery against the bone at the time of the injury had weakened the wall and predisposed to aneurism.

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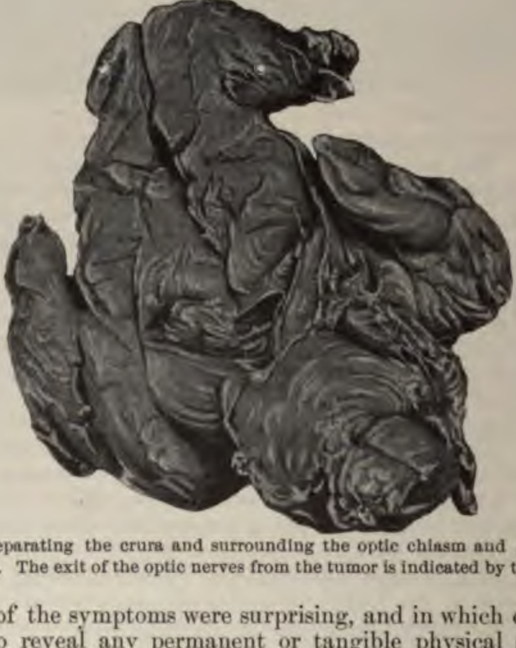
[1] Starr: Brain Surgery, Case xix.

Symptoms. While the symptoms of brain tumor are in many cases very clear and characteristic, so that there need be little doubt in regard to the diagnosis, both of the existence of a tumor, of its nature, and of its location, it must be stated that there are other cases in which no characteristic symptoms at all appear.

Numerous cases have been recorded in which large tumors have been found unexpectedly after death, even in locations in the brain in which the existence of a tumor would presumably have produced very marked symptoms. Thus, in one case under my observation, a large glioma occupied the entire white substance of the frontal lobe upon the left side, yet the patient had suffered only from occasional attacks of epileptic convulsions, not preceded by an aura, and never unilateral, was as intelligent as ever up to the time of his death, though his family had noticed some irritability of temper at times, and on this account had occasionally questioned his responsibility for certain peculiarities. This man was under observation by most competent physicians for several years, and was supposed to have epilepsy. His eyes were examined and found to be normal within a few weeks of his sudden death, which took place in a convulsion. He had never had the ordinary symptoms of a brain tumor. Schoenthal[1] has recorded a case of supposed hysteria in which the variability of the symptoms were surprising, and in which careful examination failed to reveal any permanent or tangible physical signs. After death a large tumor of the right frontal lobe was found. Mayer[2] has collected several such cases. Buzzard has recorded similar cases. Bramwell[3] has de-

FIG. 130.



Sarcoma of base separating the crura and surrounding the optic chiasm and greatly stretching the optic nerves. The exit of the optic nerves from the tumor is indicated by the white dots.

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[1] Berlin. klin. Woch., 1891, No. 10. See also Williams: Lancet, October 14, 1893.

[2] E. Mayer: Inaug. Dissert., Berlin, 1891.

[3] Intra-cranial Tumors, p. 12.

scribed an enormous tumor lying in the central region upon the cortex of the right side in which there were no symptoms of paralysis whatever. And many tumors have been found in and about the cerebellum which have failed to produce the ordinary symptoms of brain tumor.

Tumors upon the base of the brain may also be found involving structures, which, during life, appear to have been capable of carrying on their ordinary functions. Thus a child under my observation suffered for five months from occasional convulsions, from headache, from spastic paraplegia, but had no difficulty whatever of vision, and no paralysis of the cranial nerves. The autopsy showed a large tumor upon the base in the median line, through which both the optic nerves passed and in which the optic chiasm lay. It must have rested upon all the motor nerves of the eyes and compressed the fifth nerve on both sides. This tumor had grown upward, filling both the lateral ventricles and obliterating the third ventricle entirely.[1] (See Fig. 130.)

I have seen a tumor of the medulla oblongata, an infiltrating glioma, which produced an apparent uniform increase in size of the entire medulla to double its ordinary dimensions, but in which there were absolutely no signs of any disease of either cranial nerves or tracts passing through this important part of the nervous system. The patient had headaches, slight optic neuritis, occasional convulsions, and a slightly staggering gait, which led to the diagnosis of tumor of the cerebellum. (See Fig. 125.)

It is thus evident that tumors in the various parts of the brain substance may develop and assume a considerable size without producing characteristic symptoms either of a local or of a general type. Such cases, however, are rare, and the careful observer will usually be able to diagnosticate a brain tumor.

The symptoms of brain tumor are very numerous, but for purposes of convenience may be separated into two categories.

I. General symptoms due to the existence of a new growth irrespective of its position. These are headache, general convulsions, double optic neuritis, and optic atrophy, changes of disposition and of mental power, vomiting, vertigo, insomnia, changes in the pulse rate, attacks of syncope, polyuria, and progressive malnutrition.

II. Focal symptoms of the disease in the cortex of the brain or beneath the cortex in the projection tracts which join the cortex to the various subcortical centres. These symptoms are unilateral spasms, monoplegia, or hemiplegia, paræsthesia or anæsthesia in one or more limbs, hemianopsia, and the various forms of aphasia. Affections of the cranial nerves and basal parts of the brain, which occur with tumors in the basal ganglia and cerebral axis, or external to the brain upon the base. These latter symptoms are frequently very complex, as may be supposed, when it is remembered that the twelve cranial nerves have extensive nuclei of origin and a long course, and that all connection between the external world and the brain passes through the cerebral axis.

In the vast majority of cases of brain tumors we find some of these general and focal symptoms present. And from their combination and the order of their development it is usually possible to arrive at a definite diagnosis. These symptoms must therefore be studied with care.

I. GENERAL SYMPTOMS occur irrespective of the location of the tumor, and depend upon its rapidity of growth, its vascularity, and its pathological character. They vary in severity from time to time, probably in accordance with the activity of the pathological process going on in and about the tumor and with the condition of blood-supply in the brain. When a tumor is growing rapidly they are very severe; if it remain stationary they may almost disappear. They are often affected by those agents which produce a temporary cerebral hyperæmia or anæmia, as is proved by the fact that hot foot baths or general hot baths may produce marked changes in the degree of general symptoms.

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[1] Transactions Amer. Neurol. Assoc., 1891. Starr: Brain Tumor.

Headache is the most prominent and constant symptom of brain tumor. It varies in severity, but is usually very intense. It may be intermittent, it may recur with periodicity, is usually worse at night in syphilitic cases, worse in the daytime in other cases, is increased by physical effort, or mental strain, or emotional excitement. It may be a dull, heavy, continuous pain, with sharp paroxysms, during which the patient cannot control his suffering. It varies in its location, is usually frontal or occipital, and the situation of the pain rarely indicates the situation of the tumor. If, however, the pain is constantly located in the occipital region the tumor is probably in the posterior fossa. The pain is often associated with indefinite cerebral sensations described as fulness, pressure, confusion, tightness, as if a band were drawn about the forehead; and these give rise to great discomfort. In infants the existence of headache may be inferred from constant motion of the head, from movements of the hands grasping the head, or pulling the hair, and from sudden outbursts of screaming without other ascertainable cause. Headache is probably less severely felt in cases of tumor in infants, as the opening of the sutures may prevent the extreme degree of intra-cranial pressure to which the headache is chiefly ascribed. In adults the headache prevents the patient from making any physical or mental exertion, and leads to a desire for seclusion and quiet.

The causes which are supposed to produce the headache are the increase of the intra-cranial pressure and the consequent stretching of the membranes; the existence of effusion into the ventricles, which is a frequent accompaniment of a tumor; the variations in the cerebral circulation produced by the pressure of the tumor, or the direct involvement of the sensitive dura mater and branches of the fifth nerve. Such effects of the presence of a tumor are more likely to follow when the new growth is in the posterior cranial fossa under the tentorium cerebelli, and in such cases headache is most constant and most severe.

The headache is often associated with a marked tenderness of the head to percussion, and if this is not due to sensitiveness of a single nerve trunk it is a very valuable sign of the location of a tumor. Such a tenderness to percussion is more commonly found in tumors lying just beneath the bone, that is cortical tumors, than in tumors deep within the brain. It has been asserted that a flatness to auscultatory percussion can be detected over a tumor, and I am able to confirm this statement, having noticed it in several cases. It is not always present.

General convulsions are the next most frequent symptom of brain tumor. They are particularly liable to occur as an early symptom in children, but may also be the first symptom of tumor in adults. Thus, in a patient of mine from whom Dr. McBurney removed a large sarcoma of the frontal lobe, the first symptom was a general convulsion which occurred two months before the headache began, and was never repeated during the three years' course of the disease.[1] In another patient under my care, who subsequently developed all the general symptoms of brain tumor, general convulsions occurred at intervals during three months before any other symptom appeared. A case diagnosticated as epilepsy has already been mentioned.

It is not uncommon to find convulsions occurring at irregular intervals from the onset of the disease to its termination. They usually occur at long intervals, but have been known to be as frequent as twenty to thirty a day. They may be slight in degree, almost of the nature of *petit mal*, with a sudden loss of consciousness, or this loss of consciousness may be attended by a little twitching of the face and eyes, with stiffening of the back and extremities, balancing movements of an automatic kind, which prevent falling, and then recovery; or they may have all the general characteristics of a general epileptic seizure followed by coma. Sometimes a peculiar general tremor follows the attack, and may last for an hour or more.

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[1] See Amer. Journ. of the Med. Sciences, January, 1893.

General convulsions may be a culmination of a local spasm; hence, wherever a convulsion occurs particular attention is to be called to the manner of its beginning, and the patient is to be carefully observed and instructed to watch for a conscious aura. The significance of local spasms will be discussed in the next section.

Convulsions are usually indicative of rapid progress in the new growth, of effusion into the ventricles, of hemorrhage within the tumor or of a secondary affection of the meninges. They may occur from tumors situated anywhere, and have no significance as a diagnostic symptom of its location. Death not infrequently occurs in convulsions in brain tumor, and hence a development or rapid increase of this symptom is a sign of danger.

Changes of disposition and of mental power occur in the course of brain tumor with considerable frequency. These have sometimes been explained by the existence of headache, the suffering producing mental exhaustion. But they are also observed quite uniformly with tumors of the brain, even when headache is not severe. In children this symptom is quite noticeable; the child becomes fretful and irritable, refuses to notice its toys or to play, or, if it does so, soon becomes wearied, and requires constant attention. It may become indifferent to things in which it was formerly interested, prefers to lie down and to keep quiet, in a manner unnatural to a healthy child, and may even become somnolent and lethargic, or it may have apparent attacks of causeless terror. In adults the mental dulness is very noticeable. The patient takes little interest in his ordinary surroundings or business, and is content to sit or lie for hours, doing nothing, and apparently with vacant mind. Such a patient is easily aroused, and replies intelligently to questions, but cannot be considered in a normal state of mental activity. On the other hand, such a patient is not demented, although late in the course of the disease a condition quite approaching dementia is often observed, especially in tumors of the frontal lobes. Attacks of maniacal excitement have been recorded, but are very rare. The usual mental state present in brain tumor after it has existed for a period of six months may be described as one of apathy. These symptoms are probably referable to the increased intra-cranial pressure and consequent compression of the brain, which interferes with the processes of association upon which all thought depends. The tumor appears by its pressure to hamper the transmission of impulses throughout the brain. Somnolence may be associated with this apathy. In the case of tumor of the frontal lobes and corpus callosum[1] (shown in Figs. 128 and 129) mental apathy was the chief symptom from beginning to end.

Double optic neuritis and optic nerve-atrophy are very important diagnostic symptoms of intra-cranial tumor. Neuritis is usually associated with other signs of increased intra-cranial pressure, but may occur without such pressure. It is present in 80 per cent. of the cases, and should be looked for in every case which presents cerebral symptoms. A marked degree of optic neuritis may exist without any impairment of vision; hence, the ophthalmoscope should be used, whether the defect of sight be present or not. But when the patient shows impairment of visual power, or limitation of the visual field for colors or for light, or is becoming blind, it will be found that optic neuritis or optic atrophy is fully developed. Sudden loss of vision appears to be more commonly noted in histories of children's cases than in those of adults, possibly because a gradual loss of sight is not detected. It is true that hydrocephalus may cause choked disks, and hence from this symptom alone a tumor cannot be diagnosticated. But in cases where the diagnosis is difficult no more important objective evidence of brain tumor can be found.

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[1] R. G. Francis: Amer. Journ. of the Med. Sciences, June, 1895.

Tumors of the cerebellum and corpora quadrigemina, and tumors upon the base of the brain and in the basal ganglia, produce optic neuritis more constantly and earlier in their course than tumors situated in the cortex or centrum ovale. Optic neuritis is usually double, though it always appears first in one eye, and is rarely equally intense in both eyes; but in a few cases it has been found in one eye only, and then is thought to indicate disease of the nerve in the orbit or in front of the optic chiasm, rather than a distant tumor. For the exact changes in the retina and for the pathological causes of optic neuritis the reader is referred to special text-books.[1]

Vomiting is a symptom of brain tumor more frequently observed in children than in adults. It may or may not be accompanied by nausea. It may occur accidentally, without special relation to the time of meals, or it may be so continuous as to threaten inanition. It occurs not infrequently on any movement of the head after the patient has been confined to bed for some time, and then it is usually associated with vertigo. It also frequently accompanies severe headache.

Vertigo is sometimes a coincident symptom, but usually occurs independently of vomiting. The patient feels dizzy, feels himself turning or falling, and things about him appear to be in motion. He grasps at near objects for support, covers his eyes with his hands, or lies down on the floor and cries out with bewilderment and distress. Like vomiting, vertigo may be excited by changes of position. It occurs at intervals, in attacks of short or long duration. It occurs more frequently with tumors in the posterior fossa, in the cerebellum, or pons, or on the base involving the auditory nerve, than with tumors elsewhere. Such attacks of vertigo are not to be confounded with the slight constant vertigo due to double vision, and secondary to paresis of the third and sixth nerves.

Insomnia may be due to disturbances of the cerebral circulation or to the intensity of the other general symptoms, and is much more rarely complained of in cases of tumor in children and youth than in adults suffering from syphilitic tumors.

Fever and changes in the rapidity and rhythm of the pulse have been observed in the course of brain tumors. The former is ascribed to inflammatory changes in the brain or meninges as a complication. The latter is regarded as evidence of increased intra-cranial pressure. Slow and irregular pulse is the form usually noted, but toward the close of life very rapid pulse has been observed. Irregular or Cheyne-Stokes respiration has also been noticed as a terminal symptom.

Occasionally attacks of syncope occur in patients with tumor of the posterior fossa, and a general feeling of weakness is not infrequently complained of. Polyuria and glycosuria may develop in the course of brain tumor[2] as a symptom of increased pressure. It has been found also in small tumors of the medulla[3] as an evidence of irritation of the vagus nucleus.

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[1] See especially Knies: The Eye in General Diseases. William Wood & Co., 1895.

[2] See Rothmann, Zeitsch. f. klin. Med., 1893, xxiii, p. 339.

[3] De Jonge, Arch. f Psych., xiii. p. 658.

A rise of temperature in the scalp over the tumor has been observed in a few cases, but is not a uniform or reliable symptom of brain tumor.

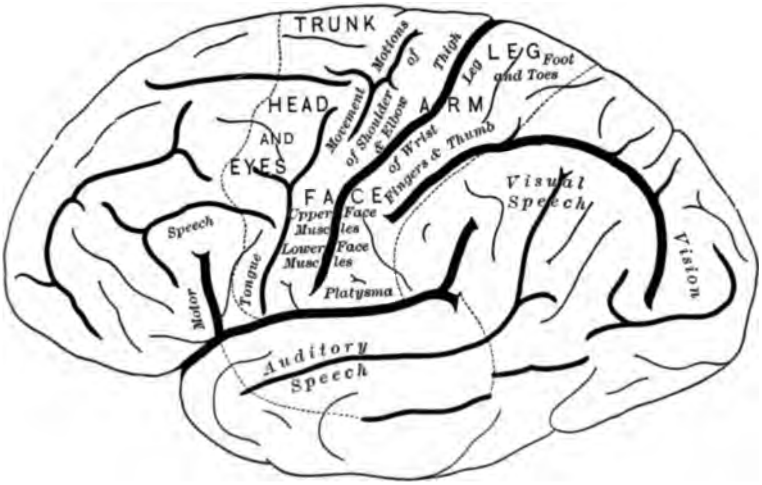
The combination of several of these general symptoms in any case should lead to a strong suspicion of intra-cranial tumor. When, in addition, local symptoms are added, the diagnosis should not be difficult. In all cases the onset of the general symptoms is gradual, their intensity increasing as time goes on, and a careful study of their mode of development and progress is a great aid in the diagnosis of the kind of tumor present, of its size, and of the rapidity of its growth.

The distress produced by the general symptoms is usually much greater than that caused by the local symptoms, and consequently treatment is often required for their alleviation.

THE FOCAL SYMPTOMS of brain tumor depend entirely upon the situation of the tumor.

A distinction is made between direct and indirect focal symptoms, the first being due to irritation or destruction of a limited area by the tumor, the second being due to interference with the function of an area by disease at a distance from it which impairs its circulation or causes pressure upon it. Thus, a tumor of the cerebellum may cause inco-ordination and vertigo as direct symptoms, and also cause paralysis of the sixth and seventh cranial nerves as an indirect symptom of displacement of the pons Varolii to one side, which stretches these nerves unduly. Tumors usually cause both forms of focal symptoms, and, therefore, much care must be given to the question of their significance in any case.

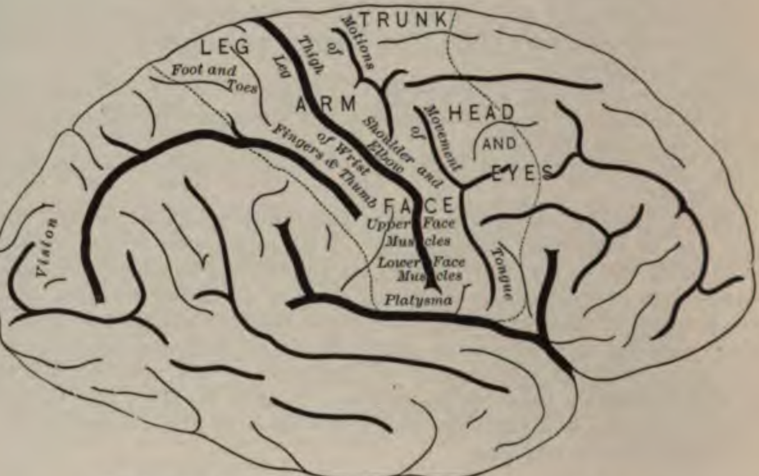
FIG. 131.



The functional areas of the cerebral cortex. Left hemisphere.

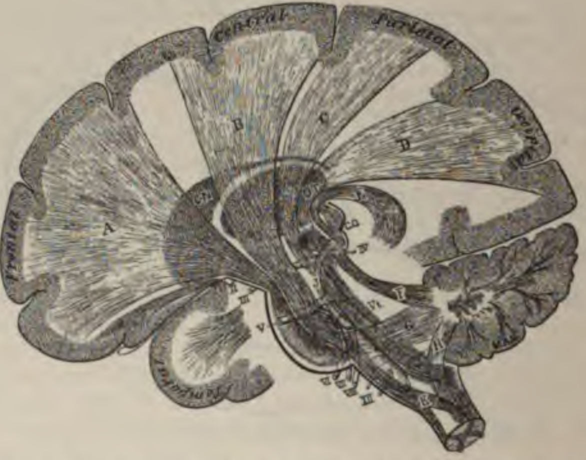
Focal symptoms may further be divided broadly into two classes, those produced by tumors in the cortex and cerebral hemispheres, and those produced by tumors upon the base of the brain affecting the cranial nerves. Figures 131 and 132 display the various functions performed by the different areas of the cortex of the brain so far as is at present known. Reference may be made to the preceding chapter upon localization of the brain functions. Figures 133 and 134 represent the tracts and association fibres which lie beneath the cortex and occupy the cerebral hemispheres above and in the vicinity of the basal ganglia, and Figure 133 displays the fact that all fibres joining the cortex with the cerebral axis are compressed into a narrow strait—the internal capsule—in their exit from the cerebral hemispheres. Figure 135 shows the base of the brain, together with the numerous cranial

FIG. 132.



The functional areas of the cerebral cortex. Right hemisphere.

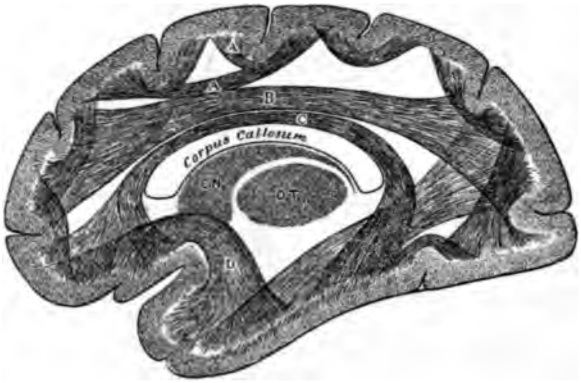
FIG. 133.



The projection tracts joining the cortex with lower nerve centres. Sagittal section, showing the arrangements of tracts in the internal capsule. A, tract from the frontal lobe to the pons, thence to the cerebellar hemisphere of the opposite side; B, motor tract from the central convolutions to the facial nucleus in the pons and to the spinal cord; its decussation is indicated at K; C. sensory tract from posterior columns of the cord, through the posterior part of the medulla, pons, crus, and capsule to the parietal lobe; D, visual tract from the optic thalamus (OT) to the occipital lobe; E, auditory tract from the intergeniculate body, to which a tract passes from the VIII. N. nucleus (J) to the temporal lobe; F, superior cerebellar peduncle; G, middle cerebellar peduncle; H, inferior cerebellar peduncle; CN, caudate nucleus; CQ. corpora quadrigemina; Vt, fourth ventricle. The numerals refer to the cranial nerves.

nerves, and from it may be deduced the various symptoms that will arise when a tumor involves any portion of the base. With the anatomy of the brain in mind it is not difficult to understand the various local symptoms that may arise from the existence of brain tumor in different localities. It should also be remembered, however, that a tumor of very slow growth, which compresses and does not infiltrate the brain, may exist in any of these locations without necessarily producing focal symptoms, until it has attained a considerable size. It is also evident that a single tumor rarely, if ever, produces a large number of focal symptoms, but that all tumors lying in any given location will produce the same symptoms. It is also evident that tumors will almost always produce unilateral symptoms, inasmuch as they rarely lie exactly in the median line. And even the tumors of the corpus callosum, of which about twenty have been recorded, have rarely been so exactly median in their situation as to give rise to symmetrical symptoms upon both sides.

FIG. 184.



The association fibres. A, between adjacent convolutions; B, between frontal and occipital areas C, between frontal and temporal areas, cingulum; D, between frontal and temporal areas, fasciculus uncinatus; E, between occipital and temporal areas, fasciculus longitudinalis inferior; CN, caudate nucleus; OT, optic thalamus.

The focal symptoms usually commence gradually and are limited in extent, but increase steadily as the tumor grows.

The focal symptoms of tumors of the frontal cortex may be enumerated as follows: Mental inactivity, forgetfulness, lack of judgment, decided change in character, irritability of temper and unusual stupidity, an inability to concentrate the attention, to think connectedly and continuously, to learn easily, to exercise self-control, lastly a state approaching mild dementia without delusions, in which the patient may become dirty and disregard all restraints of decency. These symptoms are especially marked in tumors of the frontal cortex and subjacent white matter.[1] They are not present in tumors lying upon the base of the frontal region on the orbital bone. They rarely appear in tumors in other regions of the cortex until the last stages of the disease, when the intra-cranial pressure is very great. A decided mental change in character and disposition, a mental apathy and a tendency to somnolence must be regarded as a local sign of frontal lobe disease. While the integrity of the frontal lobes is necessary to complete mental action, yet no special loss of mental faculty can be said to result from their destruction. Nor is there any apparent way to detect from the mental symptoms in which hemisphere of the brain the tumor lies. The diagnosis of tumor of the frontal lobes is, therefore, rarely made from a study of direct focal symptoms. It is to be remembered, however, that a tumor when situated in other regions of the cerebral hemispheres, excepting only the temporo-sphenoidal lobe, will

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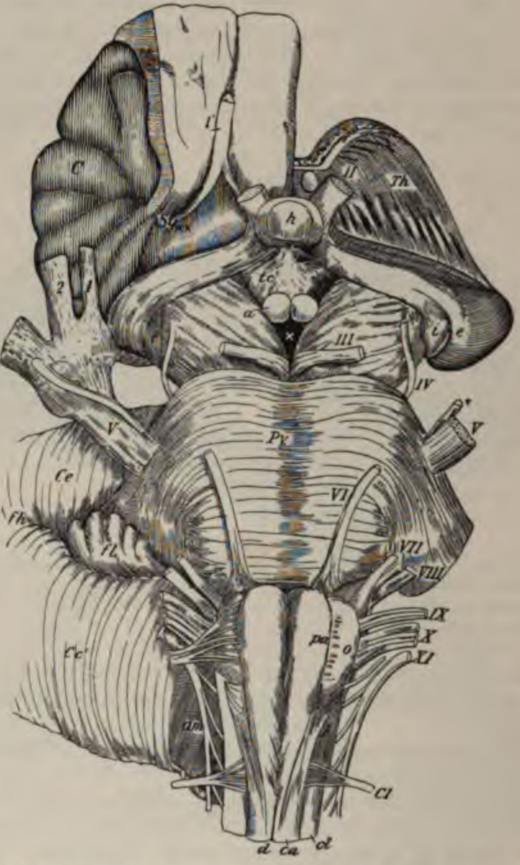
[1] Ferrier: Lancet, June 4, 1892.

FIG. 135.



The base of the brain and the cranial nerves, crura, pons, and medulla (ALLEN THOMPSON). *I* to *XII*, the cranial nerves; *Th*, optic thalamus; *h*, pituitary body; *tc*, tuber cinereum; *a*, corpora albicantia; *P*, pes pedunculi; *i*, interior; and *e*, exterior, geniculate body; *PV*, pons Varolii; *pa*, anterior pyramid of medulla; *o*. olive; *d*, decussation of anterior pyramids; *ca*, anterior column of spinal cord; *cl*, lateral column of spinal cord; *Ce*, cerebellum; *fl*, flocculus of cerebellum.

produce other direct focal symptoms, the absence of which may lead to the suspicion that a tumor is situated in the frontal lobe.

Tumors in the frontal lobe frequently produce irritation of the cortex. While such irritation is limited to the frontal cortex we have no evidences of its occurrence, but there is a tendency in the brain for irritation, starting in one part to radiate outward to adjacent parts, like the ripple upon a lake when a stone is thrown in. While irritation starts from the frontal region it frequently appears to radiate backward, and thus involve the central region of the brain, and under these circumstances symptoms referable to the central region appear, namely, localized spasms, paræsthesia, and paralysis.[1]

Bruns has recently recorded[2] three cases of frontal tumor in which there had been some disturbance in the balancing power, and a staggering gait similar to that observed in cerebellar disease; but this symptom has not been uniformly noticed. It is known that the frontal lobes and cerebellar hemispheres have a crossed connection, and it is possible that this symptom may prove of some diagnostic value if confirmed. It may have been due to an irritation of the cerebellum conveyed along these connecting tracts.

Tumors situated in the third frontal convolution of the left hemisphere in right-handed persons and of the right hemisphere in left-handed persons produce motor aphasia with agraphia, the patient being able to understand language both written and spoken, but being unable to give expression to his ideas. When this symptom develops as the result of brain tumor the disturbance of speech is gradual in its onset, the patient noticing first a hesitancy in speech, a loss of words, and possibly a misuse of words before he loses the power of expression. The aphasia is less complete in brain tumor than in those apoplectic conditions which are the common cause of aphasia.

Tumors involving the motor area of the brain give rise to well-marked and distinctive symptoms. The special functions of the motor area are displayed in Figures 131 and 132, and the symptoms that are produced by a tumor beginning in any portion of this area are easily deduced from reference to these figures. If the tumor is cortical in its situation it almost inevitably gives rise to local symptoms; these may be in the form of tonic or clonic convulsions, occurring at intervals, either limited to one part of the body, such as the face, or hand, or foot, or extending from the part first invaded to other parts in a definite order of succession; the extension being commonly from face to arm, to body, and then to leg, or in the reverse order. Such attacks, first described by Hughlings Jackson, are known as attacks of Jacksonian epilepsy. If it be remembered that the irritation starting from one part of the cortex and radiating outward to other parts produces a spasm beginning in the muscles represented in the centre first irritated, and extending to other muscles represented in adjacent centres, it will be evident that the exact order of the spasm aids one to determine the situation of the tumor, for it is evident that a spasm beginning, we will say, in the thumb and extending then to the fingers, to the hand and up the arm to the shoulder, will indicate an initial irritation of the thumb-centre of the cortex, extending then to the centre for the fingers, hand, and arm. A convulsion beginning with the turning of the head and eyes to one side indicates a tumor situated in the anterior middle portion of the motor zone, and if that irritation extends from this point backward or downward the spasm will extend from head and eyes to arm or face respectively. Thus the point of beginning of a spasm and its order of extension are the most important localizing symptoms of brain tumor in the motor area. Such a spasm is usually followed by temporary paralysis, and as the tumor increases in size and the spasms increase in frequency and extent, the paralysis may remain permanent between the spasms. Spasms due to cortical irritation are almost invariably attended by numbness and tingling in the part first affected by spasms, so that such a tingling is often a valuable indication of the location of irritation in the cortex. This tingling may even precede the spasm, and has been termed by Seguin the "signal symptom" of a cortical irritation. When such a paræsthesia is a permanent symptom the tumor is more likely to be situated behind the fissure of Rolando than in front of it. When the irritation produced by a tumor in the motor zone is very intense, the local spasm, after extending from one limb to the entire side of the body, may even become general, be associated with loss of consciousness, and terminate as a general convulsion. While such an order of extent preceding the general convulsion is of great diagnostic importance, a general convulsion beginning suddenly, with loss of consciousness and without any local spasm, is not to be regarded as indicative of disease in the motor area. There are cases of brain tumor in which the motor area has been generally compressed and destroyed without the production of these local spasms, but with a progressive and increasing paralysis alone. It is thought that such a paralysis not associated with spasms indicates that the situation of the tumor is in the white matter beneath the cortex and not in the cortex itself, and that the symptoms are due to an invasion of the motor tract in its passage toward the internal capsule. Some ataxia is commonly associated with the paralysis produced by tumors, and a marked inco-ordination or disturbance of the muscular sense, like anæsthesia, points to a situation of the tumor behind the fissure of Rolando. The condition of the deep reflexes is altered in all cases of tumor in the motor area; a marked increase in the tendon reflexes at the elbow, wrist, knee and ankle is one of the early signs of paralysis. There is no atrophy in the paralyzed muscles, but merely a slight wasting from disuse, and there is no change in the electric reaction of the muscles. There should be no difficulty in differentiating a cerebral paralysis, even of the monoplegic type, from spinal or nerve-trunk paralysis, even in infancy, because of these points of contrast. The careful study of the localizing symptoms of tumors of the motor region has resulted in successful localization of such tumors and in their successful removal from the brain; in fact, there is no region in the brain in which a tumor is more easily accessible or more possible of early diagnosis than in the motor area.

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[1] See a case of my own, American Journal of the Medical Sciences, Jan., 1893.
[2] Deut. Med. Wochen., 1892, p. 138.

Tumors of the parietal region, including the superior and inferior parietal lobules are not uniformly attended by distinctive and local symptoms, but in a considerable number of cases disturbances of sensation and of muscular sense in the limbs of the opposite side have been observed. It has been stated already that tumors in the posterior central convolution are more liable to produce sensory symptoms than those in the anterior central convolution. And it is possible that tumors lying in the parietal region have caused sensory symptoms by pressure upon the sensory tracts on their way from the posterior portion of the internal capsule to the central region of the brain. If this is so, then ataxia and anæsthesia appearing in connection with a tumor of the parietal region would be an indirect local symptom rather than a result of a lesion in the sensory centres themselves. This is not the place to enter upon the discussion with regard to the existence of a muscular sense as distinct from the sensory centres and the motor centres, yet there are many facts which indicate the existence of muscular sense centres in the parietal lobes of the brain.[1]

The symptom of word-blindness, to be discussed later in connection with aphasia, is almost uniformly due to a lesion of the inferior parietal lobule in the left hemisphere in right-handed people and in the right hemisphere in left-handed people.

Tumors lying in the lower half of the parietal region are necessarily near to the visual tract which passes outward from the internal capsule to the occipital lobe, and hence, defects of vision of the form of bilateral homonymous hemianopsia have been recorded in connection with tumors in this locality.

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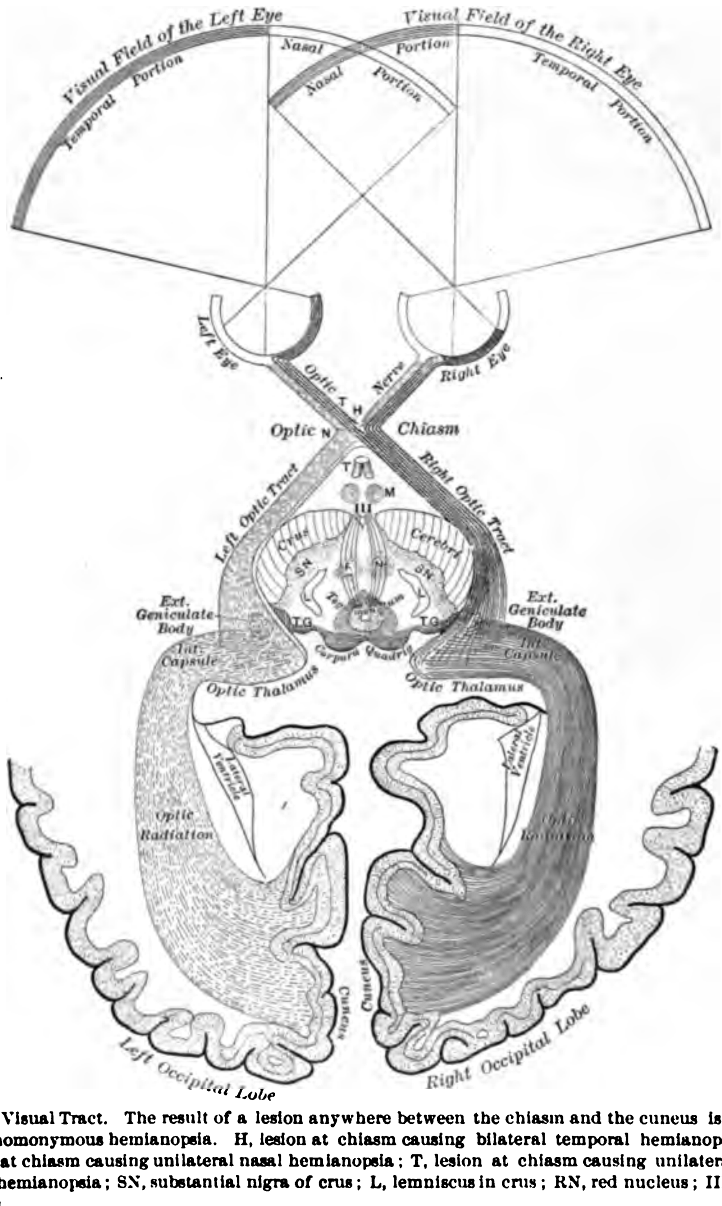
[1] Starr and McCosh: American Journal of the Medical Sciences, August, 1894, A Contribution to the Localization of the Muscular Sense.

Tumors of the occipital region of the brain given rise to the important local symptoms of lateral homonymous hemianopsia. (See Fig. 136.) Recent

FIG. 136.



The Visual Tract. The result of a lesion anywhere between the chiasm and the cuneus is to produce homonymous hemianopsia. H, lesion at chiasm causing bilateral temporal hemianopsia; N, lesion at chiasm causing unilateral nasal hemianopsia; T, lesion at chiasm causing unilateral temporal hemianopsia; SN, substantial nigra of crus; L, lemniscus in crus; RN, red nucleus; III, third nerves.

researches by Henschen[1] have proven conclusively that the visual area of the cortex to which all impulses from the eyes are sent lies in the calcarine fissure upon the median surface of the hemispheres and includes the cuneus and the cortex beneath it, which lies within the calcarine fissure. In order to reach this situation the visual tract makes a wide curve from the internal capsule backward almost semi-circular in shape with its convexity outward. (Fig.136.) The apex of the occipital lobe is narrow and the calcarine fissure is deep, hence, at its termination there is little lateral expansion of this tract, and therefore a lesion in almost any part of the occipital lobe at its apex or even its convexity is quite liable to invade the visual tract and to produce the same symptom as is caused by a limited lesion in the calcarine fissure or its adjacent cortex.

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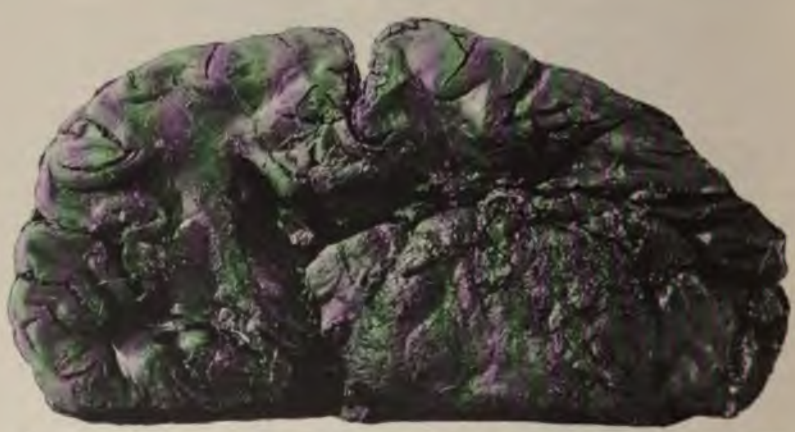
[1] Pathologie des Gehirns. Upsala, 1892 to 1894. Bd. 1, 2, and 3.

Henschen's researches show that a lesion at almost any portion of the occipital lobe, or even, as has already been seen in the parietal lobe, if sufficiently deep to compress the white matter of this region will produce hemianopsia; and it is to be expected that in any lesion of the nature of a tumor in which small areas of tissue are not destroyed, but considerable areas are primarily affected and adjacent regions are strongly compressed, symptoms of hemianopsia will be frequent.

The occipital lobe is easily accessible to the surgeon, and therefore it is important that tumors here should be discovered as early as possible. It is not generally appreciated that hemianopsia is a symptom often entirely overlooked by a patient, a case having been recently reported by Bleuler[1] in which, though well-marked hemianopsia existed, the patient was entirely unaware of any visual defect. It is therefore exceedingly important that the extent of the visual field in both eyes should be carefully tested in every case of suspected brain disease, each eye being tested separately.

FIG. 137.



Gliosarcoma of right temporo-occipital region, causing hemianopsia, and later hemiplegia.

Lesions of the occipital lobe are capable of producing a disturbance known as blindness of mind, in which the patient, though seeing an object no longer recognizes it as having been previously known. This is a condition allied to word-blindness, in which the patient is suddenly deprived of the power of reading. This condition appears to be more frequent in lesions upon the left side of the brain in right-handed persons, and upon the right side of the brain in left-handed persons. It may also occur when a lesion is entirely sub-cortical, as well as when it is in the cortex of the occipital lobe. This symptom will be discussed further in connection with aphasia.

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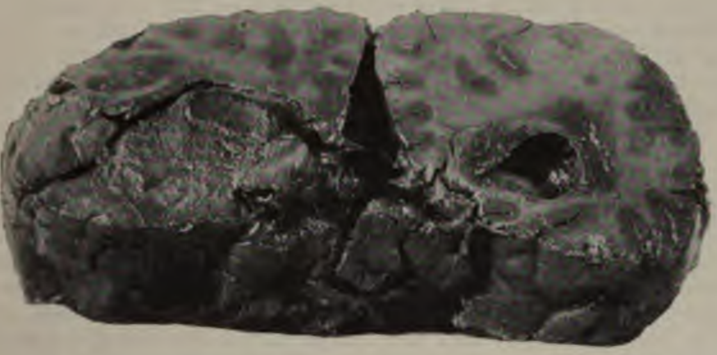
[1] Archiv für Psychiatrie, vol. xxv. p. 39.

Tumors of the temporal lobes or tumors lying upon the base of the cerebral hemispheres above the tentorium or upon the petrous portion of the temporal bone give rise to few recognizable symptoms. In a few such tumors disturbance of taste and smell seems to have been present when the apex of the temporal lobe had been invaded by the growth, and when the uncinate gyrus had been destroyed. There are no known symptoms produced by disturbance of the lingual lobule.

Tumors invading the first and second temporal convolution upon the left side in right-handed persons and upon the right side in left-handed persons produce the form of aphasia known as word-deafness, in which the person is unable to recall the names of places or persons, and cannot understand what is said to him.

It is believed that the first and second temporal convolutions are the termination of the auditory tract, and that each hemisphere is related to both ears. Deafness of cerebral origin is, however, rare, and is not often observed in tumors of these convolutions.

FIG. 138.



Glioma of left temporo-occipital lobe obliterating the ventricle. The distention of the ventricle in the left hemisphere is seen.

Tumors lying within the Sylvian fissure and upon the island of Reil produce numerous symptoms on account of their pressure upon adjacent parts; thus, by pressing the third frontal convolution they cause motor aphasia; by invading the operculum they produce facial paralysis; by pressing upon the inferior parietal lobule, or the superior temporal convolution they may produce various symptoms of sensory aphasia; and by direct pressure upon the island of Reil, and upon the underlying external capsule, they may cause paraphasia. If this pressure is transmitted more deeply to the internal capsule, symptoms of tumor in this region may also be caused.

Tumors lying on the median surface of the brain, upon the gyrus fornicatus, gyrus marginalis, or upon the hypocampal convolution are not easily diagnosticated. Ferrier believes that in the latter convolution are situated the tactile centres. The majority of physiologists (Munk, Horsley, Schæffer) and of pathologists deny this, and recently a case of glioma of this region without sensory symptoms has been reported by Humphrey.[1] Tumors in this region are of rare occurrence, and no focal symptoms can be assigned to them as yet.

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[1] Humphrey: Brit. Med. Journ., Aug. 27, 1893.

Tumors of the corpus callosum are not at all common, only twenty cases being so far recorded in literature.[1]

The symptoms in a majority of these cases have been chiefly general symptoms, viz: headache, vomiting, optic neuritis, epileptiform attacks, physical inertia, hemiparesis, disturbance of intelligence—principally in the form of dementia. These were the symptoms in one case under my observation. The last-named symptom is the only one that appears to be of constant occurrence in these cases, and in many of them it has been preceded by symptoms quite comparable to those of hysteria. The majority of cases of tumor of the corpus callosum have therefore resembled tumor of the frontal lobes. When tumors occur in the corpus callosum they are more likely to be situated far forward about its knee, rather than in its thinner posterior part. (See Fig. 128.)

Our knowledge of the function of the corpus callosum is very imperfect, though it is evident from its structure that it associates the action of the two hemispheres. The corpus callosum has been wholly wanting in the brain in a few cases, and although this is occasionally associated with imbecility it is a condition which has been found in persons who presented no symptoms during life.[2] It is evident, therefore, that tumors in this localty cannot be positively diagnosticated, and the situation of such tumors deep between the hemispheres forbids the possibility of their removal.

In the enumeration of the symptoms produced by lesions in the different parts of the brain *aphasia* is an important one, in fact there are few tumors in the left hemisphere of the brain in right-handed persons, excepting those situated in the apex of the frontal lobe or at the summit of the central region which do not cause more or less disturbance of speech. Our present knowledge of the mechanism of speech has advanced far beyond the point attained by Broca, who located speech in the third frontal convolution, the so-called Broca's centre, or even by that reached by Wernicke, who made the distinction between sensory and motor aphasia, the first due to lesions of the temporal region, the second to lesions of Broca's centre.

It is now known that as the physical basis of any word, be it noun or verb, there exists a number of mental images acquired through different senses located in different regions of the brain, and joined together in a conscious unit by a series of association tracts. The word "concept," long used by psychologists to denote a congeries of mental images making up an idea, may be adopted by the pathologist to indicate this collection of mental images. To be complete such a concept must have all its parts intact and the connection between those parts also intact. If we take as an example a word such as "bell" or "rose," and if we study the process in the mind of the child as it acquires its primary knowledge of some particular bell or rose, we at once see that this particular concept has a very limited extent, consisting of the mental images of the object (1) as seen, (2) as felt—shape, size, temperature, hardness, (3) as smelt, (4) as tasted, if the object has odor or taste, (5) as heard, if the object is audible. These five mental pictures comprise the mental image of the object, and the separate mental images are united with all the others, so that when one rises in memory it inevitably recalls all the rest.

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[1] Bruns: Berlin klin. Woch., 1886, Nos. 21 and 22. Bristow: Dis. of the Nervous System, p. 271. Lutzenberger: Neurol. Centralb., 1890, p. 251. Berkley: American Journal of the Medical Sciences, June, 1890. Oliver: University Medical Magazine, April, 1891. Francis: Amer. Journal of the Medical Sciences, June, 1895.

[2] Bruce: Brain, xii. p. 171.

As another illustration, we may take the verbs "to run," "to sew." We call up to the mind memory pictures of some individual in action, or of some act of our own, with its attendant sensations, and thus, as the basis of the verb "run," as well as of the noun "rose," we must think of a congeries of mental images closely associated with one another. So far our analysis of thought would apply equally well to a child who had not learned to speak, to a deaf and dumb person, or to a healthy man; but in the man it is evident that there is added to this mental image a "word image" quite distinct in its parts from the mental image, and consisting of memory pictures of the word as heard, as seen in print or writing, as pronounced by muscular effort, or as produced in writing by movements of the hand. Each of these separate word-pictures is joined with the others and forms a "word-concept;" each part of this word-concept is also connected with every part of the mental concept which the word enables us to convey to others. Thus it becomes evident that the psychological term "concept" (German, "Begriff") is not a simple thing, but is a very complex thing, implying as its physical basis an activity of widely distant cortical areas in each of which a separate memory picture is located, and of long or short association tracts running in every conceivable direction between these various areas. Now if such is the complexity of the physical basis underlying such simple concepts as a particular bell or rose, or a simple act, like running or sewing, it is evident that the physical basis of more complex concepts, such as the class books or vases, or the complex acts implied by the verbs "to educate," "to civilize," are innumerable. It becomes equally evident that for the conduct of thought, for

FIG. 139.

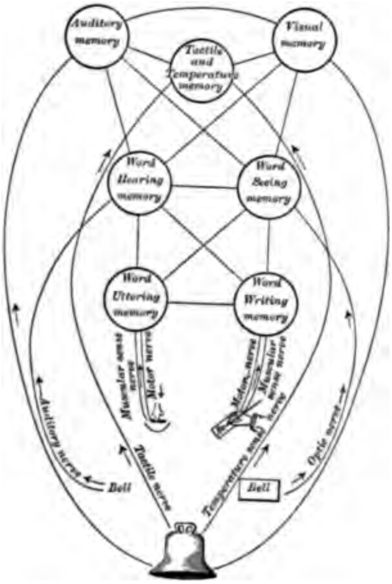

Diagram to illustrate the concept "bell" and to show the varieties of apraxia and aphasia. The memory pictures are relics of past perceptions received through different senses. The association makes up the mental image bell. The word image is made up of the memories of the sound and appearance of the word, and of the uttering and writing-effort memories; these are joined together. The mental image and the word image are also joined with one another, making up the concept "bell."

the perfect action of the mind in dealing with concepts, be they simple or complex, an integrity of the entire brain is necessary both in its cortical extent and in the white subjacent association tracts. With these facts in mind it is absurd to speak of intellectual centres in the brain, or to suppose that such faculties as memory or imagination can be obliterated by a single lesion.

Small lesions of the cortex in various parts may injure these concepts by depriving them of some constituent mental image, and small lesions in the white matter may disturb the use of these concepts by dissociating images which should be closely bound, and hence it is evident that lesions in various parts of the cerebral hemisphere will produce disturbance in the use of our mental concepts, which may be manifest as an inability to recognize places or objects, the condition of mind-blindness and mind-deafness, or mind-paralysis, or a condition of aphasia, word-blindness, word-deafness, inability to speak or to write.

When the lesions of the brain in certain situations are considerable in extent, involving an entire convolution, the symptoms of aphasia are very well marked. Thus, a lesion involving the third frontal convolution produces motor aphasia, the patient being unable to articulate distinctly, to pronounce words, to talk voluntarily, to read aloud, or to repeat a word after another. Agraphia is usually associated with this form of aphasia, the patient being unable to write voluntarily, or copy, or write from dictation. In two or three cases, however, agraphia alone has been noticed, and the lesion has been limited to the posterior portion of the second frontal convolution.

Word-deafness is produced by a lesion involving the first and second temporal convolutions, and in this condition the patient is unable to recall the spoken name of objects, to understand speech, and hence, is often unable to say what he wishes.

Word-blindness is due to a lesion situated in the inferior parietal lobule, including the supramarginal gyrus and the angular gyrus. It is true that a lesion in this situation almost inevitably divides an association tract, passing from the occipital to the temporal lobe, and joining the memory picture of words seen with that of words heard. Hence, it is somewhat uncertain whether word-blindness is due to the lesion of the cortex or to the lesion of the subjacent white tract; but a lesion in this situation always produces a loss of power to understand printed or written words, to read aloud, and to copy.

But while these various well-marked forms of aphasia indicate large lesions in these various situations, it must be admitted that the majority of cases of aphasia are not easily assigned to one of these particular types, that they partake, in some degree, of all the various types, and that some in fact, while they present the features of each type in slight degree, do not present all the features of any one type completely. Such cases of aphasia we must ascribe to very small lesions involving only certain portions of the speech cortex, or involving a few of the many association tracts. When, for example, a patient cannot read, but can copy what he sees, and in the act of copying becomes conscious of the meaning of the words which he has copied, thus being able to read by the aid of his muscular sense, when he cannot read by mere vision, it is evident that he has not lost the power of recalling printed words, because, through the aid of his muscular memories he is able to awaken these memory pictures, but that some lesion in an association tract has cut off the impulses that ordinarily reach the word-memory picture by the way of the eye and the occipital cortex. This may be likened to a break in a railroad which compels the passenger to take a round-about route, with several changes, to reach his destination.

We are not yet able to locate exactly the various lesions producing these complex forms of so-called association aphasia or transcortical aphasia. But we must not be too hasty in our localization in aphasic symptoms, unless they present well-marked characters and belong to one of the chief clinical types.

These are the chief symptoms produced by disease in the cortex of the brain or in the subjacent white matter of the centrum ovale.

Tumors of the basal ganglia, including the lenticular and caudate divisions of the corpus striatum and the optic thalamus, are not very rare. Our knowledge of the functions of these ganglia is very imperfect, and the necessary loss of function which must occur when they are destroyed by softening cannot be detected during life. It has been suggested that these ganglia are to be regarded as vestigial bodies, like the appendix vermiformis, without active function in man. Tumors, however, in this region, almost always produce pressure upon the internal capsule through which pass the most important motor and sensory tracts, and which lies between these ganglia, and hence symptoms of the nature of hemiplegia, hemiataxia, hemianæsthesia, and hemianopsia are observed when the tumor invades one or more of these tracts. It may even be possible to detect the progressive growth of a tumor here by the succession of symptoms produced, one tract after another being invaded by the disease. Motor symptoms are more common in cases of tumors of the corpora striata. Sensory symptoms occur in cases of tumors of the optic thalami. When a tumor invades the posterior part of one thalamus, homonymous hemianopsia is an invariable symptom. This will be easily understood by reference to Fig. 136.

It has been supposed that lesions in the optic thalamus give rise to disturbances in the recognition of the position of the limbs of the opposite side, and that in consequence awkward positions are assumed, and awkward movements, athetoid in character, are made (Meynert). It has also been affirmed that a tremor quite similar to that of multiple sclerosis may be caused by a tumor of the thalamus.[1] It has been noticed that disturbances in facial expression and in the natural play of facial feature in speech and thought have been noticed in patients suffering from disease of the optic thalamus.[2] The face is affected on the side opposite to the lesion, and does not act in smiling, but can be moved voluntarily. It has been thought that certain inhibitory functions are exercised by the thalamus, and that undue emotional reactions occur when it is diseased. Disturbances in the body-temperature have been observed in a few cases.[3] Cases are on record to support each of these statements, yet it must be admitted that numerous negative cases, that is, cases of tumor of the thalamus in which these symptoms were wanting throw much doubt on the diagnostic significance of the effects mentioned.[4] My own view of the functions of these ganglia is that they preside over many acts which are commonly performed in a purely automatic manner, for example, the posture of the body, the ordinary gait, the facial expression, and probably emotional control; that these acts can, however, be performed voluntarily as well as automatically, and hence, when the automatic mechanism fails, a volitional act takes its place, which practice soon perfects, leaving the individual quite unconscious of his defect, and hence, causing few or no symptoms. It is, therefore—on this theory—clear that tumors of the basal ganglia cause few recognizable symptoms.

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[1] D. H. Cooke: Lancet, May 28, 1892.

[2] Kirilzew: Neurol. Centralb., 1891, p. 310. Nothnagel: Zeitsch f. klin. Med., 1889. Bechterew: Neurol. Centralbl., 1884, p. 102.

[3] Cowan: Lancet, December, 1892. Lloyd: Med. News, January, 1892.

[4] Wharton Sinkler: University Med. Mag., October, 1893.

Tumors of the corpora quadrigemnia present symptoms of a focal character which are diagnostic.[1] These consist of a staggering gait similar to that seen in cerebellar disease and symptoms of paralysis or paresis in the muscles controlled by the ocular nerves. The co-ordination in movements of the eyes, the reflex motions of the eyes in following a light, or turning toward a sound, and the power of steady gazing at an object are controlled by automatic centres in the corpora quadrigemina. Disease here frequently produces nystagmus and interferes with these automatic movements. The symptoms are not referable to a disease of one third nerve alone, for the pupils are often unaffected. They are often bilateral and more nearly resemble the symptoms of ophthalmoplegia externa. Hence, it is possible to distinguish lesions of the corpora quadrigemina from lesions in the crus cerebri, which cause true third-nerve paralysis. The eyeballs are not often equally affected, one moving more than the other; but, on the other hand, neither eyeball, as a rule, is totally paralyzed. The superior and inferior recti are more commonly affected than the lateral muscles. It is the combination of these ocular symptoms with the reeling gait which will lead to the diagnosis of tumor of the corpora quadrigemina when the other (general) symptoms of brain tumor are present:

The focal symptoms of tumors at the base of the brain are necessarily very complex. Suffice it to say that tumors lying upon the base of the brain will necessarily compress one or more of the cranial nerves, either upon one side alone or upon both sides, in case the tumor is near the median line. The situation of the tumor can usually be determined by noting the order in which the various cranial nerves are invaded from first to twelfth. The reader is also referred to Chapters XV. and XXIV. for the consideration of lesions at the base, and to Chapters XXVI. and XXVII. for lesions of the cranial nerves.

Tumor of the crus cerebri is characterized by the combination of hemiplegia of one side with third-nerve paralysis of the opposite side. The tumor lies on the side on which the third nerve is affected. To this combination bilateral hemianopsia may be added if the tumor compresses the optic tract.[2]

Tumors in the upper part of the pons or in the crus cerebri affect the third and fifth nerves, producing external strabismus, with dilatation of the pupil and ptosis, and also tingling pain and anæsthesia of the face with ulceration of the cornea, and sometimes grating of the teeth during sleep. Tumors in or near the lower half of the pons involve the sixth and seventh and eighth nerves, causing internal strabismus with contracted pupil paralysis of the face, including inability to close the eyes, and deafness, with vertigo. Alternating hemiplegia is also produced by a lesion in this region. Tumors invading the medulla may disturb the action of the glosso-pharyngeal, pneumogastric, spinal accessory and hypoglossal nerves, producing difficulty in deglutition, irregular respiration, irregular pulse, flushing of the skin, with sweating, sometimes unilateral, projectile vomiting, polyuria or glycosuria, retraction of the head or rolling of the head upon the pillow, and lastly, paralysis of the tongue, with inability to articulate distinctly or to swallow.

When the tumor lies upon the base of the brain, it not only invades the cranial nerves, but it presses upon the various tracts which traverse the crura, pons, and medulla. Thus it may give rise to hemiplegia or hemiataxia or hemianæsthesia, according to the tract invaded. These symptoms are easily understood by reference to Fig. 133.

Tumors of the pons, producing pressure upon the middle peduncle of the cerebellum, produce a tendency to stagger in walking. The patient usually staggers toward the side opposite the tumor, but this is not an invariable rule.

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[1] Nothnagel: Brain, xii. 21. Hall: Heidelberg Dissert, 1892.
[2] Venal: Bullet de la Soc. Anat., Paris, Jan., 1891.

Tumors of the pons quite uniformly produce a loss of the tendon reflexes at the knee. The control of the bladder and the rectum is also frequently impaired.

Tumors of the cerebellum are exceedingly frequent, both in children and in adults, and present almost all of the characteristic general symptoms of brain tumor. The situation of the cerebellum held down in a small cavity by the tentorium cerebelli is such that a small growth in the posterior fossa is capable of producing an increase of intra-cranial pressure quite early in the course of the case. The tumor in the posterior fossa will also compress the fourth ventricle by displacing its floor or roof, and as a result of such compression an accumulation of fluid in the ventricle, and subsequently in the lateral ventricles, is usual. Hence, symptoms appear earlier in the course of the disease in tumors about the cerebellum than in tumors elsewhere in the brain. Headache, occipital or frontal, is usually the first symptom, and optic neuritis appears very early in the course of the case. The percentage of cases having optic neuritis is much greater in tumors of the cerebellum than in tumors in the cerebral hemispheres.

The focal symptoms of tumors of the cerebellum are not very numerous. They are first staggering in walking, sometimes attended by a strong tendency to stagger or to fall in a particular direction; secondly, symptoms referable to a compression or displacement or stretching of the cranial nerves lying in the posterior fossa.

Tumors in the middle lobe of the cerebellum uniformly produce cerebellar ataxia or a staggering gait. Tumors in the hemispheres of the cerebellum near to and compressing the middle lobe are attended by the same symptoms. So also are tumors which compress the peduncles of the cerebellum, especially the middle peduncles in their course to the pons, or the superior peduncles on their way toward the corpora quadrigemina. Tumors lying within the hemispheres which do not invade these parts do not always produce a staggering gait. Hence, a diagnosis of a tumor of the cerebellum is easy when the tumor lies near to the median line, but is impossible when it lies near the surface in the lateral portion of the hemispheres. Thus in the tumor shown in Fig. 122 there was no staggering. The early appearance of staggering as related to the general symptoms is, therefore, an important sign in favor of a cerebellar tumor; while the appearance of staggering many months after the development of optic neuritis will merely indicate that a tumor in the hemisphere has finally reached or encroached upon the middle lobe.

The early appearance of cranial nerve symptoms, such as paralysis of the face, deafness, disturbance in swallowing, symptoms referable to the vagus nerves or paralysis of one-half of the tongue, indicates that the tumor lies near to the medulla and pons, that is, upon the inferior surface of the cerebellum. The appearance of these symptoms late in the disease, long after the development of optic neuritis, will indicate that the growth has finally reached the inferior surface, or more probably that its size is so great as to have displaced the cerebral axis. Such a tumor is shown in Fig. 123.

The characteristics of cerebellar ataxia are a staggering gait with steps of irregular length and position, the body swaying like that of a person intoxicated. This ataxia of the legs and of the body is attended by a decided sense of vertigo, and does not usually persist when the patient lies down. These characteristics, together with the fact that the knee-jerks are exaggerated in cerebellar tumor, will enable any one to distinguish this disturbance of gait from that appearing in locomotor ataxia. Cerebellar ataxia is due to a disturbance in the mechanism of equilibrium in so far as this depends upon impressions coming to the brain from the muscles of the trunk and legs. When the staggering is in one direction only it is an indication that the middle peduncle of the cerebellum is invaded by the tumor. This unilateral staggering is not a common symptom. In four-fifths of the cases recorded the patient has staggered away from the side on which the tumor lies; in the remaining fifth the patient has staggered toward the tumor. The direction of staggering is not, therefore, an absolute sign of the side on which the tumor lies. When the middle peduncle of the cerebellum is invaded there are usually cranial nerve symptoms produced, and these are always upon the side on which the tumor lies. Hence, the combination of the staggering toward one side with cranial nerve symptoms of the opposite side will indicate inevitably which peduncle of the cerebellum is involved. Thus in the case shown in Fig. 123 the diagnosis was made before death from this combination of symptoms. When the superior peduncles of the cerebellum are invaded, nystagmus, paralysis of the ocular motor muscles, and blindness are developed. In fact it is impossible to differentiate a tumor invading the superior peduncles of the cerebellum from a tumor of the corpora quadrigemina.

Diagnosis. The review of the symptoms just described will convince the reader that in the majority of cases of tumor of the brain there are sufficient evidences of the existence of disease within the cranial cavity, and that the gradual development and progress of the disease will enable the physician to come to the conclusion that it is of the nature of a tumor. If in any case these symptoms which are present are carefully classified, the general symptoms being separated from the local symptoms, and the order of appearance of the local symptoms fully determined, it will usually be possible to reach a conclusion as to the situation of the tumor. The combination of local symptoms is sometimes as characteristic as their order of appearance and method of extension, as, for example, in cases of tumors of the crus or of the corpora quadrigemina.

It is not to be forgotten, however, that tumors of the frontal lobes and tumors of the temporal lobes, especially those situated in the right hemisphere, often fail to cause any local symptoms. The absence of distinct local symptoms, therefore, will point to these localities as the probable position of the suspected tumor.

It is not to be forgotten that many local symptoms are produced indirectly by pressure upon parts not far from the tumor, but yet not directly invaded in its growth.

It is also to be remembered that tumors pressing upon large vessels of the brain may so disturb the circulation as to produce symptoms quite similar to those of thrombosis, and these symptoms may be due to suspension of function of a part lying at some distance from the tumor.

The diagnosis of the variety of the tumor present can only be reached by careful study of the general history of the patient and by a consideration of those facts which have been already mentioned in the discussion of the varieties of brain tumor.

It is therefore evident that in the diagnosis of the disease there are always three questions to be settled: first, the existence of a tumor; secondly, its situation; thirdly, its variety. A study of the symptoms will usually enable the physician to reach an answer to the first two questions, but the answer to the last question will always remain uncertain.

The symptoms of brain-abscess may be the same in kind as those of brain-tumor, since both produce an increase of intra-cranial pressure and a progressive destruction of brain tissue. In their origin, mode of development, progress, and termination, however, there are not infrequently marked differences. Brain abscess develops most commonly after severe injuries or in conjunction with suppurative affections of the inner ear and of the nasal and orbital cavities, and with caries of any of the cranial bones which lie in contact with the membranes. These rarely occur prior to the development of brain-tumor. The symptoms common to tumor and abscess may develop after a blow on the head, but when the condition is one of abscess the symptoms appear in more rapid succession, with greater severity, and more frequently with fever than when the condition is that of tumor. Furthermore, the symptoms of abscess, after appearing suddenly, often subside, the abscess becoming latent, and all symptoms disappearing for months or years, and then breaking out again with suddenly fatal termination. This course contrasts markedly with that in a case of a tumor, where a gradual progress with slowly-increasing intensity of all the symptoms is found. The constant addition of new symptoms is usual in tumors, and a temporary remission rather than intermission of the symptoms is the rule when the progress is not continuous. There may also be some points of distinction found in the individual symptoms. Thus, headache is more severe and paroxysmal with tumor; optic neuritis is much more commonly found with tumor; mental changes are more gradual and constant with tumor; and local symptoms are slower in onset and more apt to develop with tumor. A complication of tumor not infrequent is cerebral hemorrhage. Meningitis is the usual complication of abscess. Lastly, a duration of from one to two years, with symptoms constantly present, points directly to tumor.

Tubercular meningitis is under certain circumstances easily mistaken for cerebral tumor. This is not true of the ordinary cases of tubercular meningitis with acute hydrocephalus, which develop marked symptoms rapidly and terminate fatally within four or six weeks. But there are a number of cases of tubercular meningitis which present a chronic course with gradual progress, and in which the diagnosis from tubercular tumor is almost impossible. It is true that the symptoms often develop rapidly in these cases, and yet this is sometimes apparently the case in tumor; for, unless a patient is carefully watched, the early symptoms of tumor may escape notice for some time. The symptoms of chronic tubercular meningitis may be the same as those described as general symptoms of brain tumor; but the headache is more severe in meningitis and more continuous; there is more likely to be hypersensitiveness to light, sound, or touch in meningitis; and optic neuritis develops less frequently, less rapidly, and with less intensity than in tumor. Tubercles upon the choroid are found more frequently in meningitis than in tubercular tumor. It is, of course, understood that a localized meningitis may give rise to the same symptoms as a small tumor, and then the differentiation is impossible. This is more common about the base of the brain, in the region of the cranial nerves, than elsewhere. It is also to be remembered that a chronic progressing meningitis may develop in the vicinity of a tumor. Here, again, the diagnosis will be impossible. A gradual subsidence of the symptoms, with recovery, will point to meningitis rather than to tumor.

Chronic hydrocephalus, while not infrequently the result of tumor or meningitis, may be due to a chronic inflammation of the ependyma of the ventricles. It then advances, and the fluid within the ventricles, producing pressure upon the brain, causes atrophy. The course is chronic, and the general symptoms are those of cerebral tumor. The local symptoms, however, differ in some respects from those of tumor. Spastic paralysis develops with chronic hydrocephalus without localized spasms, and is always bilateral; the lower limbs are affected more intensely than the upper. The child presents the extended, adducted, stiff legs, with overlapping knees, rigid muscles, increased tendon reflexes, and the spastic gait so familiar in infantile diplegia (see Chapter XVII); and, in addition, the hands move without proper co-ordination. As the disease progresses the crura cerebri and pons may be displaced by the pressure of the fluid, and irregular symptoms due to stretching of the cranial nerves may appear. These, with the paraplegia, may lead to a suspicion of a tumor of the pons or base of the brain, and only by the order of development of the symptoms can the differentiation be made.

The diagnosis between cerebral tumor and cerebral hemorrhage will be necessary only in a few cases in which the onset of the symptoms has been very sudden. There are a few cases of tumor, chiefly glioma, in which the growth has been latent for some time, and has then given rise suddenly, after a blow on the head, or exposure to the sun, or some other accidental influence, to well-marked symptoms. These are usually both general and local, more noticeably the latter. The suspicion of a tumor will be aroused if, after such an apoplectic stroke, the symptoms persist and increase instead of subsiding, and if headache, convulsions, and optic neuritis appear. Hemorrhage alone never gives rise to the last-named symptom.

There are a few general diseases which present certain symptoms somewhat similar to those occurring in brain tumor, and these should always be kept in mind. They are extreme anæmia, with defective vision, from myopia or hypermetropia; chronic lead-poisoning and chronic diffuse nephritis or contracted kidneys. The knowledge that these diseases may simulate brain tumor will lead the physician to be upon his guard. It is not necessary to mention the numerous points of the differential diagnosis which will enable a definite conclusion to be reached in any case.

The Course of the Disease. The general history of the progress of a patient suffering from brain-tumor has to some extent been indicated in the discussion of the symptoms. Suffice it to say that a gradual increase in the number and intensity of the various symptoms is usual. In some cases the general symptoms precede the local symptoms by several months, and optic neuritis does not often appear within three months of the beginning of the symptoms, unless the tumor is in the cerebellum or on the base of the brain. In some cases the local symptoms appear before the general symptoms, especially if the tumor is located in the motor cortex, when the case may be regarded for some time as one of cortical epilepsy until the general symptoms of brain-tumor supervene.[1]

As the case goes on, and both local and general symptoms become more numerous, the suffering of the patient becomes more intense.

If the case is one of gumma it may be possible to relieve the symptoms, and, by a progressive course of treatment, to cure the patient entirely. Under these circumstances the symptoms gradually subside and become less in intensity up to recovery. In other cases it is possible to locate the tumor absolutely in a position accessible to the surgeon and to remove it by operation, and under these circumstances the recovery of the patient is gradual but progressive after the operation is over. The brain resumes its functions after the pressure of the tumor is removed, or when a portion of the brain has been injured in the removal of the tumor a recovery may be imperfect, with some defects of sight, or motion, or sensation remaining. In those cases which are not subject to specific treatment, and in which the tumor cannot be removed, the course is progressively downward, the patient suffering more and more intensely as the tumor grows, and finally passing into a state of coma or dying in convulsions.

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[1] Kocher: Zeitsch. für Chirurgie, June, 1893.

The average duration of the symptoms is said to be three years, but individual cases vary greatly.

There are a few cases, however, in which, either spontaneously or under specific treatment, a tumor has ceased to grow, and the patient has apparently recovered and remained quite well for some months or even years, the brain apparently resuming its function. Two such cases have come under my observation. In one the recovery lasted four months, sudden death following, due to the rupture of a cyst which lay at the side of a sarcoma. In the other an interval of eight years occurred, after which the symptoms returned and caused death. In the last case optic neuritis, which was present at the first attack, subsided entirely, but recurred at the second attack. The tumor was a sarcoma in the cerebellum. In both cases there was no evidence of syphilis, yet the treatment, which was apparently successful, was inunctions of mercury and large doses (300 to 400 grains daily) of iodide of potassium.

Prognosis. It is evident from this statement of the course of the disease that the general prognosis in brain tumore is unfavorable. We have seen from a table on page 471 that but 7 per cent. of tumors of the brain are open to operation. It is therefore evident that in the vast majority of the cases we cannot give the patient any hope.

Treatment. 1. *Medical Treatment.* In the course of the case it is usually necessary to treat the symptoms of the disease. Headache can usually be very much relieved by a free use of phenacetin, antipyrin, or antifebrin. The doses of these drugs which must be used are larger than those commonly employed, and it is my practice to begin with the ordinary dose, and rapidly increase the number of doses given, combining with the drug any heart stimulant, caffeine being the one preferred. If the patient be carefully watched while this is being done it will soon be found possible to give safely twenty grains of antipyrin, fifteen to twenty grains of phenacetin, or ten grains of antifebrin at a dose, and this dose may be repeated after three hours, provided the headache returns. In a few cases the headache may be benefited by ice-bags to the head, by hot baths, or by ergot. If these remedies are useless, resort must be had to morphine, but this drug is especially unsatisfactory in the treatment of headache from brain tumors unless very large doses are given. Vomiting and vertigo in brain tumor are best relieved by the use of bromide of sodium or by hydrobromate of hyocine in $\frac{1}{100}$ gr. dose repeated every four hours.

The course of optic neuritis cannot be arrested, though it may be somewhat delayed by cupping the temples. While strychnine will at any time, when given hypodermically, improve temporarily the power of vision, yet it does no permanent good, and is thought by some to hasten the progress of the disease.

Epileptiform convulsions may be reduced in frequency by the free use of bromides, but cannot be arrested as long as the disease goes on. It is thus evident that the treatment of the general symptoms of brain tumor is exceedingly unsatisfactory. There is no treatment known that will in any way affect the local symptoms, massage and electricity to paralyzed limbs being mere palliatives, capable only of maintaining the nutrition of the muscles.

There is always a possibility in every case of brain tumor that the disease may be syphilitic in origin. It is always imperative, therefore, as soon as the diagnosis is established, to try the effect of specific treatment. My preference is for the use of inunctions of mercury, one drachm of blue ointment being rubbed in at night after a hot bath, a different part of the body being selected for each application, and after the application being covered with bandages so that the process of absorption of the ointment remaining upon the skin may occur during the night. At least two ounces of blue ointment should thus be used, and if improvement is evident the inunctions may be repeated after an interval of two weeks. The occurrence of salivation will necessitate the cessation of the use of mercury temporarily. At the same time large doses of iodide of potash should be given, beginning with twenty grains three times a day, and increasing the dose one or more grains daily until 100 grains three times a day have been reached. The iodide may be given in Vichy water or in milk, before or after meals, according to the condition of digestion. My preference is to give it before meals.

Care in the regulation of the diet, the use of simple and nutritious food, frequent massage to aid digestion, and a daily hot bath of temperature of 100° continued for fifteen to twenty minutes are essential during the taking of these large doses of iodide.

When the tumor is of a gummatous nature a decided improvement in all the symptoms, and particularly in insomnia and headache, should be observed within a month. Such improvement may, however, occur in cases of either sarcoma or glioma or cystic tumors. It is therefore necessary to keep up this treatment for two months longer. If the improvement continues and the patient gradually recovers it is probable that a gumma has been absorbed. In a case recently under my observation in which the symptoms in January, 1893, were intense headache, insomnia, mental apathy, staggering gait, great general weakness, optic neuritis in both eyes, with partial blindness in the right eye and total paralysis of the right third nerve, partial anæsthesia of the right side of the face, and paresis of the right sixth and seventh nerves, there was by November, 1893, a complete recovery, which still persists, an occasional nocturnal headache being the only symptom remaining. Five courses of inunction have been employed, and iodide has been given to the extent of 250 grains a day, the dose being varied from time to time when symptoms of intolerance appeared. Thus in ten months a basal gumma of considerable size has been absorbed by persistent treatment.

If the tumor present is a sarcoma or glioma, and an improvement has occurred during the first months of treatment, such improvement will not always persist, and therefore a return of the symptoms during the course of specific treatment is a pretty sure proof that the tumor is not of the nature of a gumma. It is useless to continue specific treatment after three months of thorough trial; it is better to refuse medical treatment or to depend entirely upon palliatives under these circumstances.

2. The *surgical treatment* of brain tumors is a subject which has awakened much interest of late, inasmuch as over 130 tumors have been operated upon within the past six years. Tumors in which surgical treatment is applicable are those which are situated in or near the cortex of the hemispheres, tumors of the cerebellum, though accessible being particulary unfavorable for operation.

Space does not permit of an analysis of these operations for brain tumor, and this subject will be discussed in another chapter. Suffice it to say that 47 per cent. of the cases in which operation has been attempted have proved successful, the tumor having been accurately located and successfully removed, with recovery of the patient. Considering that this operation is a new one, that it has been attempted rashly in some cases as a last resort, where there was no probability of success, and where failure was inevitable, it may be expected that the percentage of recoveries will in the future be much higher. It is, of course, a discouraging feature that but 7 per cent. of tumors of the brain are open to operation, and these statistics would indicate, therefore, that but three brain tumors in a hundred will probably be cured by surgical treatment. Nevertheless this treatment should be carefully considered, and whenever the diagnosis of a tumor in an accessible location can be made the surgeon should be called in as early as possible to remove it.

Horsley has recently advised that in cases in which the tumor is inaccessible a large opening be made in the skull and that a drainage-tube be passed within the dura to relieve, if possible, the intra-cranial pressure. I have seen this done; but it seems to me of problematical value, as it merely prolongs the misery in a hopeless condition.

The varieties of brain tumor which are most favorable to surgical treatment are sarcoma and fibroma. These tumors are encapsulated, are easily separable from the brain substance, and can be removed without any laceration of the brain tissue. Figure 124 shows such a tumor removed from the frontal region by Dr. McBurney at my suggestion.

Glioma and glio-sarcoma are not as easily removed, they usually infiltrate the brain substance, penetrating into the white matter even when they arise in the cortex, and it is necessary in their removal to incise the brain tissue. They are very vascular, and often contain cysts filled with fluid, and hence their removal is much more difficult. Both varieties of tumor, however, have been successfully removed. After removal they are liable to return, but in two or three cases operations have been done a second and even a third time, with the result of prolonging life.[1] When a gumma does not yield to specific treatment there is no reason why it should not be removed, provided it is accessible, and it is not unlikely that if a gumma were very large and were removed that specific treatment subsequently administered would eventually prove curative. The chief difficulty in the way of such removal is the infiltration of the pia with gummatous material and the difficulty of getting this away without great hemorrhage; this difficulty is not, however, insuperable.

Carcinoma of the brain is the most unfavorable tumor for operation, both because of the fact that it is usually secondary to carcinoma elsewhere, which is bound to kill the patient, or because of its liability to return. The same difficulties which attend a removal of glioma are met with in operating upon carcinoma.

Cysts of the brain are easily emptied when found, but the mere abstraction of the fluid gives no permanent relief, as it is bound to reaccumulate. It is necessary therefore either to drain the cyst, and thus secure its closure from the bottom by the approximation of its walls, or else to remove the whole of the cyst by dissecting it out from the brain. The latter is a dangerous process, and is sure to be followed by the formation of scar tissue, which in itself is a constant irritant to the brain. Drainage, therefore, appears to me to be the best form of treatment for cysts.[2] If, however, the cyst is merely a part of a glioma the removal of its wall and adjacent tissue gives most hope.

The removal of tubercular tumors has been already discussed in the section upon that subject.

I cannot but believe that the operation for tumor of the brain will be more widely performed in the future, that cases will be operated upon earlier in the disease as our knowledge of diagnosis increases, and that the day will come when as little fear will be felt in opening the cranial cavity as is at present felt in the opening of the abdomen. And I believe that the statistics at present available, which would seem to indicate that but 3 per cent. of brain tumors can be successfully removed, will be materially changed within the next ten years.

For details of the operation, the rules of guidance in finding various areas of the brain, and a general discussion of the subject, the reader is referred to the chapter upon Surgery of the Brain.

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[1] Czerny: Verhand. Deut. Gessel. für Chirurg., 1892.

[2] See Kocher: Zeitsch für Chirurg., April, 1893.

CHAPTER XVII.

FOCAL DISEASES OF THE BRAIN.
(CONTINUED.)

BY F. X. DERCUM, M.D.

THE CEREBRAL PALSIES OF CHILDHOOD.

CHILDREN, as a result of diverse pathological causes, operating both in intra-uterine life and in early childhood, and of various accidents to which they are exposed during birth, are subject to a variety of palsies presenting special clinical features. These palsies constitute a group by themselves.

Etiology and Pathology. As the name "cerebral palsy" implies, there is in every case a lesion in the motor area of the cortex, or at some point in the encranial portion of the motor tract, that is, in the white matter subjacent to the motor area, in the motor portion of the internal capsule, in the crus, pons, or pyramidal tracts. Clinically the cases resolve themselves into two groups; first, those in which the lesion is widely diffused and involves both sides of the brain, producing paralysis on both sides of the body, that is, diplegia; and secondly, those in which the lesion is limited to one side of the brain, producing unilateral palsy or hemiplegia. As might be conjectured, the detailed symptoms in a given case depend upon the location of the lesion and but little upon its nature. Similar symptoms may be produced by widely different causes, anything, in fact, that interferes with the nutrition or the development of the motor area or tract, such as inflammation, hemorrhage, thrombosis, neoplasm, etc. Unfortunately, our knowledge of the various morbid processes at work depends more upon inference than upon observation. It is very common to find at the autopsy atrophy and sclerosis of the convolutions, together with large cysts, but these changes are in the majority of cases the outcome of some pre-existing and acute process. They are evidently terminal in character, and must be regarded as a result rather than as a cause.

With reference to their etiology, cerebral palsies resolve themselves, *first*, into those beginning in intra-uterine life; *secondly*, those occurring as the result of accidents during birth; and *thirdly*, those acquired in infancy.

The *first* group presents the following etiological features. In some of the prenatal cases simple developmental arrest is the causal factor. The brain in such cases is embryonically defective and growth fails to take place, either because of lack of formative power or because of some gross anomaly in the vascular supply. A certain number of these cases, however, are due to gross pathological conditions, such as meningo-encephalitis, ependymitis, and, perhaps, hemorrhage and thrombosis. In syphilis and, perhaps, other infectious processes, we have a possible cause of prenatal vascular lesions, though the evidence upon this point is meagre.

Trauma of the abdomen of the mother, in rare instances, plays an important part. This is proven by the interesting case reported by Gibbs.[1]

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[1] Cited by McNutt: Apoplexia Neauotorum, Amer. Journ. of Obstetrics 1885, xviii. p. 73.

The child, which was stillborn, presented the symptoms of hemiplegia with atrophy and contractures, the latter being so marked that they could not be overcome without cutting the tendons. On the parietal bone of the opposite side was found an ecchymosis, while the hemisphere showed the remains of an old clot. There was a history of a severe blow to the abdomen of the mother during her pregnancy.

At times, and probably with truth, prenatal arrest of development has been attributed to a maternal impression, such as fright or other great emotional disturbance.

Occasionally children born prematurely present later on the symptoms of spastic palsy. Here feeble development of the motor cortex and tracts offers the most probable explanation. We should remember in this connection, however, that the crania of premature children are imperfectly developed, and that as a consequence they are less able to protect the brain from injury at birth. The two causes should not be confounded. In the one instance, there would be a history of a gradually acquired palsy; in the other, the history of a palsy dating from birth. In another, though rare class of cases, the children are apparently normal at birth, but there is such an extreme feebleness of development or instability of the motor tract that early degenerative changes take place. Freud[1] records two cases of cerebral diplegia in the children of a physician who had married his niece. The three remarkable cases of hereditary infantile spastic paraplegia recorded by Gee,[2] also belong to this group; father, daughter, and son were affected. As Freud points out, there is here a relation to Friedreich's ataxia. There is the same tendency to premature death—premature wearing out—of nerve tracts.

The *second* group, which includes cases having their origin in trauma during birth, is quite large. The head of the child in its passage through the pelvis is, as we know, exposed to varying conditions of pressure, and it cannot cause surprise that under certain circumstances, *e. g.*, when this pressure has been excessive or unduly prolonged, that encranial lesions ensue. The first to properly appreciate these facts and to recognize their relation to the spastic palsies of childhood was Little.[3] He tells us that the "severe lesions caused by the mechanical compression and laceration and extensive hemorrhage within the skull, when they do not destroy life, give rise to permanent deformity of the cranium, to atrophy of the injured portions of the brain, and are the causes of many cases erroneously described as congenital idiocy." It has been repeatedly demonstrated that the lesion in these cases is meningeal hemorrhage, and to Sarah McNutt[4] belongs the credit of clearly establishing the relation of these hemorrhages to the cerebral palsies (see Fig. 140). She showed, furthermore, that hemorrhages are apt to occur at the base of the brain in vertex presentations, and at the vertex in breech presentations. It is exceedingly probable when they occur at the base that vital structures are interfered with, and that as a consequence, the child promptly dies, while when they occur at the vertex the child lives on later presents the well-known symptoms of chronic spastic paralysis. The hemorrhage appears to come from the veins of the pia, and even at times from the longitudinal sinus. The effused blood causes compression, and possibly, also, inflammation of the central portions of the vertex. As already stated, the lesions commonly found are those of atrophy and sclerosis with or without the formation of cysts (porencephalus), changes which can certainly be regarded as terminal to meningeal hemorrhage. The lesion most frequently involves both sides of the brain, though it may be limited to one hemisphere. According to Sachs,[1] the congenital cases of hemiplegia almost equal in number the congenital cases of diplegia, and there can be no question that both forms are frequently the outcome of similar causes. It is not impossible, further, that the prolonged asphyxia to which children are sometimes exposed during birth is a factor in the subsequent cortical degeneration. Finally, the fact that the majority of congenital cerebral palsies occur in first-born children is a confirmation of the importance of birth traumata as etiological factors. A few cases of cerebral palsy have been traced to injury by the forceps. Thus, Osler[2] found nine cases of such injury in one hundred and twenty infantile hemiplegias, all told. It is certain, however, as Gowers[3] points out, that the injury is due less frequently to the forceps than to the dystochia which renders their employment necessary.

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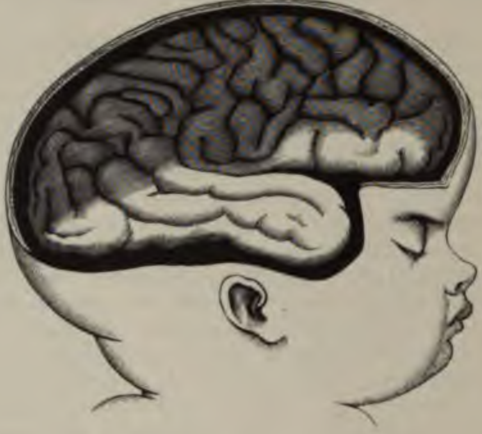
[1] Freud, Ueber Familien Formen des Cerebralen Diplegia, Neurolog. Centralbl., 1893, xii. pp. [illegible]42.

[2] Gee, Hereditary Infantile Spastic Paraplegia, St. Bartholomew Hosp. Reports. 1889. xxv. p. 81.

[3] Little, On the Influence of Abnormal Parturition, Difficult Labors, Premature Birth, and Asphyxia Neonatorum on the Mental and Physical Condition of the Child, Especially in Relation to Deformities. Obstetrical Transactions, 1862, vol. iii. p. 263.

[4] Loc. cit.

FIG. 140.



Showing the location of a meningeal hemorrhage occurring at birth. (McNUTT.)

In the *third* group, which includes the acquired cases, a number of causes are found, and among these the infectious diseases play the dominant *rôle*. Cerebral palsies have been observed to occur during or just after scarlet fever, measles, whooping-cough, typhoid fever, diphtheria, and even vaccinia. In a certain number of autopsies in more recent cases unmistakable evidences of hemorrhage, thrombosis, embolism, and other changes have been found. In children, we should remember, meningeal and cortical hemorrhage are far more frequent than in adults, and this is probably true of other vascular derangements. Vascular lesions were present in sixteen out of ninety autopsies of hemiplegia collected by Osler.[4] In what manner these lesions are produced is not definitely known. It is probable that in some instances a local arteritis results in thrombosis and in others in hemorrhage, while in infectious diseases complicated by endocarditis, embolism is the

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[1] B. Sachs: Cerebral Paralysis of Early Life. Trans. of the Tenth Internat. Med. Congress, vol. iv. p. 128.

[2] Osler: The Cerebral Palsies of Children. London, 1889.

[3] Gowers: Dis. of the Nervous System, 1894, ii. p. 414.

[4] Loc. cit.

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Occasionally children born prematurely present later on the symptoms of spastic palsy. Here feeble development of the motor cortex and tracts offers the most probable explanation. We should remember in this connection, however, that the crania of premature children are imperfectly developed, and that as a consequence they are less able to protect the brain from injury at birth. The two causes should not be confounded. In the one instance, there would be a history of a gradually acquired palsy; in the other, the history of a palsy dating from birth. In another, though rare class of cases, the children are apparently normal at birth, but there is such an extreme feebleness of development or instability of the motor tract that early degenerative changes take place. Freud[1] records two cases of cerebral diplegia in the children of a physician who had married his niece. The three remarkable cases of hereditary infantile spastic paraplegia recorded by Gee,[2] also belong to this group; father, daughter, and son were affected. As Freud points out, there is here a relation to Friedreich's ataxia. There is the same tendency to premature death—premature wearing out—of nerve tracts.

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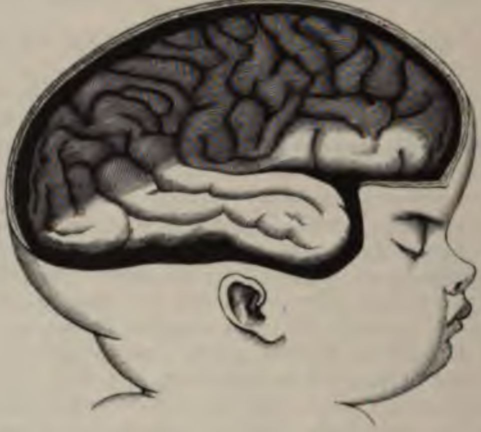
[1] Freud: Ueber Familien Formen des Cerebralen Diplegia, Neurolog. Centralbl., 1893, xii. pp. 512, 542.

[2] Gee: Hereditary Infantile Spastic Paraplegia, St. Bartholomew Hosp. Reports. 1889. xxv. p. 81.

[3] Little: On the Influence of Abnormal Parturition, Difficult Labors, Premature Birth, and Asphyxia Neonatorum on the Mental and Physical Condition of the Child, Especially in Relation to Deformities. Obstetrical Transactions, 1862, vol. iii. p. 263.

[4] Loc. cit.

FIG. 140.



Showing the location of a meningeal hemorrhage occurring at birth. (McNUTT.)

In the *third* group, which includes the acquired cases, a number of causes are found, and among these the infectious diseases play the dominant *rôle*. Cerebral palsies have been observed to occur during or just after scarlet fever, measles, whooping-cough, typhoid fever, diphtheria, and even vaccinia. In a certain number of autopsies in more recent cases unmistakable evidences of hemorrhage, thrombosis, embolism, and other changes have been found. In children, we should remember, meningeal and cortical hemorrhage are far more frequent than in adults, and this is probably true of other vascular derangements. Vascular lesions were present in sixteen out of ninety autopsies of hemiplegia collected by Osler.[4] In what manner these lesions are produced is not definitely known. It is probable that in some instances a local arteritis results in thrombosis and in others in hemorrhage, while in infectious diseases complicated by endocarditis, embolism is the factor at work. Strümpel has advanced the theory that some of these cases are due to an inflammation of the cortex, the lesion being similar to that which takes place in acute poliomyelitis. That a polioencephalitis corticalis acuta actually exists as a pathological entity remains to be proved. The symptoms, however, often suggest this lesion. This was the case, for instance, in twenty cases out of one hundred and sixty collected by Sachs and Peterson.[1] In a large number of cases the paralysis is ascribed to convulsions. Thus, twenty-one out of sixty-four cases in the collection of Sachs and Peterson, in which the causes had been ascertained, presented convulsions as the initial symptom. It is exceedingly difficult to estimate properly the value of these facts, as it is probable that the convulsions are more frequently the *result* of the lesion than a primary cause. It is not, however, inconceivable that a toxic agent acting upon the cortex should produce convulsions, and that these, if sufficiently intense and prolonged, should lead to permanent changes in the nutrition of the cortex. Trauma also plays a *rôle* in some of the acquired cases. Thus, penetrating wounds and, more especially, falls upon the head are occasionally followed by the symptoms of cerebral palsy. In one instance, in a patient in the Pennsylvania Institution for Feeble-Minded Children, hemiplegia followed ligation of the carotid artery. As the reader has probably inferred, acquired palsies are most often hemiplegic in type. Thus, of ninety-one cases, according to Sachs and Peterson, eighty-three presented the symptoms of hemiplegia.

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[1] B. Sachs: Cerebral Paralysis of Early Life. Trans. of the Tenth Internat. Med. Congress, vol. iv. p. 128.

[2] Osler: The Cerebral Palsies of Children. London, 1889.

[3] Gowers: Dis. of the Nervous System, 1894, ii. p. 414.

[4] Loc. cit.

As the reader has already learned (see Chapter XV.) lesions of the motor area of the cortex are followed by a degeneration in the fibres of the motor tract. In various autopsies in the cerebral palsies of childhood these degenerations have been traced. Occasionally, however, all evidence of these secondary scleroses is wanting. Thus Gierlich[2] found an atrophy of the motor tracts in the pons, the medulla and in the spinal cord down to the lumbar region, but there was no degeneration. Such nerve fibres as were present were normal in appearance. The number of fibres, however, was diminished, especially in the pons. Gierlich offers as an explanation that in destruction of the motor centres in embryonal or infantile life the encasing of the axis cylinders with myelin ceases, and that the axis cylinders thus unprotected degenerate and are absorbed through the active metabolic processes of childhood without any reactive inflammation taking place in the interstitial tissue. This ingenious theory has much to commend it. It renders clear facts which otherwise seem inexplicable.

Hemiplegia and diplegia, as we have seen, may be due to the same morbid process, and either form may be congenital or acquired. However, the large majority of the congenital palsies are diplegic, while the acquired palsies are most frequently hemiplegic; and, though a sharp distinction cannot be drawn between them, each form, notwithstanding, presents an average of peculiarities which renders a separate consideration desirable.

Hemiplegia. The relative frequency of hemiplegia may be inferred from the statistics of Osler,[3] who in a collection of 151 cases of all forms found 120 presenting this symptom. That it is only infrequently congenital is proven by the collection of Sachs and Peterson,[4] who found only 22 congenital out of 105 cases. As regards sex, Osler found in 135 cases 75 girls and 60 boys, while Sachs and Peterson in 140 cases found 42 girls and 63 boys. It cannot be said, therefore, that either sex predominates. Again, as regards the side affected, Osler found it occurred 79 times on the right side and 56 times on the left side, while Sachs found it to occur 52 times on the right side and 53 times on the left side. It is fair, therefore, to assume that neither side is especially predisposed. The great majority of the acquired hemiplegias occur within the first two or three years of childhood. This statement is borne out by both the statistics of Osler and by Sachs and Peterson.

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[1] Sachs and Peterson: Journ. of Nervous and Mental Diseases, 1890, xvii. p. 295.

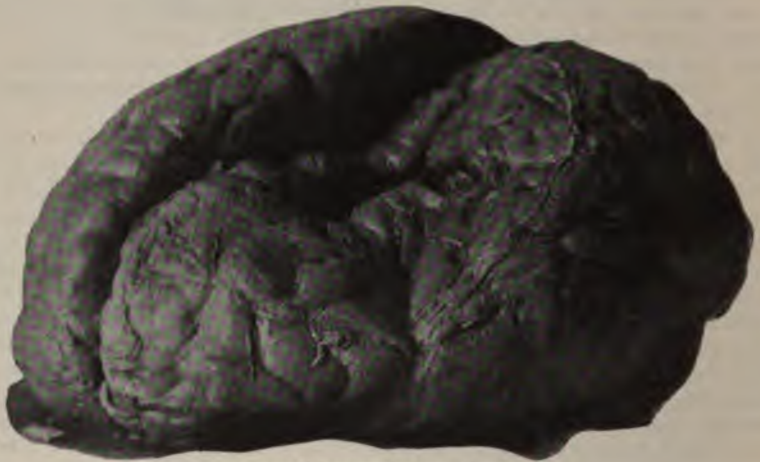
[2] Gierlich: Ueber secundäre Degeneration bei cerebraler Kinderlähmung. Archiv f. Psychiatrie, 1891, vol. xxiii. p. 201.

[3] Loc. cit.

[4] Loc. cit.

The morbid anatomy of hemiplegia based on 90 autopsies collected by Osler, is as follows: Sixteen cases, none of which survived the attack long, suffered from thrombosis, embolism or hemorrhage. In seven there was plugging of the Sylvian artery, in nine hemorrhage. Three were congenital, one was under three years, six were over ten years. In 50 cases atrophy and sclerosis were found. The sclerosis was usually diffuse and involved either an entire hemisphere or a single lobe, or it was confined to one or two convolutions. In a few cases it was found in patches. Nodular projections of sclerosed tissue, that is, hypertrophic scleroses, were sometimes found. In 24 cases porencephalus (see Fig 141) was found; that is, cysts filling up space normally occupied by brain tissue, and extending down from the surface of the brain to a variable depth into its substance. (For a detailed account of this condition see page 319.) In six the paralysis was congenital; in eighteen it was acquired; in eleven of these it came on in early life and after convulsions. The extent of the lesion varied from a few convolutions to half of the hemisphere. In all of the cases the lesions involved the motor area. In some instances the primary lesion is undoubtedly a meningitis. Oliver[1] has reported one such instance. In rare instances the symptoms of infantile hemiplegia are the result of a tumor involving the motor area. Such a case is recorded by Donkin[2] in which a tuberculous tumor involved the Rolandic region. Another instance is shown in Fig. 142, in which the left motor area was invaded by an extensive angiolithic sarcoma. There was marked contracture and retardation of growth of the right arm and also some arrest of growth in the leg.

FIG. 141.



A case of porencephalus. (LLOYD.)

Diplegia. This form, as we will presently see, resolves itself into two subgroups; first, *diplegia proper*, that is those cases in which both upper and lower limbs are affected on each side of the body; and, secondly, *paraplegia*, in which the paralysis is limited to the legs, or in which it is very slightly marked in the arms. Though congenital in the large majority of cases diplegia may also be acquired. Thus Sachs and Peterson[1] present twenty cases of congenital diplegia, seven of congenital paraplegia, and but four each of acquired diplegia and paraplegia. The causes of congenital diplegia we have already sufficiently considered. In the acquired cases Sachs found that one resulted from convulsions, two followed measles, one began with fever, one followed cerebro-spinal meningitis, two hydrocephalus, while in still another the cause was unascertained. The morbid anatomy of cerebral diplegia proper is based upon sixteen autopsies, also collected by Osler. All of the cases revealed destructive lesions of the motor centres of the cortex, diffuse atrophic sclerosis being most common. Descending degeneration was present in some cases, not noted in others, and absent in one when searched for. Rarely extensive internal hydrocephalus gives rise to the symptoms of spastic diplegia. Such an instance is presented in Fig. 143.

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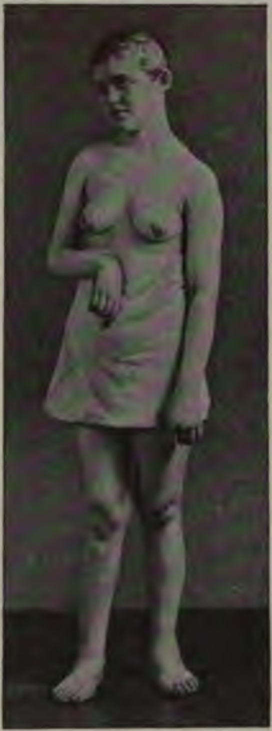
[1] Oliver: British Medical Journal, 1890, vol. i. p. 284.
[2] Donkin: Westminster Hospital Reports, 1891, vol. vii. p. 1.

FIG. 142.



Spastic hemiplegia from angiolithic sarcoma of the brain, the symptoms dating from five years of age. Jefferson Hospital.

But one record of autopsy, that of Foerster, was found by Osler in cerebral paraplegia. In this there was general cortical sclerosis, slight dilatation of the lateral ventricles and some descending degeneration. To this we must add another autopsy recorded by Ferguson,[2] who found a patch of sclerosis at the upper end of each fissure of Rolando, with descending degeneration in the motor tracts, except in the columns of Türck.

Symptoms. The symptoms and course of the affection vary in accordance with the mode of origin, that is, whether prenatal, congenital or acquired, and also in accordance with the extent and distribution of the lesion. In the prenatal cases where there has been marked arrest of development, such striking features as microcephaly or other deformity of the cranium, with more or less marked hemiplegia, single or double, with or without contractures, may be observed at birth.

In the cases due to trauma at birth, we have generally the history of a protracted or difficult labor of a first birth. When born the child may present symptoms more or less marked of asphyxia, and frequently it suffers from convulsions. Owing to the child's general condition, the paralysis may not be noticed, and indeed several days may elapse before it attracts attention. Sooner or later, however, it becomes very evident. Convulsive seizures, if they occur at this time, are oft repeated, and may be general or limited to one side. After these symptoms disappear, and as time passes, we observe that the development of the child does not follow a normal course. Thus, it does not learn to walk at the proper time. Walking is very much delayed, and in many instances never acquired. If the child be closely examined it is found that there is more or less marked rigidity in both arms and legs, and that there is a tendency in the arm to assume the position of contracture seen in adult hemiplegics in whom secondary degeneration has occurred, while the legs become extended and firmly adducted. Often one leg is crossed over the other. In diplegias, to which this description especially applies, the legs suffer, as a rule, more severely than the arms. Indeed, as already stated, the arms are occasionally very little involved, and in some cases not at all, the disease then constituting the sub-group above mentioned as cerebral paraplegia.

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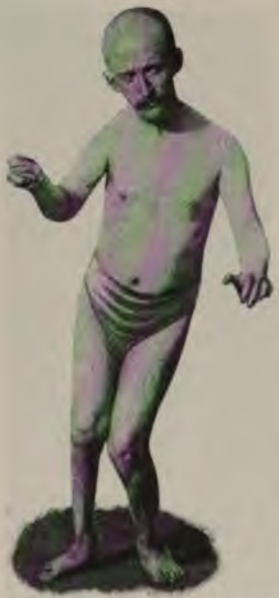
[1] Loc. cit.

[2] Ferguson: American Journal of Obstetrics, 1891, vol. xxiv. p. 928.

FIG. 143.



Diplegia from internal hydrocephalus, Philadelphia Hospital.

If a hemiplegia has resulted, the symptoms are likewise those of a spastic palsy; but in this instance, contrary to what we find in the diplegias, the arm is much more affected than the leg. Further, we notice as the child develops, that the limbs on the hemiplegic side fail to grow as rapidly as their fellows, and that, when compared with these as time progresses, they appear, in addition to the contractures which they present, much smaller. As could be expected upon *à priori* grounds, the tendon reactions are markedly increased, that is, if the contractures are not so severe as to prevent motion of the limbs. Thus in hemiplegia the knee-jerks are plus, while ankle clonus is often obtained. In the diplegic form, while the knee-jerks are plus it is impossible often to obtain an ankle clonus, owing to the rigid fixation of the foot.

In the acquired forms the symptoms may, as already stated, come on as a result of various infectious diseases and trauma, but may also make their appearance in the midst of apparent health. The onset, as a rule, is sudden, generally with convulsions, which may be intermittent and repeated. Some fever, though this is generally slight, may accompany the attack. Occasionally, however, the range of temperature may be very high, 107° F. being mentioned by Sachs.[1] During the convulsion the child is in a condition of coma, and paralysis may not be noted until consciousness returns. At times, however, the convulsion is more marked on or limited to one side—the side which afterward reveals paralysis. The lesion having been produced, the symptoms follow the course pursued by other cerebral palsies. Most frequently, as we have seen, a hemiplegia is established, though sometimes a diplegia results.

A number of symptoms pertaining more or less to all forms remain to be noted. Mental defects are present in many cases. That they are especially marked in the prenatal forms, those in which there has been cortical agenesis, is not to be wondered at. That they should also be frequent in the congenital forms and in those acquired cases in which the affection has come on in the earliest years of infancy, is merely what we would expect to find. Thus Sachs and Peterson[2] in their statistics of eighty cases found that fourteen presented idiocy, fifteen imbecility and six feeble-mindedness. Of those in which the palsy had been acquired under three years, six presented idiocy, eighteen presented imbecility and ten feeble-mindedness. Of those from three to five years none presented idiocy, three imbecility, and two feeble-mindedness; of those from five to ten years, three presented imbecility and two feeble-mindedness, while of six acquired after ten years of age, but one presented feeble-mindedness. The inference from these figures is obvious. The earlier the onset the greater the mental impairment. Further, the proportion of grave mental defects, such as idiocy and imbecility, is greater in those cases in which the lesion has been most extensive. Thus in seventeen cases of diplegia, eight presented idiocy, seven imbecility and two feeble-mindedness. In nine cases of paraplegia again, more than half (five) presented idiocy, one imbecility and three feeble-mindedness. Of forty-five cases of hemiplegia, but seven presented idiocy, thirty-one presented imbecility and sixteen feeble-mindedness. In another case there existed epileptic insanity.

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[1] Sachs: Cerebral Hemorrhage, Thrombosis and Embolism, Keating's Cyclopædia of Diseases of Children, 1890, vol. iv. p. 537.

[2] Loc. cit.

Cerebral palsies are frequently complicated by epilepsy. This is present, especially in the hemiplegic form. Most frequently it does not make its appearance until one or more years have elapsed after the onset of the paralysis. It shows a disposition, further, to persist and often to increase, and is of itself in many cases responsible for the imbecility present. It may begin in and may be limited to the limbs of the affected side, or it may be general. In diplegic palsies convulsive seizures frequently occur at birth or for a brief period thereafter. The tendency is to an early cessation of the attacks. In but few instances do they develop into epilepsy. On the other hand, about 50 per cent. of all hemiplegics are epileptics.[1]

In addition to the various symptoms thus far detailed various disorders of motion may be added to the paralysis. Thus there may be tremor, chorea, spasms or athetosis. (See p. 264.) In Osler's collection, thirty-one cases of hemiplegia presented such disorders. One presented tremor, twenty-four presented chorea, and six cases presented mobile spasms with athetosis. In diplegic cases ataxic and athetoid movements most exaggerated in kind may be present. When spasm and chorea are equally associated it has been been termed chorea spastica. When the hands, fingers and toes present slow, irregularly recurring movements the condition is termed athetosis, and when they are present in diplegic cases the condition is termed double athetosis. (See Fig. 144.) Post-hemiplegic disorders of movements were first described by Weir Mitchell. No change has ever been observed in the electrical conditions of the muscles. Sensation is never lost or even impaired in the cerebral palsies of childhood. In a few cases there is hypersensitiveness to touch (hyperæsthesia).[2]

As we have seen, the various forms of cerebral palsies present many symptoms in common. Each form, however, presents a number of special features. Some of these have already been alluded to, but our purpose will be served best by rehearsing briefly the symptoms especially pertaining to each group.

HEMIPLEGIA. This, as we have seen, is most frequently acquired. The onset is characterized by convulsions and coma. In a few cases these symptoms are absent. When present they are associated with fever, which may be transient or persistent, usually not very high. At times the onset is associated with delirium. Vomiting is also occasionally noted, and sometimes there is soreness of the general surface. When consciousness returns the hemiplegia is discovered. It is usually, though not always, profound. It is marked in the arm, leg, and sometimes in the face. In the latter the lower half only is involved, the orbicularis palpebrarum and brow muscles escaping. The hemiplegia is thus of the type met with in the adult. After a time signs of recovery set in. The paralysis in the majority of cases dis-

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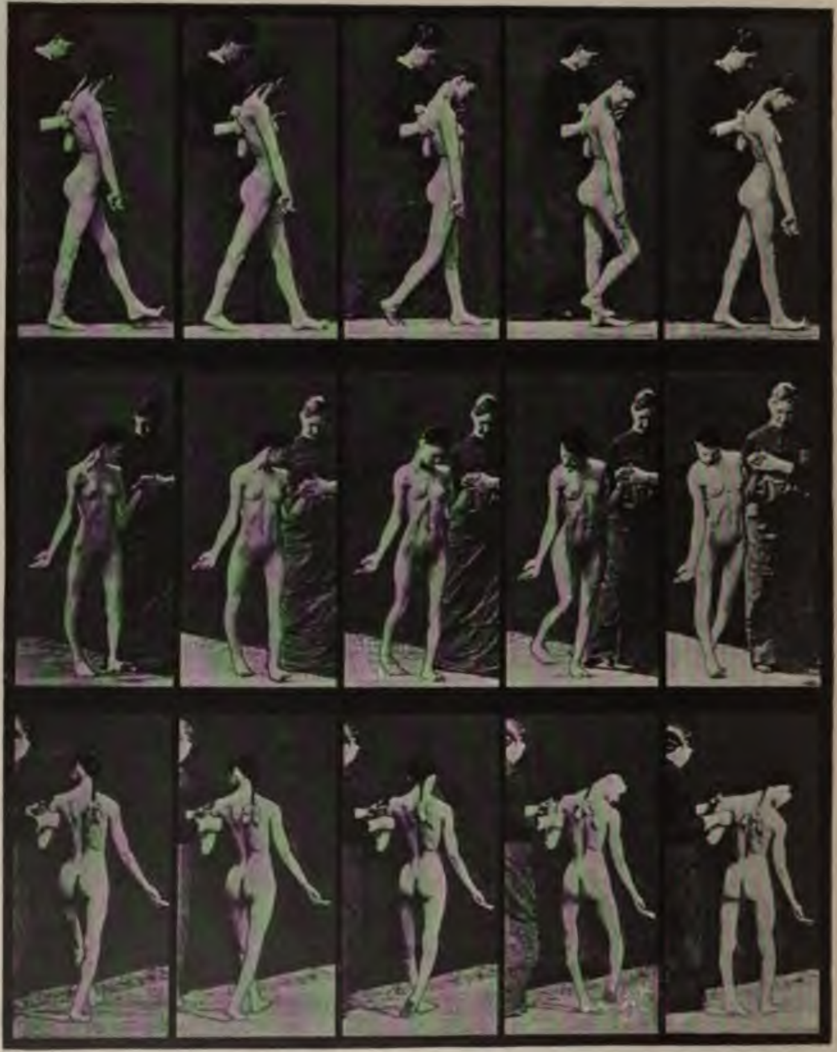
[1] Because of the intimate relation existing between cerebral palsies and epilepsy Freud goes so far as to consider common epilepsy as a congenital or acquired disease (not a neurosis) in which the diplegia or hemiplegia pertaining thereto has disappeared. This he claims is also true of many cases of imbecility and idiocy. Revue Neurolog., 1893, i. p. 177.

[2] Railton: British Med. Journ., 1891, i. p. 1382.

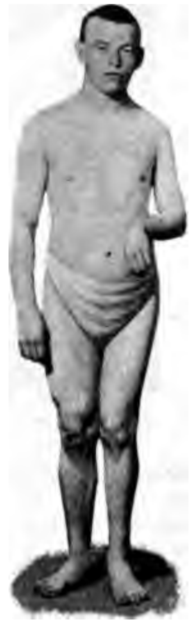
FIG. 144.



Illustrating the walk of a young girl presenting spastic diplegia with choreiform and athetoid movements. From serial photographs made simultaneously from three different points of view by Eadweard Muybridge from a patient of the writer.

appears from the face altogether, becomes less pronounced in the leg, but remains marked in the arm. In rare instances the facial paralysis persists. Thus in a case reported by Koenig[1] it persisted in the face, while it disappeared from the limbs.

FIG. 145.



Spastic hemiplegia with epilepsy. (Philadelphia Hospital.)

After a time the child recovers sufficiently to resume walking, but the gait is hemiplegic. Sooner or later rigidity of the paralyzed side makes its appearance. In a few cases (in two noted by Sachs) it fails to appear. The arm assumes the position seen in the secondary contracture of the hemiplegia of the adult. The leg suffers less from rigidity than the arm, but the tendency is for it to assume the position of extension. The knee-jerks are exaggerated, and ankle-clonus is often present. Choreiform or athetoid movements may also make their appearance. In one instance Féré[2] noted a peculiar intermittent contraction in the fascicles of the large muscles of the paralyzed limbs. The arm grows at a lessened rate than its fellow. This is also true of the leg, though to a less extent. By the time adult life is reached the difference in the limbs of the two sides is generally very great, the arm appearing when compared with its fellow as though atrophied. The difference in the length of the legs is generally sufficient to cause a more or less marked limp in the gait. In some cases vasomotor disturbances, such as coldness and blueness of the extremities is noted. Often the hemiplegia is accompanied by aphasia. This is frequently temporary, but at other times the power of speech returns only after months and years. The tremor, choreiform movement and athetosis which appear in the paralyzed limbs in some of these cases have already been considered. These disorders of movements are, however, much less common than in the diplegic form. They were present in but thirty-two cases out of one hundred and twenty collected by Osler. The relation of mental defects and of epilepsy to hemiplegia have already been considered.

The student must remember that the above description is applicable to the vast majority of cases. In rare instances aberrant symptoms are met with, due doubtless to peculiarities in the location of the lesion. Thus Menz[3] reports a case of the sudden paralysis of one side occurring in a child of ten months, followed by slow improvement, but with the appearance of hemichorea and hemiathetosis. Associated with these phenomena was the very unusual one of bilateral oculo-motor palsy most marked on the side opposite the hemiplegia. The lesion is supposed by Menz to have been in the left crus. Wallenburg reported a similar case.

DIPLEGIA. This group, as already stated, falls naturally into two sub-groups, the *diplegias proper* and the *paraplegias*. There is usually a history of injury at birth or of difficult labor. Shortly after birth, or perhaps at the time of birth, it is noted that the limbs of the child on both sides are not moved as freely as they should be, and that they are more or less rigid. Occasionally these symptoms are observed to follow a febrile attack or a convulsion. There is no local wasting observable in any case.

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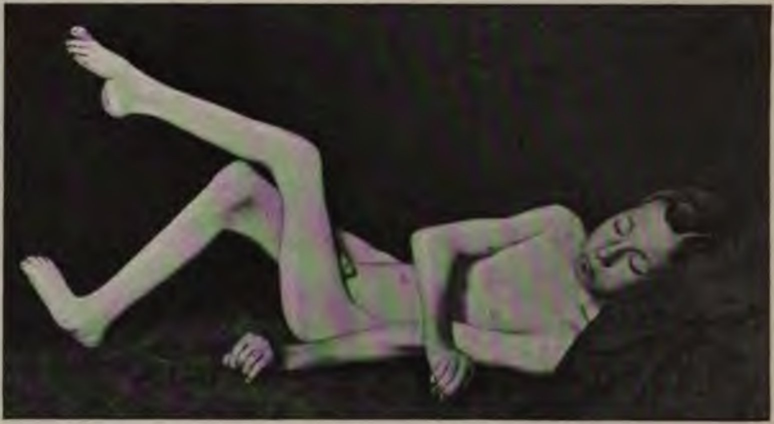
[1] Koenig: Deutsche Med. Wochenschr, 1893, xix. p. 1014.
[2] Nouvelle Iconograph. de la Salpêtrière, 1890, iii. p. 82.
[3] Menz: Wiener klin. Wochenschrift, 1892, v. p. 604.

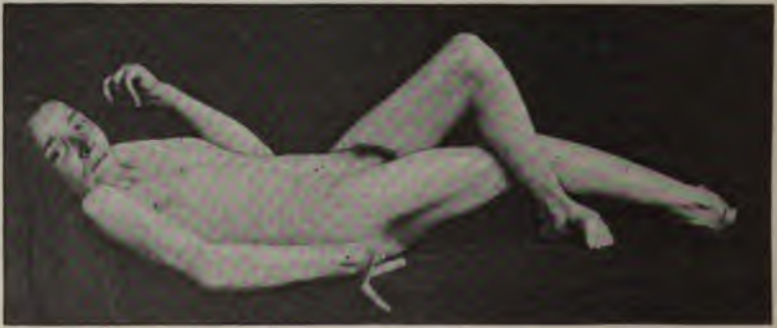
However, there is usually a general retardation of growth. The paralysis is always accentuated in the legs. The latter are firmly adducted, extended, and frequently crossed at the knees, while the feet assume the position seen in talipes equinus or equino-varus. The knee-jerks can be elicited in most cases, and are much exaggerated. The ankle, as a rule, is so fixed by contracture that no clonus is obtained. The arms present, in addition to marked rigidity, spasm, exaggerated and choreiform movements. The condition is further complicated by an association of movement. When a child makes an attempt to grasp an object with one hand the opposite member usually opens and closes in unison. The majority of these patients are unable to walk. When walking is possible the movements are very irregular, the tendency to adduction producing a gait known as cross-legged progression, whilst the effort is attended by an increase in all of the choreiform and athetoid movements. These movements affect not only the legs and arms, but may

FIG. 146.



Spastic diplegia, congenital; presenting choreiform movements. (Philadelphia Hospital.)

FIG. 147.



Spastic diplegia, congenital; presenting choreiform and athetoid movements. (Philadelphia Hospital.)

also affect the face, so that the latter is constantly distorted by rapidly changing and grotesque grimaces. Among rare motor disturbances should be mentioned intention tremor. Railton[1] reports a case in which such a tremor was present in the arms and legs, the resemblance to disseminated sclerosis being quite marked. As already pointed out, the mental defects in these cases are generally profound. They have already been considered. Speech when acquired is usually drawn, hesitating and spasmodic in character. Frequently these patients can with difficulty be understood.

Paraplegia. Here the symptoms of spastic diplegia are met with as before, but on the whole, to a much less marked degree. As the name implies, the condition is limited to the legs, though in a small number the arms are slightly involved. The legs present the phenomena of rigidity with exaggerated knee-jerk. The condition may be noted at birth or may make its appearance shortly after. A history of difficult labor is often present. Occasionally the paraplegia is acquired. The tendency to extension is so marked that the feet assume the position noted in diplegia proper, namely, talipes equinus or equino-varus with marked adductor spasm. (See Fig. 148.) The condition may be comparatively slight or very pronounced. Many of these patients are able to walk with comparatively little difficulty. The

Fig. 148.

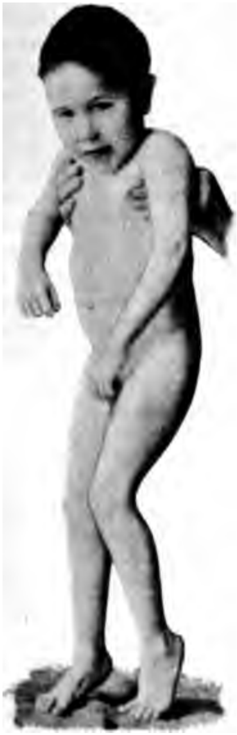


Fig. 149.

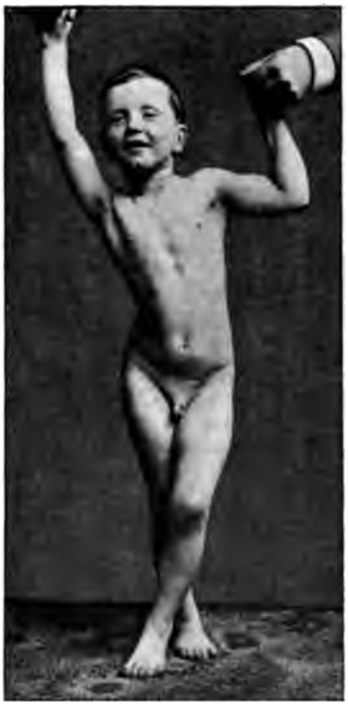


Fig. 148.—Marked plastic paraplegia; walking or standing alone impossible. Infirmary for Nervous Diseases, Philadelphia.

Fig. 149.—Spastic paraplegia; crossed-legged progression. (Jefferson Hospital.)

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[1] Loc cit.

limbs being more or less adducted, the patient tends to throw one leg in front of the other as he walks. (See Fig. 149.) In other words, there is cross-legged progression. Intelligence is often impaired, but to a much less extent than in diplegia proper.

Diagnosis. Cerebral palsies present symptoms so characteristic that error in diagnosis is not likely to occur. Difficulty can only arise in the early stages of the acquired forms. As pointed out by Strümpel, poliomyelitis and acute cerebral palsy are frequently ushered in by similar symptoms; but in such cases the progress of the case and the hemiplegic type of the symptoms will enable us to make the differential diagnosis. It is also easy, as a rule, to relegate a given case to its proper group, namely, to decide whether it is hemiplegic, diplegic, or paraplegic. It is not, however, by any means easy to form an opinion in most cases regarding the nature of the lesion. It is important, first, to determine whether the lesion in a given case was prenatal, whether it is due to trauma at birth, or, lastly, whether it was acquired. Here the history and special symptoms already detailed come greatly to our assistance, and if they can be relied upon the facts determined are often of great value to the family of the patient. Thus, if it be shown that a given case is acquired the fact is of far less significance in the family history than if the evidence points to a prenatal origin.

Cortical lesions predominate largely in the cerebral palsies of childhood. If in a given case we have coma, convulsions or delirium, the inference is generally justified that the lesion is cortical. However, as is well known, extensive intra-cerebral hemorrhage may also give rise to coma and initial convulsions, but the convulsions are not repeated. In meningeal hemorrhage the convulsions recur with great frequency. The differential diagnosis between thrombosis, hemorrhage, and embolism must be made upon general principles, the symptoms not differing in children from those in adults, and for the consideration of these the reader is referred to Chapter XV. It should also be remembered that at most this differential diagnosis can only be raised to a degree of probability. Fortunately, these distinctions have but little practical value.

Care should be taken not to overlook tumors in the Rolandic region, which in children, as we have already seen, may give rise to symptoms closely resembling those of ordinary infantile hemiplegia. This point is of especial importance, because tumors in the Rolandic area are surgically readily accessible. If in a given case a legitimate doubt exists, there is no objection to an exploratory trephining.

Prognosis. The prognosis differs somewhat in the various forms. Thus, diplegia proper, the result either of prenatal or congenital causes, offers but little prospect of improvement. It will be remembered that grave mental defects are apt to occur, idiocy being indeed very common. Striking exceptions to this rule are, however, occasionally met with, and the physician should make his prognosis with due caution. It is impossible in a given case to say just to what extent the intellect will be affected. In the paraplegic form the prognosis is distinctly less unfavorable than in diplegia proper. From what we know of the morbid anatomy the brain lesion is far less extensive, and in keeping with this fact the disturbance of the intelligence is much less marked. Many paraplegic children present no appreciable defect.

As regards the paralysis, considerable can be accomplished in some cases. Every now and then a child unable to walk acquires this power with increasing development. In this respect the prognosis is much more favorable in paraplegia.

As regards the acquired form, the prognosis must be considered under two separate heads: first, the prognosis of the affection at the time of the onset; and, secondly, at the time of the established disease.

Inasmuch as cerebral lesions so often follow convulsions, and are so often the outcome of infectious diseases, the physician should, when attending a child suffering from sudden grave cerebral symptoms, especially convulsions and coma, state to the family the possibility of a paralysis resulting. He should be especially careful not to belittle an attack of infantile convulsions, nor should he feel justified in an entirely favorable prognosis, even if no paralysis be discovered at the time, as its appearance may be somewhat delayed. A paralysis having been once established, and involving, as it most frequently does, the face, arm, and leg, it is safe to assume that the paralysis of the face will disappear, and that the paralysis of the leg will grow so much less that the child will in time again be able to walk. However, the physician should not neglect to state to the relatives that this result does not invariably follow, and, further, he should lay emphasis upon the fact that recovery is rarely complete. It is generally accompanied by a diminished rate of growth in the limbs of the affected side, so that the leg will not only be stiff and awkward in its movements, but in course of time will be shorter than its fellow. The arm especially will be small and contracted. Absolute frankness on the part of the physician to the patient's family often saves him from unjust criticism. Paralysis and contractures having been once established, the prognosis differs in no respect from that in the diplegic forms. The paralysis, depending upon structural change, can be but slightly improved. The contractures, however, may be materially benefited by treatment.

Treatment. Little can be said in regard to preventive treatment, except in those forms due to traumata at birth. The relation of prolonged and difficult labor to cerebral palsies has already been considered. Too often in works upon obstetrics the dangers to the mother alone are considered, while the equally grave dangers to the child are neglected—dangers which are not only immediate, but which have the most profound bearing upon the future intellectual and physical well-being of the child. There can be no doubt that in a number of cases labor should be hastened for the sake of the child, and it is probably true that more cases of cerebral palsy are due to hesitation in the application of the forceps than could possibly be attributed to injury by these much decried instruments.

Hemorrhage having occurred in the trauma of childbirth, the propriety of trephining for the purpose of removing the clot suggests itself. This procedure was first advocated by Sarah McNutt, and later by Sachs, though it has never, to the writer's knowledge, been attempted, probably because the symptoms of paralysis do not immediately make their appearance. Although it is probable that the clot having once formed, has done extensive damage to the cortex, no procedure can be too radical that promises benefit to these unfortunate diplegic and imbecile children.

The treatment of the acquired form resolves itself into the treatment of the initial period and of the established disease.

A child in convulsions should be actively treated, cold should be applied to the head, bromides should be given by the mouth, while mercurials should be used to produce free purgation. In short the practitioner should be guided by general principles. The following, however, should be especially borne in mind, inasmuch as it is exceedingly probable that in some cases the convulsion is itself the direct cause of the later oncoming hemiplegia. Every effort should be made to prevent the recurrence of the attack, and to attain this end we should not hesitate to resort to the careful use of chloroform. Sachs insists that the physician should remain with the child for a number of hours until all danger of a repetition of the convulsion has disappeared. If the onset of a cerebral palsy be due to trauma prompt surgical interference is indicated. No harm can be done by an exploratory trephining, and if a meningeal clot be found great good may follow its removal. (See also Chapters XIII. and XXXIII.)

The treatment of the established condition resolves itself into the management of the paralysis and of the mental state. In diplegia proper the various remedies at our disposal have but little influence on the paralysis or the contractures. However, they should always be resorted to when possible. They consist of massage, passive movements, and faradism. While in severe cases but little impression is made by these remedies, in the less marked forms, especially those which approach in character paraplegia and in simple paraplegia itself, great good is often accomplished by persistence in their use. The parts should be gently kneaded and rubbed for a specified time daily, while the contractures should be overcome as much as possible by gentle movements. It is well in these cases to use some lubricant, as olive oil. Not only does the oil render the treatment more pleasant to the child, but it is not impossible that a small percentage may be absorbed. Faradism is a remedy which at times enables us to accomplish decided results. Some judgment must, however, be exercised in its use. The rule that I lay down to the nurse or to the mother who has been instructed in the employment of the slowly interrupted faradic current, is to apply the poles to those muscles which are overstretched and weakened by the contractures. That is, in the arm they should be applied to the extensor muscles, both of the arm and forearm, whilst in the leg they would be applied especially to the flexor group, *i. e.*, posteriorly to the thigh, anteriorly to the leg. The unskilful use of faradism, I have reason to believe, at times increases the contracture. Used according to the method I have described, the contractures are generally diminished. In hemiplegia there can be no doubt that the persistent use of massage and faradization actually accelerates the growth of the limb besides markedly diminishing the contractures.

The problems presented by defective intelligence are to be met by slow but persistent efforts at education. These it is not necessary to detail here. (See p. 335.) The principles laid down by the elder Seguin still hold good. They consist of the methodical education of the limbs, first by passive and later by voluntary motion. Finally, the case permitting, systematic gymnastics should be instituted. As the same time, an elementary kindergarten method of instruction should be pursued. Great care should be taken not to attempt too much in too short a time. This is especially true of attempts to teach the child to walk or to perform other simple movements. Some cases, indeed, are benefited by prolonged periods of rest in bed, with but little time devoted to walking or other exercise. It is frequently necessary to divide various tendons in order that the foot may assume a normal position. Orthopedic surgery sometimes yields here brilliant results. For the methods pursued the reader is referred to the chapter on Surgery.

CHAPTER XVIII.

DISEASES OF THE SPINAL CORD.

By JAMES HENDRIE LLOYD, M.D.

MALFORMATIONS OF THE SPINAL CORD.

Malformations of the spinal cord are either congenital or acquired. The former are comparatively few in number; the latter are not so uncommon, and they embrace a variety of pathological conditions. Of the former, the best example is that deformity of the cord seen in spina bifida; of the latter, the most conspicuous is syringomyelia. Yet even in these two examples the distinction between congenital and acquired deformities is somewhat arbitrary, because syringomyelia, while a disease usually of adult life, depends probably upon an inherent developmental weakness in the cord. It may be claimed similarly that Friedreich's ataxia and the various dystrophies which have as their base a gliomatous or degenerative change in the spinal cord, due to heredity, are examples of malformations of the cord; but obviously they do not fall within the accepted definitions of the term, and will not be described here. So, too, the defects of the spinal cord, sometimes seen in idiocy, might with justice be included among congenital defects; they are usually, however, of the nature of fine histological changes and system-lesions which do not come naturally under the head of deformities.

True malformations of the spinal cord of developmental origin are exceedingly rare. Spurious cases, due to bruises and cuts inflicted post-mortem, are not so rare, and have given rise of late years to a quite voluminous literature. These malformations may be included under the one term, *heterotopia*, which signifies a misplacement of either the gray or white matter. According to Van Gieson,[1] of the thirty-one cases of alleged malformations of the spinal cord now on record, only six are genuine examples. The most common form of true heterotopia is that in which a small portion of gray matter or of the gelatinous substance of Rolando is separated from the rest of the gray matter and lies imbedded in the white tracts. The accompanying illustration of a case of Pick's (Fig. 150) is an example of this. Such an anomaly is insignificant, and would give rise to no symptoms during life. The same is true of the very few instances of anomalous distribution of white tracks which have been observed, especially in the isthmus.

The examples of spurious or pseudo-heterotopia include a great variety of displacements of gray and white matter, or both, giving rise to the appearances of extraordinary malformations. The most striking of these is the so-called doubling of the cord, examples of which have been honestly reported and seriously discussed. Illustrations from a case are given here to serve as a warning (Figs. 151, 152, 153, 154). Such cases have been reported as

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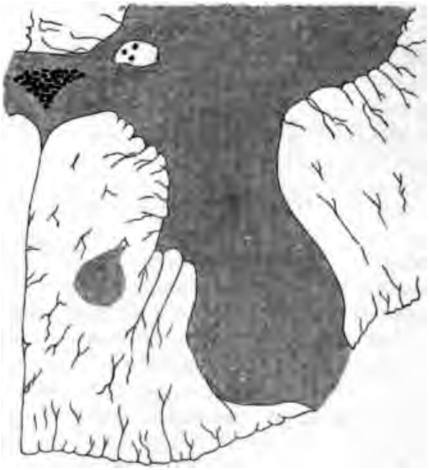
[1] "A Study of the Artefacts of the Nervous System," etc. N. Y. Med. Journ. 1892, vol. ii. pp. 337, 365, and 421.

"rudimentary cords" and "duplications." There can be no doubt that they are *artefacts, i. e.*, that they are produced post-mortem by accidents and by rough handling. Van Gieson has studied critically all recorded cases in his elaborate paper. Similar mistakes are no longer excusable.

FIG. 150.



Heterotopia of the spinal cord. The shaded circular area in the posterior column indicates the heterotopic fragment of gray matter. (PICK.)

A number of monstrosities of the cord, incompatible with life, have been observed. Among these are congenital absence of the cord, or *amyelia*, which is usually associated with absence of the brain; *atelomyelia*, or partial absence of the cord; *diastemato-myelia*, in which the cord is divided into two halves, each surrounded by its membrane—a condition probably allied to some forms of spina bifida; and, finally, *diaplomyelia*, a union of two cords, as seen in some kinds of double monster (to be distinguished from the pseudo duplications or artefacts already referred to).

FIG. 151.

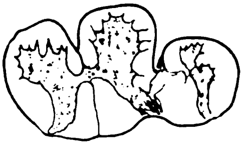


FIG. 152.

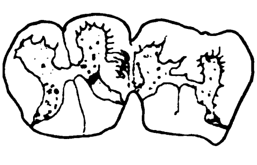


FIG. 153.

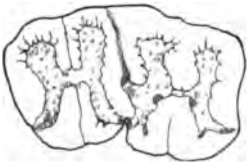
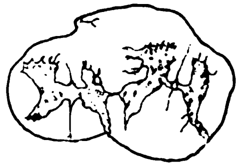


FIG. 154.



Artificial duplication of the spinal cord. (FÜRSTNER and ZACHER.)

There are some varieties of asymmetry of the spinal cord, associated especially with defective development or amputation of a limb, and these may be congenital. They need not be described in detail here.

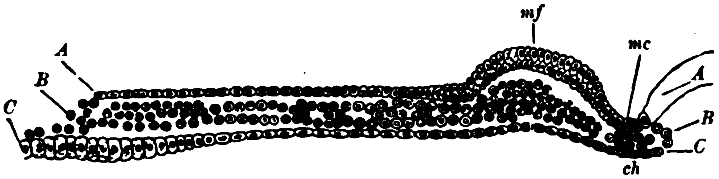
SPINA BIFIDA.

The one conspicuous example of malformation of the spinal cord is that which is an accompaniment of *spina bifida*. This will now be described.

MORPHOLOGICAL NOTE. In order to understand clearly the exact nature and the mode of formation of spina bifida, it is necessary to know the successive stages in the development of the spinal cord and the vertebral canal.

In the embryo-chick the elementary spinal cord, or medullary groove, has already become a conspicuous object before the end of the first day. In transverse section, at about the eighteenth hour, the blastoderm presents the following appearances (Fig. 155): Its three layers—epiblast, mesoblast, and hypoblast—have become differentiated. The medullary groove, which is to form eventually the spinal cord, is marked by a depression, at the base of which is a dense group of mesoblastic cells. The groove itself is formed exclusively of the epiblast, which lines its involuted surface; in other words, the groove is an involution of the epiblast. The group of mesoblastic cells at the base of the groove is the notochord, which in time gives place to the bodies of the vertebræ. On either side of the groove is a fold of epiblast, the medullary fold. These are the simple elements which eventually form the spinal cord.

FIG. 155.

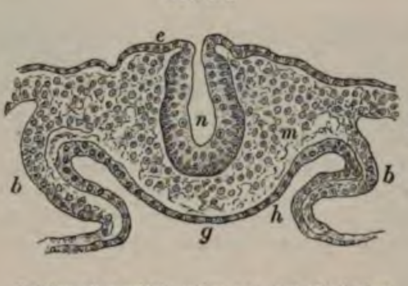


Transverse section of a blastoderm incubated for eighteen hours. The section passes through the medullary groove (*mc*) at some distance behind its front end. *A*, epiblast; *B*, mesoblast; *C*, hypoblast; *mc*, medullary groove; *mf*, medullary fold; *ch*, notochord. (BALFOUR.)

Before the end of the twenty-four hours these medullary folds increase in every dimension, and, growing in elevation, lean over toward each other, thus tending to form an arch, which, of course, encloses a long canal, the neural canal (Fig. 156.) These folds coalesce first near the head, and last at the caudal end.

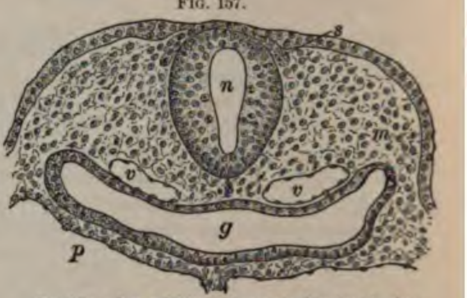
Before the end of the second day the medullary folds in the chick have coalesced to form this neural canal or tube (Fig. 157). The neural tube thus formed becomes gradually separated from the epiblastic layer, which coalesced externally to it, and which forms henceforth the epithelial layer of the skin of the back. In the meantime there has been formed on either side of the neural tube a dense body, composed of mesoblastic cells, and known as a mesoblastic somite (not shown in the illustrations). From this somite is formed eventually the vertebra and the trunk muscles. This mesoblastic tissue is gradually prolonged between the neural tube and the external epiblastic layer, and thus forms the arches of the vertebræ. From this mesoblastic tissue are formed also the deeper layers of the skin, the connective tissue and the bloodvessels, and the meninges of the cord. It is important, in order to understand the genesis of the various types of spina bifida, to keep in mind the fact that the neural tube is primarily a mere infolding of the epiblast, or external epithelial layer, and that its enclosure by bone and connective tissue is a secondary or later process.

FIG. 156.



Section of a rabbit-embryo at the ninth day, showing open neural tube. *e*, epiblast invaginated and thickened within the neural canal (*n*); *m*, the mesoblast; *b*, the body cavity; *g*, still open gut, lined with hypoblast. (PIERSOL.)

FIG. 157.



Section of a rabbit-embryo at the tenth day, showing closed neural tube. *n*, neural canal; *s*, segmental or posterior ganglia; *m*, mesoblast; *g*, gut-tube; *vv*. primitive aorta; *p*, thoracic cavity. (PIERSOL.)

The walls of this neural tube, formed entirely of epiblast, gradually undergo great increase in thickness by proliferation of their cell-elements, and from these walls is formed the entire cerebro-spinal system. The central canal of the cord persists through life as a minute tube, lined with epithelial cells.

In the early embryo the neural tube, or future spinal cord, is as long as the primitive organ mapped out by the somites, which will eventually form the vertebral column. In the course of development, however, this vertebral column grows faster than the neural tube, so that in the fully developed man, for instance, the spinal cord is not nearly so long as the vertebral canal, but extends to only the level of the second lumbar vertebra. In spina bifida of the lumbo-sacral region, however, it is common to find the cord extending down to that low level, which extension is readily understood by reference to the facts of embryology. It illustrates the fact, too, that spina bifida dates from almost the earliest period of development.

Morbid Anatomy. Spina bifida is a developmental defect, dating from a very early period of embryonic growth. It is caused by a failure of the vertebral arches, which grow from the mesoblastic somites, as just described, to coalesce dorsad to the spinal cord and between it and the superficial epithelial layer of the skin of the back. This failure is accompanied in various degrees, according to the case, by a failure of the other tissues, which also spring from this mesoblastic tissue, notably the corium of the skin and the meninges of the spinal canal, likewise to coalesce. Hence, there results a cleft or aperture in the vertebral column, through which protrudes, as a hernia, a sac which contains, according to its type, one or more of the contents of the spinal canal.

The types, or varieties, of spina bifida may be grouped in four classes,[1] according to the degree of deformity. While they are identical in origin, they differ widely in extent and character of tissue involved, and these differences are of vital importance clinically, and especially with reference to treatment.

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[1] Bland Sutton (Lancet, Feb. 25, 1888), in his admirable discussion on "Evolution in Pathology," makes six classes. The Committee on Spina Bifida of the London Clinical Society (Trans., vol. xviii.) recognizes only three. I think that four classes can properly include all varieties. There are sub-types which merge into each other, but which do not require special designations.

1. ***Meningocele.*** In this variety, which is the simplest and least disabling, one or more of the vertebral arches have failed to coalesce. The skin of the back, however, is usually perfect, and covers the sac. The meninges also are well formed, but protrude into the sac, or, more correctly, they constitute the sac, so that its cavity is continuous with the subarachnoid space. The cord, and consequently the nerves, do not extend into the sac (Fig. 158). The meningocele has a tendency to become pedunculated, and sometimes it is cured spontaneously. It is not dangerous to life, and usually does not cause paralysis nor deformity of the legs. It is a rare form.

FIG. 158.

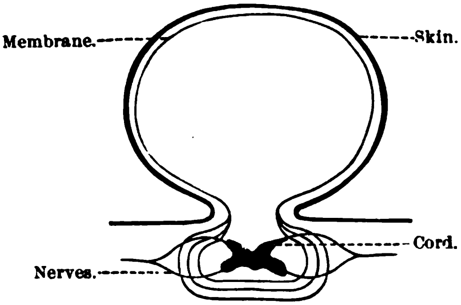


Diagram of a meningocele.

2. *Meningomyelocele.* This is the most common form of spina bifida, and is not unusually fatal. When the patient survives, it usually causes paralysis and deformities in the legs and feet. In this form not only have the vertebral arches failed to coalesce, but often the corium of the skin and the spinal meninges, which are likewise products of the mesoblast, have failed to develop dorsad to the cord. Hence, in the worst of these cases there is no meningeal cavity and no true skin behind the cord, which lies in immediate contact with the epithelial layer of the surface of the back (Fig. 159). It thus appears that in the primitive involution of the epiblast to form the cord, the latter never became separated from the epithelial or epiblastic layer. Therefore, as the

FIG. 159.

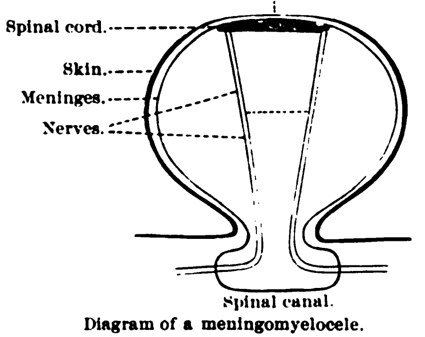
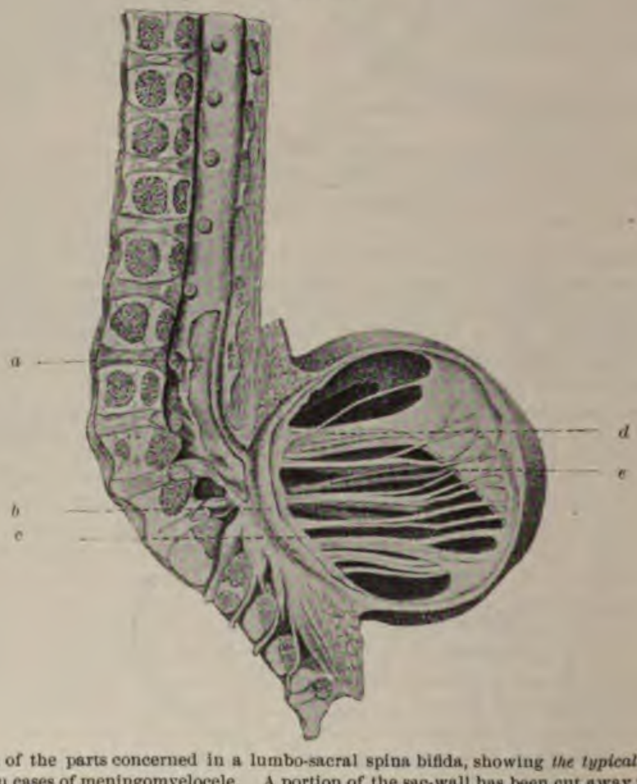


Diagram of a meningomyelocele.

sac protruded and distended more and more it drew the attached cord with it, so that the cord extends through the cleft in the vertebral wall, and is firmly imbedded in the wall of the sac. In this position its shape is usually deformed; it exists, indeed, as a layer of neural tissue over the posterior and inner surface of the sac. On transverse section in some cases the central canal may be seen in this layer (Fig. 159). The nerve-trunks of the segments of the cord involved are seen springing from this layer of nerve-tissue and coursing forward through the sac to gain the intervertebral foramina. Thus it is seen that the cord is pulled into the sac, and the nerves run, not *into* the sac, but *out* of it (Fig. 160).

FIG. 160.



Dissection of the parts concerned in a lumbo-sacral spina bifida, showing *the typical anatomical disposition* in cases of meningomyelocele. A portion of the sac-wall has been cut away to show the interior. *a*, surface of cord, covered with arachnoid, exposed by removal of portion of dura mater; *b*, dura mater entering into the formation of the sac-wall: *c*, arachnoid lining the sac; *d*, lower portion of the spinal cord, crossing the interior of the sac; some of the nerve-roots pass forward upon it, toward the intervertebral foramina; the other nerve-roots arise from the posterior wall of the sac in a vertical series and traverse the space horizontally; *e*, falciform process continuous with the pia mater, separating the anterior and posterior roots of the nerves of the left side; there is a corresponding process on the right side. (London Clinical Society.)

In meningomyelocele, because of the defective development of the corium in some cases, the extreme posterior surface of the sac is covered with an exceedingly thin membrane, devoid of hair and sebaceous glands and of any of the characteristics of true skin. This thin membrane represents the epiblast. In some cases, however, the skin is better developed. Occasionally there exists a small pit-like depression, or umbilicus, on the posterior surface of the sac. This marks the point of attachment of the spinal cord to the inner surface of the sac, and is a sign of some value in diagnosis.

In this form of spina bifida there usually is destruction or failure of development of some elements of that portion of the cord that is within the sac, and some degeneration of the nerve-trunks arising from it. Hence there result various degrees of paraplegia, paralysis of the bladder and rectum, club-foot, and sensory and trophic disorders in the regions below the level of the lesion.

3. *Hydromyelocele.* Occasionally, as seen in a few cases, the spinal cord not only adheres to the original epiblastic layer, as described in the last group, but as the resulting sac develops and expands, it, too, dilates in its central canal. Hence its tissue becomes a lining layer to the sac, the cavity of which is not continuous with the subarachnoid space, but with the central canal of the cord.[1] In this variety the nerves consequently do not run *through* the sac, but along its sides or imbedded in them. This form can readily be mistaken for a simple meningocele, in which no neural tissue is in the sac. This is because of the fact just stated, that the nerves do not run conspicuously through the sac, but are embedded in its wall between its external epithelial layer and its internal neural layer, representing the spinal cord. From the practical standpoint of treatment this distinction is of the utmost importance. Hydromyelocele is a rare form of spina bifida.

4. *Myelocele.* It occasionally, but very rarely, happens that not only the vertebral arches, but also the medullary folds, fail to coalesce. This is even a more serious defect than any of those varieties of spina bifida already described. In those, it will be recalled, the neural tube was always properly closed by the coalescence of the medullary folds; it was only in the cases of the vertebral arches and other mesoblastic tissues, as the true skin, that coalescence had failed. In simple myelocele, however, the coalescence of the medullary folds has failed, usually in only a limited portion of the cord. Of course the vertebral arches also have failed to unite, so there is seen a protuberance, usually small, on the back, usually in the lumbar region, in the centre of which is a small opening. This opening leads directly into the cerebro-spinal canal, and the small protuberance is not a sac but a mass, usually red and pulpy, of neural tissue. From this opening cerebro-spinal

FIG. 161.

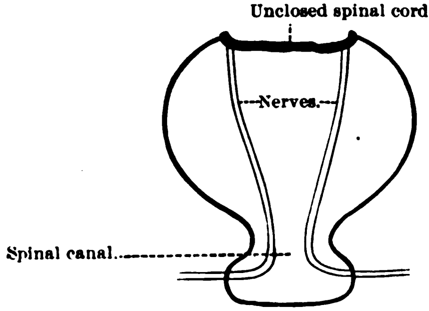


Diagram of a myelocele.

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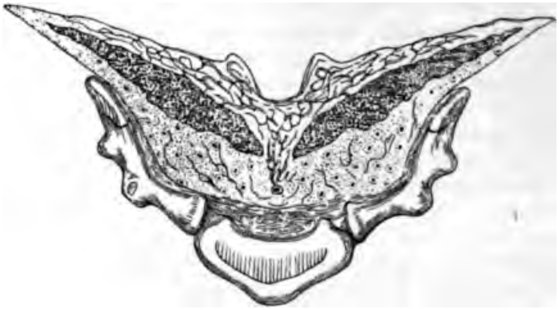
[1] This variety is called, by most writers, rather unfortunately, *syringo*-myelocele. This term seems to ally it with syringomyelia, which, however, is now known to be *not* a dilatation of the central canal, but a new gliomatous formation. It were better, therefore, if this variety of spina bifida were called *hydro*-myelocele, which term is used here.

fluid constantly oozes. This protuberance has somewhat the appearance of a nævus. Myelocele is incompatible with prolonged life; even in cases in which the defect is limited in extent, death usually occurs in a few days. According to Sutton[1] myelocele of great extent has been seen by Lebedeff in the chick.[2] Sutton believes, probably correctly, that this form of spina bifida is not so rare as represented, but is cast aside as an "atypical" form.

FIG. 162.



Transverse section of neural tissue, constituting the undeveloped spinal cord, in a myelocele. (SUTTON.)

A comparatively harmless form of spina bifida, in which a slight cleft exists without protrusion of a sac, is called *spina bifida occulta.*

In a very few cases of spina bifida the sac is divided by membranous partitions, so that it forms a multilocular cyst.

Congenital tumors of the sacro-coccygeal region occasionally occur, the nature of which may be obscure. They have given rise to much confusion in the past, and a voluminous literature, especially in France and Germany, has sprung up about them.[3] It is evident that there are several varieties of these congenital sacral and coccygeal tumors, one of which is a spina bifida depending upon defective development of the laminæ of one or more bones of the sacrum. In some of these cases the coccyx is wanting. A peculiarity of some cases of this sacral type of spina bifida is the protrusion of the sac anteriorly into the pelvis. The causes for this probably lie in the fact that for some reason this is the direction of least resistance. The tumor sometimes forms, as pointed out by Giraldes, in the sacro-perineal region. It usually pushes the rectum and anus before it. Thus Giraldes, quoted by Molk, refers to a case in an adult female, in whom a perineal cystic tumor was punctured by a surgeon, and who died very soon, as a consequence, with the tetanoid symptoms of meningitis. An autopsy demonstrated that the cyst was attached to the sacrum and communicated with the vertebral canal. Emmet[4] and Thomas[5] each have punctured such a pelvic cyst by mistake, and both have lost their patients in consequence.

Spina bifida is occasionally associated with other developmental defects, as encephalocele, hydrocephalus, harelip, cleft palate, ectopia of the viscera, and imperforate anus. These defects are of the kind that are caused by failure of coalescence in various regions of the embryo-body. Hydrocephalus is reported as a not infrequent complication (Fig. 163). Encephalocele is seen especially at the posterior base of the skull, involving the cerebellum.

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[1] Op. cit.

[2] Embryologists know that deformities in the chick are not uncommon, especially if the eggs are handled much in the course of artificial hatching. Such specimens, however, are usually cast aside.

[3] See especially Des Tumeurs Congenitales de l'extremite inferieure du tronc, by Molk, Strasbourg, 1868; and Resherches sur les Tumeurs Congenitales de la Region Sacro-coccygienne, by Lachaud, Paris, 1883.

[4] Am. Journ. Obstetrics, 1871.

[5] Gaillard's Med. Journ., March, 1885, p. 237. See also, for report of a similar case in which death followed labor, Gaz. Méd. de Paris, 1838, p. 10.

Causes. The causes of spina bifida are altogether obscure. As stated already, the deformity has been seen in the chick, in which it extends sometimes throughout almost the whole extent of the spine. Monstrosities of various kinds are produced rather easily in the embryo-chick in the course of artificial hatching. This suggests the probability that trauma or shock may interfere with proper development, and may be capable of producing spina bifida. This, of course, is a mere hypothesis. Heredity has seemed to have an influence in a very few cases. In one instance, in the list prepared by the London Clinical Society's Committee, three successive cases of spina bifida occurred in one family; while Demme[1] refers to two instances in which there had been three cases, and three in which there had been two cases of spina bifida in one family. Camper, quoted by Ashhurst,[2] noted spina bifida in twins. In a few instances congenital club-foot and harelip have been reported in one or other parent of children with spina bifida.

Clinical History. Spina bifida occurs rather more frequently in females than in males. According to the tables compiled by the London Clinical Society's Committee, from returns of the Registrar General of England, of 1768 cases, 779 were in males and 989 in females.

According to Chaussier, one case of spina bifida occurred in the Paris Maternité in about one thousand births.

The position of the tumor is most commonly in the lumbar, lumbo-sacral, or sacral region. It is comparatively infrequent in the cervical and upper dorsal region. The reason for this preponderance of the lesion in the lower region of the spine lies in the fact that the medullary folds in the embryo close over last at the caudal end. Occasionally two tumors are seen, but this is extremely rare.

The malformation is usually discovered at the birth of the child. It varies in size at birth in different cases, and, of course, is more likely to be overlooked when it is very small. In the occult form, which may persist through a comparatively long life, and which is not marked by a tumor, the deformity may be readily overlooked.[3] Schon[4] reports a case of spina bifida occulta in a girl aged thirteen years. The defect was in the fifth lumbar and upper sacral vertebræ. The first symptom noted was scoliosis, which was first seen in the patient's seventh year. The lumbar region was covered with a dense growth of hair (hypertrichosis), and the skin was pigmented from the twelfth dorsal vertebra to the coccyx. The size of the sac at birth is not always a criterion of its dangerous character. It may be no larger than the end of a large thumb, and yet it may increase in size rapidly. Moreover, it cannot be judged by its size to which type it belongs, and upon its type depends, to a large extent, the prognosis. Thus, if it is a simple small meningocele, it may enlarge, even quite rapidly, and yet the possibility of a cure is greater in this than in any other variety. If it is a meningomyelocele or an hydromyelocele, it may be accompanied with club-foot. If it is a pure myelocele, it will present only a small fleshy excrescence, in the centre of which careful inspection would probably reveal a leaking fistula communicating with the cerebro-spinal canal. In every form the tumor almost invariably occupies the region of the spinous processes; exceptions are very rare, as in those cases that are reported as protruding anteriorly. In some cases only one or two vertebræ are involved, in others a larger number. The lesion, as said already, is far more common in the lower dorsal, lumbar, and sacral regions than in the upper part of the spine.

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[1] Quoted by Lond. Clin. Soc.'s Committee.

[2] Am. Syst. of Med., vol. v. p. 757.

[3] Lancisi saw a case in which the tumor did not declare itself until the fifteenth year, and Akin refers to a case in which the lesion did not show until the twentieth. Dict. des Sciences Med., 1818, art. "Hydrorachis." tome 22.

[4] Berl. klin. Wochenschrift, 1894.

The child at birth may appear otherwise in normal health. Many observers report, however, that the children with spinæ bifidæ are not well nourished, and that they soon begin to pine and fail. The growth of the tumor varies. The great majority of patients born with spinæ bifidæ die within the first year, and the majority of these within the first three months, and many of these deaths are caused by a rapid distention and rupture of the sac. In some cases, however, the growth is very slow at first, or even not perceptible, so that the child, in a small proportion of cases, survives for some years, or may even attain adult life.

The appearance and constituents of the sac vary according to its type, to understand which it is essential to refer constantly to the facts as illustrated by embryology. In some cases the sac is covered with good healthy skin; in these the probability is that the type is not the worst. Even though not a simple meningocele, the covering of healthy skin is a support and protection. In other cases the healthy, fully developed skin ceases at the base of or near the summit of the sac, the remainder of the sac being covered with a thin, delicate membrane. This membrane is not the dura mater, but the epithelial layer of the skin formed by the epiblast. This form should suggest to the observer the probability that the tissue of the spinal cord is included in the sac either as a meningomyelocele or an hydromyelocele. An umbilicus, marking the attachment of the cord, may possibly be seen. In such a case the dura mater does not intervene between the cord-tissue and the epithelial layer, as is commonly said; but the cord, usually spread out as a layer of neural tissue in direct contact with the sac, which is formed only of this delicate epithelial membrane, is an important constituent of the sac, and sends nerves forward, *through, and out of* the sac to their respective foramina.[1] The opening in the bones may sometimes be felt with the fingers, but it is doubtful if the nerve-cords passing out of the sac into the vertebral canal could be distinguished by palpation. In some of these cases the walls of the sac at, or soon after, birth are much distended, and they may even be inflamed or ulcerated. Occasionally the sac soon begins to leak, the fluid either escaping by a small opening or oozing out through many pores. On the other hand, the surface of the sac may be rugous or coreaceous. The tumor itself is usually tense and elastic, and its contents fluctuate. Pressure on it causes often some significant symptoms; thus its volume may be reduced somewhat by pressure, but brain-symptoms result. These are uneasiness, then stupor or even coma, and convulsions. Holmes[2] says that convulsions are common during all stages of the disease, but especially after the sac has burst, when they are often the immediate cause of death. When associated with encephalocele, according to Treves,[3] the fontanelles bulge when pressure is made on the sac. When the child cries the sac distends.

The fluid in the sac of a spina bifida has been proved frequently to be identical with the cerebro-spinal fluid. It is watery, with a few salts, and with only a slight trace of albumin. Of course, in case inflammation and suppuration have occurred, the fluid is altered accordingly.

In some cases the tumor is pedunculated and the opening into the vertebral canal is small; in such cases the probability is that the sac does not contain nerve-tissue, *i. e.*, that it is a meningocele. If so, it would not be attended with paralysis.

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[1] This is constantly misunderstood by surgeons, who speak of the nerves running *into* the sac, and who overlook this thin layer of tissue representing the spinal cord.

[2] Surg. Treat. of the Dis. of Infancy and Childhood.

[3] Int. Encyc. of Surgery, vol. iv.

In grave forms of spina bifida involving the spinal cord and nerves symptoms of paralysis in some form are usually observed. Thus there may be an almost complete paraplegia with anæsthesia, and involvement of the bladder and rectum. It is rather more common, however, to observe modifications of this extreme type. Amyotrophic paralysis of some muscle-groups is not uncommon, due to involvement of the nerves and anterior horns. This form presents the usual symptoms of wasted muscles, coldness, and mottling, with alterations in the electro-tonus. Club-foot, especially talipes equino-varus, is a common result. Paralysis of the bladder and rectum is a common complication. Anæsthesia in various areas is often present. In one case under the author's care trophic lesions were seen.

The following case occurred in a girl who was admitted into the Home for Crippled Children under the writer's care. She presented some typical symptoms:

C. E., female, aged nine years, had a spina bifida of the lumbar region, involving the first three vertebral arches. She was paralyzed in her limbs and had badly impaired feet, walking on the backs of her feet, *i. e.*, presenting an extreme varus. There was some wasting of the muscles below the knees. There was slight foot-clonus on the right, none on the left. The patellar reflexes were abolished. The patient had no control over the bladder, and diminished control over the rectum. The tactile sense was very blunt below the knees, if not abolished. Above the knees a line of demarcation of the anæsthesia was quite well marked. Slight sensation was preserved on the inner side of the leg. The thighs were sensitive. To heat and cold sensation below the knees was abolished, above the knees it was normal. All the muscles of the legs and thighs responded normally to the faradic current, except the peroneal group. In this there was no response to the strongest currents. To galvanism in strong currents there was complete abolition of response in the peroneal group. Slight modal change was noted in the flexors of the big toe. There was no serial change. The mother reported that the child was liable to the formation on the legs of "bladders," *i. e.*, bullæ, leading to trophic sores. These sores left a scar after healing. In the bullous stage they resembled pemphigus. On admission one of these trophic suppurating sores was seen on the left great toe, looking almost like a perforating ulcer. The fronts of the tibiæ were marked by the scars of these sores. It was evident in this case that the trophic and reflex centres in the lumbar enlargement were impaired; probably also some of the nerve-trunks. The mother said that at birth the tumor was the size of an end of a thumb. It had not increased much if any at first, and the child had learned to walk and its legs seemed all right until it was three years old. The tumor had been growing rapidly during the last two years. The sac was very firm, covered with apparently healthy skin, and was not as yet overstretched.

Another interesting case occurred in a boy, a picture of whom is presented here (Fig. 163). He was seen by me in the Methodist Hospital. H. C., aged about seven years, had a spina bifida in the lumbo-sacral region, and had also hydrocephalus, with convergent strabismus. He had paralysis of the bladder, rectum, and legs, and presented double club-foot in minor degree. The case was apparently one of meningomyelocele. I advised against operation in his case, as I had no doubt that the sac contained tissue of undeveloped spinal cord.

In pelvic spina bifida, instances of which have been reported by Emmet[1] and by Thomas,[2] the cyst is firmly attached to the sacrum. It pushes the rectum forward, and in some cases causes obstruction of the rectum, and consequent distention of the bowel. In some of these reported cases the patients had reached adult life. Emmet's patient was apparently about forty-five years of age, and Thomas' was nineteen. In the latter case the lady had noticed that she was not in a normal condition, and sought relief because she intended to marry. The inconvenience, however, had been slight, and there were no paralytic symptoms. In a few hours after the cyst had been tapped grave symptoms of involvement of the brain and cord appeared. The patient had intense headache, maniacal delirium and rigidity of muscles, and she failed rapidly and died.

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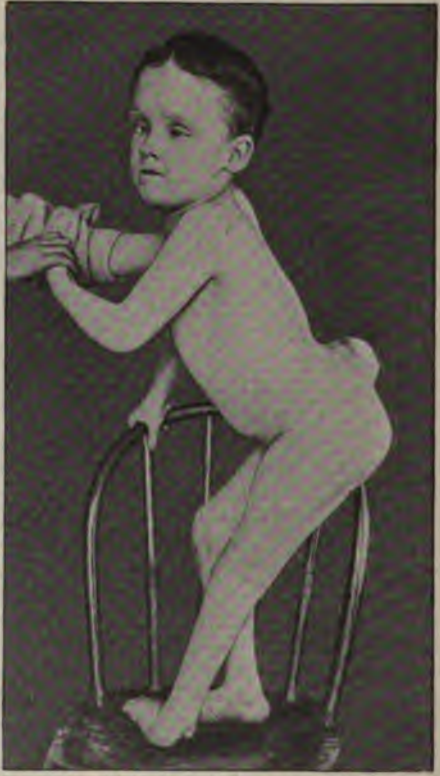
[1] Op. cit.

[2] Op. cit.

FIG. 163.



Spina bifida with hydrocephalus. (Methodist Hospital.)

Diagnosis. The diagnosis of spina bifida can be established sometimes in early cases by the detection of the orifice into the spinal canal. This can be felt, with its bony outline, at the base of the tumor. The distention of the sac when the child cries, the umbilicoid pit in the walls of the sac, the absence of true skin over the summit of the tumor, are all characteristic signs upon which reliance can be placed. It is not probable that confusion could occur to any ordinarily intelligent observer between either a lipoma of the back or a spinal caries and a spina bifida.

Congenital tumors of the sacro-coccygeal region have given rise, as already said, to considerable confusion in diagnosis. There are several varieties of these congenital tumors, which, as Sutton has pointed out,[1] may be divided into four groups: dermoid cysts, tumors arising from the post-anal gut, forms of spina bifida, and teratomata, with remains of suppressed embryos. The differential diagnosis between these various forms in very young children is no doubt difficult, and reliance must be placed in cases of supposed spina bifida upon the symptoms just described. Especial difficulty has been encountered in cases of pelvic spina bifida, which has been graphically described by Thomas.[2] In these cases the tumor is firmly adherent to the sacrum, and is behind the rectum, which is pushed forward—a most important distinction. In doubtful cases a small quantity of fluid should be withdrawn and tested. If it prove identical with cerebro-spinal fluid the diagnosis of spina bifida may be considered established.

Prognosis. Spina bifida is an exceedingly grave affection, and the prognosis in the case of the newborn child is always doubtful. As said already, more than one-half of the cases of spina bifida die in their first year, and the majority of these in the first three months. In all forms, except simple meningocele, the probability is that if the child survives it will have club-foot and either partial or complete paraplegia, with incontinence of urine. The pelvic or anterior form appears to be attended with less immediate risk to life and limb. This is shown by the fact that all four cases of this very rare type, referred to in this paper, were in adult females; but they all eventually proved fatal, three from surgical interference, and one from the effects of childbirth. On the other hand, according to Treves, patients with spinæ bifidæ have lived to thirty-seven, forty-three, and fifty years of age.

Treatment. The treatment for spina bifida is almost entirely surgical. Some of this surgical treatment seems to be conducted with too much disregard for the pathology of the affection, and hence meets with mortifying results. Thus Holmes[3] relates how he *excised* a spina bifida from a girl aged eight years, who had paralysis of the sphincters. The child succumbed promptly to meningitis, opisthotonus, etc., and died. The surgeon looked for nerve-trunks to warn him away from dangerous ground, but, apparently, he ignored the fact that in the type of spina bifida to which his case evidently belonged the sac itself is composed partially of undeveloped spinal cord, and, therefore, that he was excising part of the cord. It is obviously impossible for any surgical procedure to restore perfect health to body and limb in cases of meningomyelocele and hydromyelocele. The protruding sac being composed partially of neural tissue, representing the undeveloped spinal cord, to either excise it or to extirpate it with irritating injections will not cure the patient. At best, if occlusion is obtained, the child can continue as hopelessly a more or less complete paraplegic. In simple meningocele, in which type no nerve-tissue is included in the sac, the prognosis is more hopeful and the prospect for cure by surgical means is much brighter. In the pelvic form, experience so far seems to prove that surgical interference is invariably fatal. For details and results of surgical treatment reference is made to Chapter XXXIII. and to the report of the Committee on Spina Bifida to the London Clinical Society.

PACHYMENINGITIS AND LEPTOMENINGITIS.

Inflammation of the membranes of the spinal cord is usually secondary to some other pathological state, and in the great majority of cases is probably the result of infection. An instance of the former is the combined pachymeningitis and leptomeningitis that occurs in caries of the spine: of the latter the inflammation and thickening of the membranes that are due occasionally to syphilis. The membranes are also involved secondarily in gross lesions, such as tumor; and acutely in some forms of infection, such as cerebro-spinal fever. They are also involved usually in more diffused lesions, such as the various forms of myelitis; in fact, the worse symptoms of meningitis are those that are due to involvement of, or pressure upon, the tissue of the spinal cord. I shall describe here only the more distinct and characteristic types of meningitis of the cord.

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[1] See discussion of Bowlby's report of "Three cases of Coccygeal Tumor," Brit. Med. Journ., 1890, i. p. 663.

[2] Op. cit.

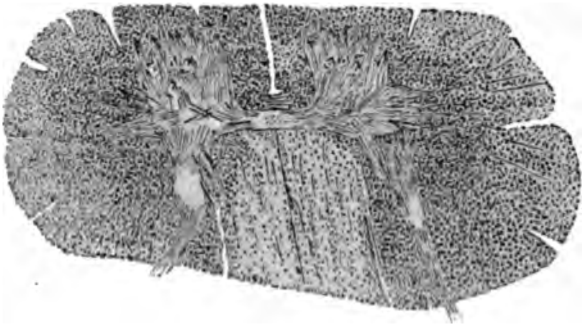
[3] Op. cit.

Causes. Caries of the vertebral bodies is a common cause of meningitis. The symptoms usually ascribed to bone-pressure in Pott's disease are in fact due, as a rule, to secondary involvement of the membranes, and even of the cord itself, by the infectious process. This will be referred to later, and illustrated with original cases. Syphilis doubtless causes inflammation of the membranes of the cord, and in this way is possibly the cause of some of the so-called system lesions. A type of spinal syphilis has been described by Erb, which is probably due to a syphilitic meningitis, the inflammation involving eventually some areas of the cord itself. Trauma is regarded almost universally as a cause of pachymeningitis, and cases occur not infrequently which confirm this view. The type described by Charcot as *pachymeningitis cervicalis hypertrophica* is caused sometimes by trauma. Exposure to cold and wet, especially in alcoholic subjects and in persons poorly nourished, is apparently a cause of meningitis, just as it may be of neuritis. Pyæmia, or infection with the streptococcus pyogenes, secondary sometimes to suppurative lung and pleural disease or to puerperal infection, is a most important cause, especially of leptomeningitis. The infections of acute diseases, such as typhoid fever and smallpox, have the power possibly of causing inflammation of the spinal membranes. Finally, cases occur in which the etiology is obscure, probably because accurate histories of the patients are not attainable.

Pathology. In cases of infection by contact, as in spinal caries, the mode of the disease-progress is easily understood. In some rare cases, however, a tubercular infection of the spinal meninges may occur quite independently of a bone lesion, just as it occurs in cases of tubercular meningitis at the base of the brain. In these cases, of course, the bacilli of tubercle are conveyed directly to the membranes by the blood, the seat of primary infection being elsewhere, and perhaps not always determinable. In pyæmic cases the lymphatics may possibly convey the germs; but it is most probable that in the cases of this and all other infective germs the blood is the means of conveyance. The exact mode of operation of cold and of trauma, which indubitably act as causes of meningitis, is not so readily understood. If we confine the idea of an inflammatory process to the action merely of a microbic organism, then there is some difficulty in including the destructive actions of trauma and cold in the category of inflammation. The facts, however, speak loudly against such a decision. The theory has been advanced that cold may generate, by a sort of vito-chemistry, a rheumatoid humor, which is the obnoxious agent; but this is a mere theory. We have, however, nothing better to offer, unless we suppose that a diminished resistance is brought about in the tissues by the depressing action of cold, and that this permits an unopposed or feebly-opposed activity of the destructive organisms which are probably present in the system at all times. In the case of trauma a somewhat similar explanation may hold; and in addition it may be recalled that the efforts at repair may constitute in themselves an almost inflammatory process; that one effect of shock or injury may be a cell-proliferation, in the line or direction of a healing process, which may overstep the mark and become in its turn a truly destructive process. In granulation tissue we witness something of this kind. In this new and not highly vitalized tissue various destructive and even pyogenic organisms may find a lodgment, and thus a truly inflammatory and even suppurative process may be established.

Morbid Anatomy. In pachymeningitis due to spinal caries the bodies of the diseased vertebræ, usually soft and carious, form a mass which is in direct contact with the dura.[1] At this point the thickened membranes and fibrinous deposits form a felted mass, which impinges more or less upon the cord. It usually extends to either side of the cord, but only exceptionally reaches entirely around it. The dura mater is often greatly thickened, and this tubercular mass, composed of broken-down tissue, the result of tubercular inflammation, is attached to it at the anterior aspect of the cord. This mass represents apparently old and degenerated tubercular matter, giant cells, and inflammatory lymph. The inflammatory process in these cases takes place largely upon the *outside* of the dura (the pachymeningitis *externa* of some authors), but not entirely so; since in advanced cases there is usually also a leptomeningitis and even a myelitis accompanying it. In some cases, in fact, as in one of the writer's cases, the dura may be perforated, the ragged edges of the membrane adhering to the carious mass in front, and thus communicating with and draining into a psoas abscess. The cord may show considerable evidence of myelitis, as softening, and some deformity, which may be due to pressure of the tubercular mass. Thus, in a recent case examined post-mortem by the writer, a large tubercular mass made pressure at one side of the cord. It had caused some unilateral symptoms during life. This pressure, however, does not usually cause a total transverse lesion. This is made evident in the accompanying figures (see Figs. 164, 165, and 166), in which, as can be seen readily, neither the white nor the gray matter is pressed much out of shape or materially destroyed. There is seen, however, a rim of degenerated tissue around the border of the cord, evidently due to contact with the inflamed meninges. This may be visible only under the microscope. Secondary upward and downward degenerations are also seen.

FIG. 164.



Section of the spinal cord from a case of Pott's disease (At the seat of caries.)

The secondary degenerations in the cord in these cases are characteristic, and are seen in the figures. At the level of the lesion the cord is misshapen, degenerated about its periphery, and presents the commencement of ascending posterior and descending lateral degeneration. At a point well above the level of the lesion the deep degeneration of the postero-internal columns (columns of Goll) is most conspicuous. (Fig. 165.) The direct cerebellar tracts to the exterior of the lateral pyramidal tracts are notably degenerate. These also, being ascending fibres, follow the Wallerian law. Finally, the region of the anterior ground bundles, including Gowers's tract, which degenerates upward, shows quite extensive degeneration. Below the level of the lesion the appearance of the cord is quite different. (Fig. 166.) There is marked degeneration of the lateral pyramidal tracts, which as they pass downward approach nearer and nearer to the periphery. The thickened dura with its layer of fibrinous deposits extends in some cases far above and below the level of the caries, and in such cases may be attended with changes on the anterior aspect of the cord coextensive with its own limits. Pus may infiltrate through the loose cellular tissue of the vertebral canal.

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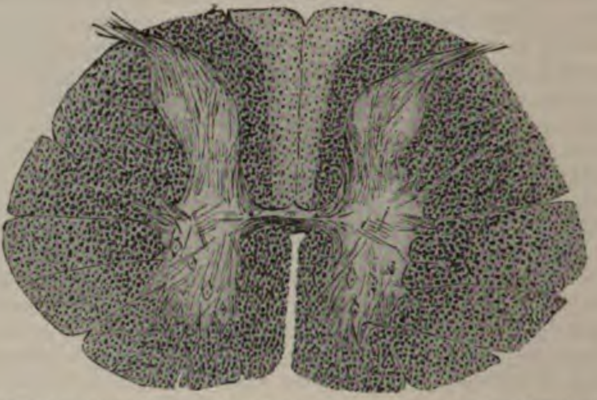
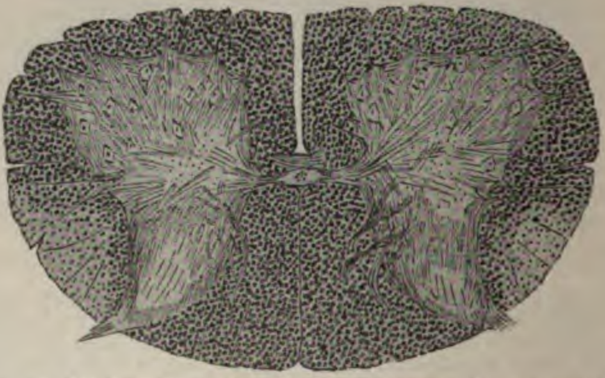
[1] This description is based largely upon cases described elsewhere by the writer. See "Pachymeningitis and Myelitis from Pott's Disease," etc., with illustrations, University Medical Magazine, December, 1893.

FIG. 165.



Section of the spinal cord from a case of Pott's disease. (Dorsal region.)

FIG. 166.



Section of the spinal cord from a case of Pott's disease. (Lumbar region.)

The form of meningitis described by some writers as pachymeningitis *interna* is of different origin from that of the form just described. It is supposed to begin in minute hemorrhages, the blood from which becomes organized, and thus leads to a hypertrophy of tissue. The process is somewhat analogous to that which occurs in hemorrhagic pachymeningitis within the cranium. It was first described accurately by Charcot; one of its favorite seats is the cervical enlargement, and one of its most common causes is trauma.[1] Compression of the cord occurs sooner or later, with impairment of the anterior horns, and with degeneration of the conducting paths, as has just been described.

Leptomeningitis of acute pyæmic origin is marked by a thickened and opaque pia-arachnoid, under which pus in some quantity is usually seen. The cord is hyperæmic. The pus is usually distributed widely in vertical extent. The process, however, is not apt to be confined strictly to the pia, but to invade also the inner surface of the dura. The tissue of the cord is evidently involved in the acute infective process, but, as pressure is not usually a marked condition, and as the course of the case is often rapid, system-lesions are not so well marked as in the more chronic forms described above.

A more chronic form of combined lepto- and pachymeningitis is described sometimes as due especially to syphilis. It consists in a proliferation of tissue without pus formation, and it encroaches gradually upon the cord. Erb[2] has described a form of syphilitic infection, which appears to invade by preference the lower dorsal and lumbar region, and to be limited almost exclusively to the lateral aspects of the cord. It becomes in time a partially transverse lesion symmetrically situated, affecting the posterior halves of the lateral columns, extending thence to the posterior horns and posterior columns. (See also Chapter XXIV.). As is seen it is not a pure meningitis, but rather a meningomyelitis. A somewhat similar slow proliferative meningitis may possibly be caused by alcohol, by exposure, and by trauma, or by all combined.

Symptoms. The symptoms of the various types of meningitis differ according to the cause, the stage of activity, and the seat of the lesion. The chronic form, as for instance the pachymeningitis of spinal caries, may approach insidiously, and its approach may be masked by the primary disease. The traumatic form, too, is often slow in its onset, and sometimes appears unmistakably only after the primary effects of the injury have disappeared. The acute form, however, may appear brusquely, even explosively, and may destroy life in a comparatively short time. This grave form is seen occasionally after puerperal infection.

In the pachymeningitis of spinal caries the earliest symptom may be pain, due to irritation of one or more of the sensory nerve-roots. This pain, reflected along one or more of the nerve-trunks of the thorax or abdomen, may appear even before the hump is noticed, and its cause may thus be overlooked. In this form the motor symptoms, however, are, as a rule, the most conspicuous. There occurs a gradually increasing paresis in the legs, with contractures in the muscles, increased knee-jerks, and ankle-clonus. The loss of power may advance to complete paraplegia. In advanced cases, and even sometimes in early stages, the bladder may become involved; control over the rectum also is occasionally lost. Sensory symptoms, excepting the initial neuralgic pains, are, as a rule, not nearly so conspicuous as the motor; in fact, a child may be almost hopelessly paralyzed, and yet preserve some degree of tactile sensibility. This is expl the fact that the anterior and lateral regions of the cord are most the inflammatory process. In a fair proportion of cases, h ally in those far advanced, anæsthesia to touch and to pain is present. This analgesia was strikingly shown in a little boy with dorsal caries under the writer's care in the Home for Crippled Children within the past year. It became necessary to circumcise the child because of a long and constantly inflamed and ulcerated prepuce. The operation was done under ether, by Dr. Hamilton, but during the subsequent changes of dressings, removal of stitches, etc., the patient was quite indifferent, and evidently felt no pain. Bed-sores occur readily in these advanced paraplegic cases. The seat of spinal caries is usually in the dorsal or dorso-lumbar region. In those comparatively rare cases in which it is in position for its resulting meningitis to involve either the cervical or lumbar enlargement of the cord there result muscular atrophy and flaccid paralysis of the corresponding limbs.

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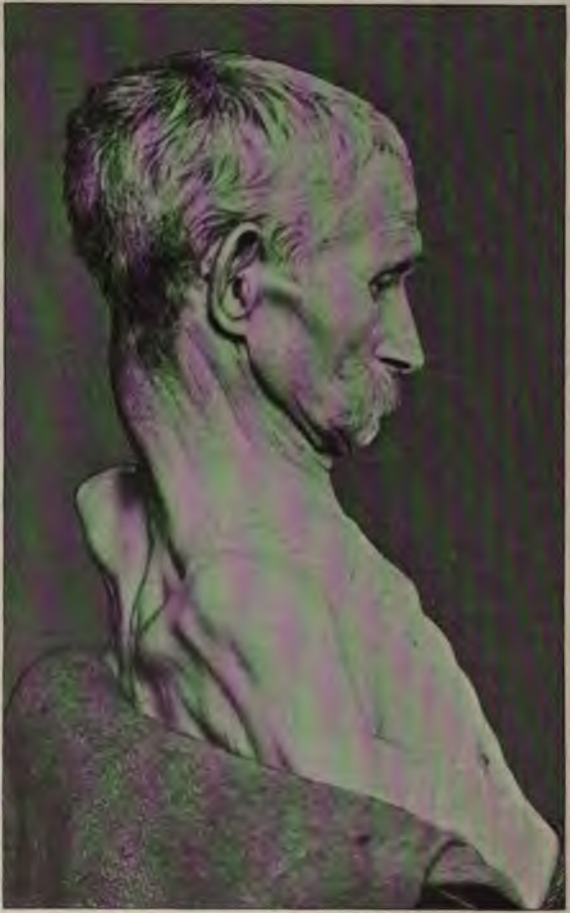
[1] For a report of cases by the writer, see a paper on " T l Region of the Spinal Cord Simulating Syringomyelia," Journal e, 1894.

[2] " Ueber Syphilitische Spinal Paralyse." Neurolog. (

FIG. 167.



Trauma of the cervical region of the spinal cord.

In the type first described by Charcot as *pachymeningitis hypertrophica cervicalis* the symptom-complex is quite characteristic and resembles markedly syringomyelia. In this form, as the name implies, the cervical enlargement is the seat of disease. Among the first symptoms is pain, of a neuralgic kind, in the occiput, neck, shoulders, and arms. Wasting of the muscles of the hand (the thenar, hypothenar, and inter-osseous groups), the forearm, and shoulder next attract attention. This wasting advances to an extreme emaciation of the muscles (Fig. 167), with corresponding loss of power. It is usually of the Aran-Duchenne type of progressive muscular atrophy, *i. e.*, flaccid and with diminished myotonus, but this is not an invariable rule, as markedly increased tonus is seen in some cases. Fibrillation of the wasting muscles occurs. Deformity of the cervical spine and scoliosis appear. As the cervical symptoms increase a spastic paraplegia, with increased knee-jerks, and usually without involvement of the bladder and rectum, becomes established. Sensory symptoms of great interest may appear in these cases. There may be thermo-anæsthesia and analgesia without abolition of tactile sensibility (the so-called *dissociation* symptom of syringomyelia) in some areas. There may be however, some areas or anæsthesia. The sensory changes may preserve an hemiplegic type,[1] Occasionally changes in the pupils occur, due to involvement of the sympathetic nerve.

Leptomeningitis of the spinal cord usually occurs as a part of the more generalized cerebro-spinal meningitis. This latter may be due to the specific contagion, usually epidemic, to which the term "cerebro-spinal fever" is popularly applied; or an equally diffused cerebro-spinal leptomeningitis may be due occasionally to tubercle (quite independent of spinal caries), or finally it may be caused by pyæmia. I once saw a patient with meningitis of the lower third of the vertebral canal caused by direct infection from an extensive bed-sore beneath the sacrum. Similar cases due to puerperal and to pulmonary infection have been noted. Primary spinal leptomeningitis, without cranial leptomeningitis, is an extremely rare affection. Macewen,[2] speaking of purulent leptomeningitis of the cerebrum, due to suppurative ear diseases, says that the pus may extend down to the cauda equina. When the affection extends thus to the spinal cord spinal symptoms are seen; these are especially the girdle-sense, severe shooting pains, and rapid and complete prostration. Other symptoms of spinal irritation occur, such as rigidity and opisthotonus, and finally the rapid onset of paralysis indicates that the ganglionic centres in the cord and the conducting nerves have been invaded by the poison of the infection. Hemorrhagic pachymeningitis (*pachymeningitis interna*) of the spinal canal occurs usually in association with the same affection within the cranium, and as an accompaniment of degenerative processes, such as occur in general paresis and in chronic alcoholism. It is consequently difficult to recognize it during life, as it is usually masked by other conditions.

The various forms of syphilitic infection of the cord doubtless involve the membranes, in some cases even primarily; but they are best described under other headings, as they are in this treatise. (See Chapter XXIV.)

To recapitulate, it may be said that meningitis within the vertebral canal has usually three stages. (1) Irritation, marked by neuralgic pains, girdle-sense, and slight beginning loss of power. (2) Paralysis, both of sensation and motion, gradually increasing. (3) System degenerations, upward and downward, trophic disorders, paralysis of the bladder and rectum. This order of symptoms, however, is subject to variation.

Diagnosis. In the early stages of spinal caries, before the appearance of the hump, it is most important to recognize the true character of the thoracic and abdominal pains that are caused by irritation of the nerve-roots. Stiffness of the spine and a focus of beginning pain and deformity can usually be detected early if the observer is alert to the significance of the first symptoms. In later stages of spinal caries the diagnosis of pachymeningitis can scarcely be in doubt.

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[1] See author's paper, already referred to (op. cit.), for a full discussion of the symptomatology of these cervical cases.

[2] See Macewen's recent work, "Pyogenic Infectious Diseases of the Brain and Spinal Cord," for a description of these forms

To differentiate a pachymeningitis from myelitis is not practicable in many cases, because the two states are likely to be associated. The early appearance and the predominance of pain, with the slow onset of symptoms of system-lesions, such as spastic paraplegia, atrophic paralysis, and various types of anæsthesia, with involvement of the bladder late in the case, or even altogether exempt, would suggest a meningitis with gradual involvement of the cord.

In some cases of the hypertrophic form of cervical pachymeningitis, the resemblance to syringomyelia is so great that the history of severe injury can alone determine the diagnosis. It is well to recall, however, that trauma has figured among the causes of syringomyelia. Severe and persistent pain in the occiput, neck, and shoulders, following trauma in these cases, is the surest diagnostic sign.

Acute leptomeningitis, either spinal or cerebro-spinal, has some resemblance to tetanus. The exacerbations of opisthotonus, the trismus, the persistent course, the history of a wound, distinguish tetanus from meningitis.

Various forms of spinal meningitis have some superficial resemblance to locomotor ataxia, but the pupillary changes, the abolished knee-jerks (abolished also in meningitis of the lumbar enlargement and cauda equina) and the ataxia, without paralysis, serve usually to distinguish the latter disease.

Leptomeningitis is to be distinguished from hysteria by the absence of the stigmata of this disease.

Prognosis. In spinal caries a pachymeningitis is a serious complication. When pus is formed in, or finds its way into, the vertebral canal, or when inflammatory lymph is organized on the ventral aspect of the cord, the case usually advances to a septic or pyæmic stage, and the paralysis, resulting from involvement of the cord, is usually permanent. In mild cases, in which presumably pus does not form, a partial or even a complete recovery may occur.

In acute cerebro-spinal leptomeningitis, especially of pyæmic origin, as in suppurative ear-diseases, death invariably results.

In cervical pachymeningitis of traumatic origin, the resulting deformities are permanent, but life is often prolonged for years.

In some cases of alleged spinal meningitis, due to various causes, good recoveries are reported. In syphilitic cases recoveries are claimed by some from specific treatment. On the whole, however, all forms of the affection are grave diseases, and a guarded prognosis is to be given.

Treatment. Surgery alone can offer any hope of relief in cases of pachymeningitis due to spinal caries. Patients have been operated on successfully and relieved from the effects of pressure by tubercular nodules and organized lymph, and have been benefited also, probably, by the drainage of the vertebral canal. In one case observed by the writer, spontaneous drainage had occurred through a psoas abscess, the dura being perforated and the perforation communicating directly with the tract of the abscess. In the main, however, the worst of the disease process is on the ventral aspect of the cord and so not easily reached by the surgeon. Moreover, not pressure so much as a secondary myelitis is the cause of the cord symptoms, and this latter cannot be relieved by surgery. For fuller details, Chapter XXXIII. may be consulted. From the pathologist's standpoint, after many opportunities to study these cases post-mortem, the opinion has been reached by the writer that comparatively few of them are likely to be benefited permanently by operation.

In syphilitic cases, or even in cases in which syphilis is only suspected, in patients presenting evidence of the early stages of meningeal irritation, an active specific treatment should be commenced at once.

In traumatic cases, and, in fact, in all cases, rest and counter-irritation must be employed. Rest is of first importance in traumatic cases, and after rest comes time. One patient, at present under observation, went back to work as a stonemason after several years' invalidism due to a cervical pachymeningitis. Counter-irritation may be used by the method of blisters or of the hot-iron. In chronic cases iodide of potassium may be employed, but it gives doubtful prospect of relief. Electricity is probably useless in any form of spinal meningitis. Symptoms must be treated as they arise. Pain may demand relief, but the danger of a patient forming the opium habit must not be despised. Bed-sores should be guarded against, and when they occur should be treated with strict antiseptic precautions, because there is always danger that they may infect the system.

INTRA-SPINAL HEMORRHAGE.

Intra-spinal hemorrhage may be divided into two classes; first, meningeal, and, second, medullary. The first is the more common; it may be subdivided into the extra-dural and the subdural. The second or medullary variety, the true hematomyelia, is a rare affection, but not so uncommon, probably, as some writers contend. Recent medical literature contains the records of some striking and characteristic instances, confirmed by autopsy, of true hemorrhage within the substance of the cord, so that it is no longer reasonable to contend on *à priori* grounds that such hemorrhages cannot occur, and that myelitis has been mistaken in all such cases for hemorrhage. The reverse is more likely to be true, especially in traumatic cases, in which the so-called myelitis is probably a necrotic softening, having as its initial lesion a blood-clot or minute capillary hemorrhages. It has been assumed, almost universally, that for some mysterious reason the spinal cord is exempt from those accidents in the vascular system, such as embolism, thrombus, and hemorrhage, that play such a conspicuous part in cerebral pathology. This assumption has always appeared to the writer to be entirely unwarranted. The explanation for it, *i. e.*, that the long course of the spinal vessels relieves them from the effects of undue blood-pressure, seems to be not in accord with sound hydro-dynamics; and the claim that capillary aneurisms are never found below the oblongata,[1] at least needs confirmation. The fact, probably, is that cases of acute transverse softening (usually miscalled myelitis) are due primarily to a vascular defect, and are not truly inflammatory, *i. e.*, the result of infection. Some of these cases, as already said, may owe their start to capillary hemorrhages or even to a small blood-clot poured out into the substance of the cord.

The great authority of Charcot has been sufficient, apparently, to establish the opinion among most writers that hemorrhage in the cord is always the result of some primary lesion, usually the necrotic process miscalled by him acute myelitis. Hayem[2] elaborated this idea in an exhaustive treatise, in which he subjected all reported cases, up to 1872, to a critical study. His conclusion was (*op. cit.*, p. 146) that there was no unobjectionable case reported of primary hematomyelia. This conclusion is based, evidently, in part upon the erroneous ideas that prevailed formerly on the subject of inflammation. Many degenerative and some necrotic processes in the cord were ascribed universally to inflammation. According to the modern microbic pathology the term inflammation has a much more restricted meaning and is applied almost exclusively to the results of infection. It cannot be proved that hemorrhage and softening of the cord in all cases are due to infection. They seem rather to be due to causes which sometimes act primarily upon the vascular system, as trauma, cold, exposure, and overexertion. That they may even be due in a few cases to vascular degeneration, similar to what occurs in the brain, is not, in the writer's opinion, conclusively disproved. From a practical point of view it is not a vital question whether the hemorrhage precedes the softening, or *vice versa;* both, in fact, may be due to the same cause, and may be practically coincident. It appears, however, from reported cases, to be an undoubted fact that hemorrhage does occasionally occur primarily, *i. e.*, independently of a precedent myelitis or softening; that it produces quite characteristic symptoms, and that it may be rapidly fatal.[1]

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[1] Gowers propounds both these explanations in his text-book on diseases of the nervous system.

[2] "Des Hémorrhagies Intra-Rachidiennes," Thèse de Paris, 1872.

A most interesting and instructive case of non-traumatic hemorrhage into the spinal cord, illustrating this whole subject, has been put on record by Kindred.[2] The patient, a man aged fifty-nine years, in previous good health, was seized suddenly with pain in the cardiac region simulating angina pectoris; there were also dorsal pain, general spinal tenderness, and the "cincture feeling" in the chest and abdomen. The patient was able, after the first shock, to walk some distance, but then paralysis of the legs came on quickly. There was also quickly beginning and rapidly increasing numbness in the lower limbs and trunk up to the level of the affected segment. There were priapism, paralysis of the bladder and rectum, diminished reflexes, embarrassed respiration, and intestinal flatus. The initial agonizing pain soon disappeared. Death resulted in six and one-half hours. Consciousness was not affected. The autopsy revealed a bulging of the fourth dorsal segment. At this point a dark-red clot, about the size of an almond, lying almost wholly in the gray matter, was found. Adjacent parts were somewhat softened and congested. There was nothing else abnormal. There was no history of trauma, fatigue, excessive venery, vertebral disease, or toxæmia. It seems to me impossible, considering the history, the abrupt onset, the rapidly fatal issue, and the authentic autopsy in Kindred's case, to regard the hemorrhage as other than primary and the slight softening as secondary. The case suggests some vascular defect as the cause of the hemorrhage.

Starkey[3] also records a case of hematomyelia, traumatic in origin. A boy, aged thirteen years, after a heavy fall on the ice, walked home. After reaching home he had pain in the left shoulder, abdomen, and legs; then he began to be paralyzed, so that in two hours he could not walk. The pain and paralysis persisted; then later there were retention of urine, tachycardia, and obstinate constipation. Sensation failed as high as the fourth or fifth dorsal spine, the anæsthesia being slightly higher on the left. A small marginal zone of hyperæsthesia was present, and there was slight numbness in the left hand. The right knee-jerk was completely abolished; the left was diminished. There was no ankle-clonus. The abdominal and cremasteric reflexes were absent; the plantar were fairly brisk. The pupils were contracted

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[1] For a further discussion of this subject the reader is referred to a paper by Dr. Joseph Collins on "Hæmatomyelia and Acute Myelitis." N. Y. Med. Record, May 27, 1893.

[2] A Case of Non-traumatic Hemorrhage into the Spinal Cord, by J. Joseph Kindred, M.D. Med. News, February, 13, 1892.

[3] "A Case of Primary Hemorrhage into the Spinal Cord." Lond. Lancet, May 23, 1891, p. 1137.

and did not respond to light. The temperature was persistently high. The anæsthesia diminished slightly toward the last. The boy lived eight days, dying of pneumonia. At the *autopsy* no bruising, laceration, or hemorrhage of the spine or membranes was found. The pia was congested, but free from lymph. At the third dorsal segment was a hard localized swelling, and at this level the cord bulged. Above and below this bulging the cord was softened and had a slight yellow tinge. On section an extravasation of blood was found occupying nearly the whole of the transverse extent of the cord. Only a little white and gray matter on the right side could be made out. Above the hemorrhage, so far as the upper cervical region and below so far as the mid-dorsal region, the anterior and posterior cornua of gray matter of the left side were converted into a long cavity containing blood. The microscope showed that the hemorrhage was the only lesion; there was no inflammation.

Etiology. The most common cause of intra-spinal hemorrhage, either meningeal or medullary, is trauma. There may or may not be a coincident fracture of one or more of the vertebræ. In most traumatic cases there may be, and probably usually is, some coincident damage to the cord-tissue. The fracture, or the damage to the cord-tissue by crush or by compression, or both of these lesions, are often so conspicuous that the cases are more properly classed under one or other of these heads. In some cases, however, there may be no fracture, and the direct injury to the cord may not be conspicuous, but most or all of the symptoms may be due to the hemorrhage. In some cases of so-called concussion of the spinal cord, due to trauma, it is not unreasonable to suppose that minute capillary hemorrhages in the cord, or small extravasations of blood in the membranes, are the real causes of the persistent symptoms.

Overexertion, venereal excess, exposure to cold, and the convulsions of tetanus and strychnine-poisoning have been claimed as causes of these hemorrhages. In one case a hard labor was held to be the cause. Some of these causes, as venereal excess, are problematical. Scurvy[1] and purpura hemorrhagica may cause meningeal hemorrhage in exceptional cases. It has been conjectured that the infectious diseases, as smallpox and typhus fever, may do likewise, but observations are lacking.

Finally, according to the view already stated, it is not unreasonable to suppose that vascular defects may produce hemorrhages in the cord or its membranes. Certainly such a view cannot be ignored until further study of the possible diseases of the spinal bloodvessels shall have shown it to be unfounded. Such disease of the bloodvessels may be caused by syphilis.

Symptoms. The onset of spontaneous non-traumatic hemorrhage, especially into the substance of the cord, is always sudden. The accident causes a shock, as in Kindred's case, which may throw the patient to the ground, or even temporarily, according to some writers, obscure consciousness. From this shock the patient may rally and be able to walk for some short time and distance, but paralysis comes on soon, and rather rapidly. The initial shock is usually accompanied with pain, often of an agonizing kind; but this pain, as in Kindred's case, may disappear in a short time. This pain may simulate angina pectoris, and if accompanied with tachycardia and embarrassed respiration, as when the lesion is in the upper dorsal region, the simulation of a heart or lung-affection may be still more marked.

In traumatic cases the onset of symptoms is not always immediate upon the receipt of the injury; this depends upon the extent both of the injury and the hemorrhage. In comparatively slight accidents, where the hemorrhage is evidently slow, the symptoms may come on very gradually. This mode of onset was seen in Starkey's case. In more severe accidents, as in falls upon the feet and buttocks, or heavy blows upon the neck or back, the effects of hemorrhage are seen more quickly. Still there may be quite extensive extravasations of blood outside of or even within the dura, with quite gradual onset of symptoms in traumatic cases.

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[1] See an instructive paper on "Hæmatoma of the Dura Mater associated with Scurvy in Children," by G. H. Sutherland, M.D. Brain, Spring No. 1894. In one of Dr. Sutherland's cases an effusion of blood-stained lymph was found beneath the dura in the dorsal region of the cord.

In true hematomyelia, traumatic or otherwise, the symptoms are usually those of a complete, or almost complete, transverse lesion of the cord. The traumatic cases are most common in the cervical region. In these cases, therefore, there is usually complete paraplegia, with paralysis of the bladder and rectum, abdominal flatus, priapism, and anæsthesia so high as the distribution of nerves arising from the highest segment involved. It is a notable fact that in most of these cases the patellar, and often the superficial, reflexes are abolished. This is in accord with observations made by Bastian[1] that a total transverse section of the spinal cord abolishes all reflexes below the line of section, and that contractures of the limbs with increased reflexes indicate that the section is not complete. Both Kindred's and Starkey's cases, as well as most of those published by Parkin,[2] support this view.

The respiration is usually involved in these cervical and upper dorsal hemorrhages. In some cases the breathing becomes entirely diaphragmatic. Tachycardia is often present because apparently of irritation of the accelerator nerves of the heart. In some cases the pupils are affected because of involvement of the sympathetic centre in the cervical cord. When this is irritated the pupils may be dilated; toward the close, paralysis occurs and the pupils are contracted to a pin-point.

The course of some of these cases is extremely rapid. In one of Parkin's cases of true hematomyelia, caused by a fracture, death occurred in about thirty-six hours. In Kindred's case of spontaneous hemorrhage death came in six hours.

In meningeal hemorrhage the symptoms usually are not so severe as in true hematomyelia, and the prognosis is better. Pain, due to irritation of nerve-roots, may be a prominent symptom. Meningeal hemorrhage of the lumbar enlargement and of the cauda equina is caused by accidents, such as falls upon the feet or buttocks. In these cases the symptoms vary according to the extent of injury and amount of blood extravasated. The distribution of sensory symptoms may follow the distribution of individual nerve-trunks. The bladder often is involved, and the paralysis is confined, of course, to the legs, which may be rather flaccid with abolished knee-jerks.

Excessive sweating and hyperpyrexia are seen in cases of intra-spinal hemorrhage, especially toward the end in fatal cases.

Diagnosis. The identification of a lesion of the cord in these cases is usually easy enough. It is not likely that such an affection could be mistaken for a disease of any other organ, yet it is well to recall that the initial severe pain, with embarrassed respiration, has simulated angina pectoris. The speedy onset of paralysis, with priapism, anæsthesia, incontinence, and abolished reflexes, would distinguish the cord-lesion unerringly.

The supreme difficulty in these cases is to distinguish intra-spinal hemorrhage from transverse myelitis, acute white softening, pachymeningitis, and fracture. The sudden onset and often rapid course of hematomyelia distinguish it to a certain extent from transverse myelitis. The differentiation, however, is often uncertain and sometimes impossible, as can be readily understood from what has already been said about myelitis. Softening and hemorrhage may, no doubt, be due to the same causes, and some cases of so-called myelitis are not truly inflammatory processes at all, but rather necrotic ones. The practical necessity for these fine distinctions between processes which usually are all equally disabling is not urgent.

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[1] Medico-Chirurg. Trans., vol. lxxiii.

[2] "Seven Cases of Intra-spinal Hemorrhage (Hæmatomyelia)." Guy's Hosp. Rep., 1891.

In traumatic cases, however, the distinction between intra-spinal hemorrhage and fracture is of importance. Unfortunately the two conditions often coexist. Deformity of the spine is the surest sign of fracture; but even in cases in which the fracture can be detected and relieved the cord may be so injured by both crush and hemorrhage that permanent benefit is not gained. It must be borne in mind in these cases that the hemorrhage from fracture is not necessarily merely meningeal; it may be within the cord, as in one of Parkin's cases. In such a case the progress is usually rapid toward death.

Morbid Anatomy. In true hematomyelia the cord usually bulges at the seat of hemorrhage. The blood may be confined entirely to the interior of the cord. In Kindred's case the clot was the size of an almond, and lay almost wholly in the gray matter. The adjacent parts may be somewhat softened. In Starkey's case the blood occupied almost the entire transverse area of the cord. Above and below the seat of hemorrhage cavities extended through the gray matter to the extent of several segments. These cavities were evidently caused by a process of necrosis set up by the hemorrhage. A similar condition was noted by Parkin, the cavity being accurately limited to the area of the gray matter. Recently Van Gieson[1] has attempted to explain certain long, slender columns of necrosis in the cord, associated apparently with myelitis. They traverse long distances in the cord, both above and below an apparently circumscribed myelitis, and cavities, forming long tubes, may result from them. He considers these exceedingly rare, and has heard of but two of these cases. These "columns of necrosis" are formed, Van Gieson thinks, by hemorrhages into the substance of the cord, especially in traumatic cases. In time these columns break down, and "perforating necrosis" occurs. This is doubtless the correct explanation of these long, narrow cavities, which, however, are not so rarely reported as Van Gieson claims, one of Parkin's cases and the one by Starkey both showing this condition. The hemorrhage takes these courses upward and downward in the cord because they are the courses of least resistance; *i. e.*, it is easier for the blood to force its way up and down through the substance of the cord than to break through the firm and resisting enveloping membrane. In true hæmatomyelia there is no inflammation of the cord or its membranes. The pia, however, may be much congested.

In simple meningeal hemorrhage the blood may be outside of or within the dura. It often extends to a great length, even to the cauda equina. The cerebro-spinal fluid is blood-stained.

Treatment. No treatment avails in cases of true hematomyelia. The accident is almost always fatal, and usually rapidly so. In cases which survive—and such may occur among the numerous patients who are said to be suffering with "concussion" or "myelitis"—the future is likely to be one of chronic invalidism and hopeless paralysis. Hemorrhages into the membranes might possibly in some cases be relieved by surgery. No other treatment could be of much benefit. Ergot or one of its preparations should be given a trial, in hope that it would tend to check a continuance of the bleeding. Laminectomy, in case a clot can be successfully diagnosticated and localized, would be a proper operation.

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[1] N. Y. Med. Journ., June 2, 1894.

INJURIES TO THE SPINAL CORD FROM FRACTURES AND DISLOCATIONS OF THE SPINE.

From the surgical standpoint much can be written about fractures and dislocations of the spine that would be inappropriate here. I shall only describe briefly the injuries to the cord that are caused by these accidents. The injuries to the membranes have been described already under the respective headings of meningitis and of hemorrhage. Reference can be made to those descriptions. The injuries to the cord itself are usually by crushing, puncturing, or tearing. In patients who survive the accident attempts at repair are made, and a consequent thickening of the membranes and increased density of the cord substance (allied to scar-tissue) may result. In other cases softening results, sometimes miscalled myelitis, and this may be associated with hemorrhage into the tissue of the cord, which has been described above.

Deformities of various kinds in the spinal column are caused by fractures and dislocations, and when present are the best evidences of the lesion. It must not be forgotten, however, that in exceptional cases little, if any, deformity may result from fracture, and yet the cord may be seriously injured. It has been claimed even that a fracture of the body of a vertebra may quite readily escape observation; this seems to have been so in a case of fracture with displacement of the body of one of the dorsal vertebræ which I saw once with Dr. Willard in the Presbyterian Hospital. Extensive crush of the cord had resulted, as found post mortem, although but little, if any, deformity of the spine had been visible during life. It has been supposed that in some of these cases the displaced fragment, or even the whole vertebra itself, having been displaced, springs back into place. In the cervical region a fracture may cause a twist in the neck and displacement of the head, so as to suggest a dislocation; in fact, these two lesions may coexist. Thus in a young colored man under my care in the Philadelphia Hospital this deformity with twist was seen. He was struck between the shoulders by some bricks which fell from a fourth story, and probably sustained a fracture of one of the laminæ and of the transverse process, with resulting hemorrhage and damage to the cord.

Symptoms. Pain and deformity are common symptoms, but the latter, as just said, is exceptionally absent. The symptoms depend upon the extent of injury to the cord and upon the level of the lesion. In severe injuries the symptoms are those of a complete or almost complete transverse lesion of the cord. There is complete paralysis of motion and sensation below the point of lesion, with involvement of the bladder and bowels. In cervical injuries there are atrophic paralysis of the arms, spastic paraplegia, anæsthesia of various grades and areas, diaphragmatic breathing, tachycardia, involvement of the irides, pyrexia, and sweating. All reflexes may be lost, as explained elsewhere, below the seat of injury if this is totally transverse. This clinical picture varies according to the cases.[1] In cases of fracture of the first and second cervical vertebræ death is usually instantaneous. In cases of fracture of the third, fourth, and fifth cervical vertebræ the phrenic nerve may be involved, and hence breathing may soon become fatally embarrassed. In cases of fracture of the lower dorsal and lumbar region, which are rare, atrophic paralysis of the legs, instead of spastic paraplegia, is found as a rule.

In the young colored man above referred to the injury was to the mid and lower cervical cord. He had paraplegia with paralysis of the bladder, but with increased knee-jerks. He had also paralysis with rapidly advancing muscular atrophy of the arms and hands, with fibrillation. There was but little anæsthesia, but there were tachycardia, diaphragmatic breathing, sweating, and some pyrexia. There had been at times hiccough. The patient had great pain on passive movement of the hands. The case ended in death, but an autopsy was not obtained.

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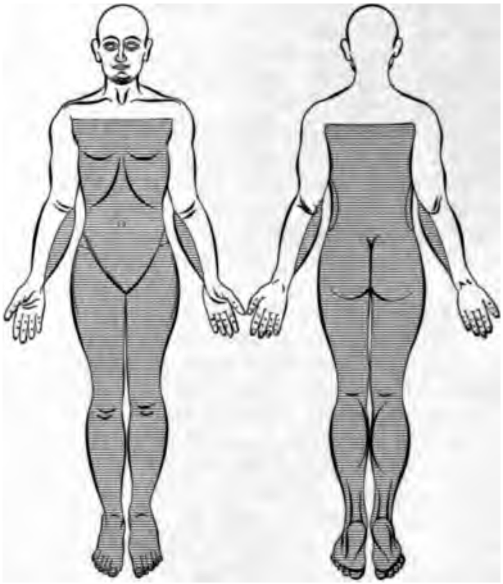
[1] See a paper by the author on "Traumatic Affections of the Cervical Region of the Spinal Cord, Simulating Syringomyelia." Journ. of Nerv. and Ment. Dis., June, 1894.

The following case of fracture of the cervical spine was seen recently by me in the Philadelphia Hospital. The report and drawings are by Dr. John A. Lichty, resident physician:

W. M., white, male, aged forty-seven years, was thrown from a cart and fell striking his right scapula and the right side of his neck. He was unconscious for one-half hour. When he came to himself he was lifted to his feet, but was unable to stand. In spite of this, when he was taken to a neighboring hospital he was refused admission because he did not seem to the examining surgeon to be severely injured, and he was rejected as a malingerer. The same day he was admitted to the Philadelphia Hospital. He was a large man, weighing about 200 pounds. He said that he felt well, except for the pain in his neck. His symptoms on admission were as follows: He had pain in the right side of his neck and in his right shoulder. He had also a peculiar tingling sensation in his right arm, as though it were "asleep." He said that his body from his shoulders down seemed dead or absent, and that he could not raise his feet. Examination revealed a swelling or prominence in the region of the right trapezius muscle, without injury to the skin and without subcutaneous ecchymosis. At the fifth cervical spine there was tenderness on pressure, and upon rotating the head there was diffused pain in the neck. No deformity could be found in the cervical spine, nor could crepitus be elicited. The pupils were equal and responded to light and distance. The throat was filled with mucus, causing a gurgling sound on breathing. The forearms were partially flexed, and the hands were flexed on the forearms. Motion in all the muscles of the upper extremities was free, except in the interossei; the patient was unable fully to open or close his hands, or to abduct and adduct his fingers. Sensation in the arms and hands was not impaired. Sensation was entirely absent from about the region of the third rib downward. Breathing was entirely by the diaphragm. There was marked increase of abdominal flatus, the abdomen being tympanitic. The bladder was paralyzed and distended with urine, and priapism was caused by the catheterization. The bowels were at first confined; later, evacuations occurred involuntarily. At the sacral region there was developed in a few hours a commencing bed-sore. There was a slight fibrillary tremor of the muscles of the legs below the knees. A similar tremor was noted in the arms, especially in the biceps, three days later. The patient could distinguish which sole was tickled or stroked, but could not distinguish the point of a pin. The patellar reflexes were absent, and there was no ankle-clonus. In the biceps the reflex was marked. There were both mucous and sonorous râles in the chest. The heart was slow and gradually became slower. The clinical history was briefly as follows: During the first two days the patient was very drowsy and slept most of the time. On the third day he became uneasy. At the end of forty-eight hours incontinence of urine set in, and twelve hours later incontinence of feces. Eighty-four hours after the injury anæsthesia was found on the ulnar sides of the forearms below the elbow, and in another day extended almost to the axilla. The strength of the biceps and brachialis muscles was not impaired. At this time the heels and tips of the toes became discolored as in trophic lesions. Anæsthesia gradually made its appearance first on the ulnar aspect of each forearm, and thence extended upward almost to the axilla (see Fig. 168). On the morning of the sixth day the patient was cheerful and hopeful; at 4 P. M. of the same day he complained of a peculiar feeling in the abdomen. His respirations were normal, but his lips were blue.

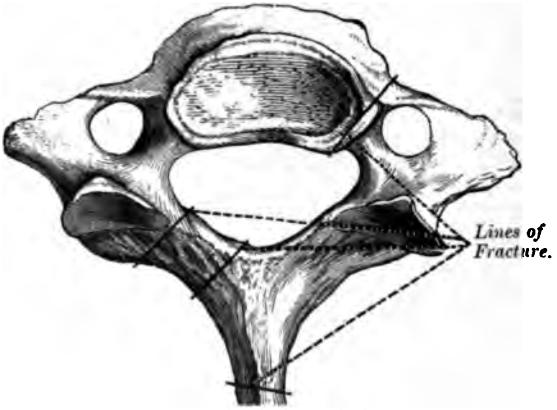
FIG. 168.



Anæsthesia in a case of fracture of the sixth cervical vertebra. (Philadelphia Hospital.)

At 4.30 o'clock he began suddenly to gasp for breath; the heart was slow but strong. Artificial respiration was begun, but in fifteen minutes he died. The autopsy showed a fracture of the sternum, between the first and second ribs. The sixth cervical vertebra was comminuted. There were two fractures of the left lamina, a fracture of the spinous process and of the right transverse process. (Fig. 169.) The body of the vertebra was dislocated

FIG. 169.

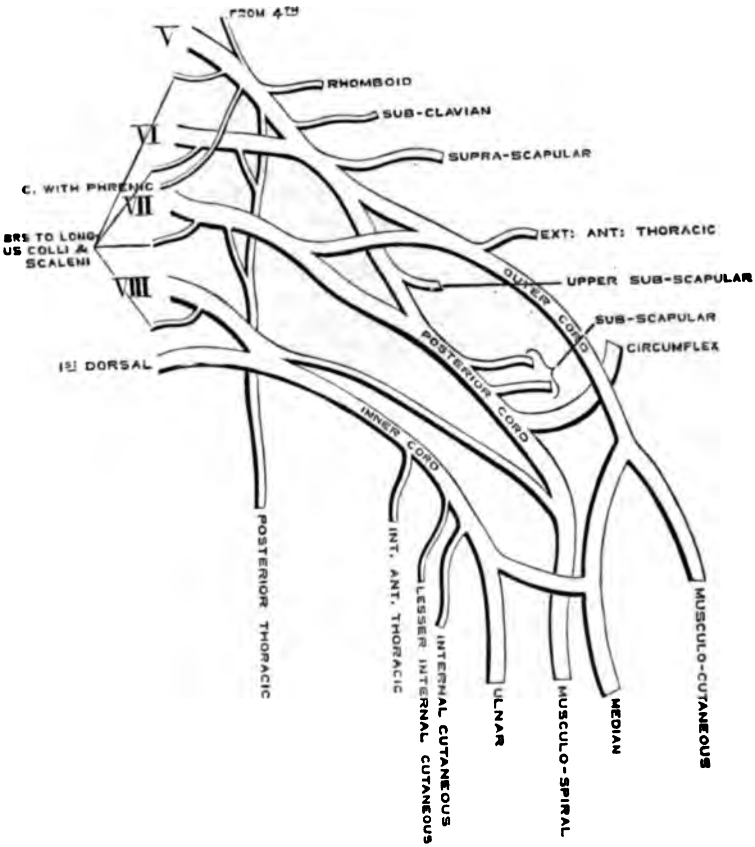


Fractures of the sixth cervical vertebra.

forward about one-half inch, and between the laminæ of the sixth cervical and the body of the seventh cervical vertebra the cord was compressed. At the point of compression there was a distinct blue line transversely across the anterior surface of the cord. Above and below this line the cord was softened. When removed it seemed to be held together only by the membranes. There was a slight extra-dural hemorrhage posteriorly. The ligamentum subflavum on the right side between the sixth and seventh cervical vertebræ was ruptured.

An *analysis of special* symptoms in this case leads to some interesting results. It is to be noted that there was not tachycardia, but slowing of the heart. This was the opposite of what is seen in some cases of cervical injury, and probably indicated that the accelerator nerves of the heart were paralyzed. This may be a theoretical explanation; the fact remains that in my observation it is rather an uncommon symptom.

FIG. 170.



Scheme showing the relation of the internal cutaneous nerve to the eighth cervical and first dorsal segments of the spinal cord. (Gray.)

The temperature in this case, instead of pursuing a febrile course, fell on the first day from a febrile point (102° F., almost) to a subnormal point, and pursued a markedly subnormal course to the end.

The distribution of anæsthesia in this case had significance as a localizing lesion. It may be noted that the inner aspects of the arms became involved gradually, whereas all the body, below a line drawn across the upper thorax, was anæsthetic. This is explained by reference to the figures. The seat of fracture was in the sixth cervical vertebra, which is opposite the seventh and eighth segments of the cord. The internal cutaneous nerve, which is distributed largely to the anæsthetic area on the inner aspect of the arm, arises from the lower limb of the cervical plexus, which in turn arises from the eighth cervical and first dorsal segments. (Fig. 170.) These two segments were involved in the crush and subsequent softening. Hence it is apparent why all the skin of the arm, except on the inner aspect, escaped. Again, the ulnar nerve arises in close proximity to the internal cutaneous, and it, too, was slightly involved, as shown by the paralysis of the interossei. The complete abolition of the knee-jerks, which was noted in this patient, is common, as already explained with reference to hemorrhage in the cord, in all cases in which the lesion is totally transverse. It is, therefore, an ominous sign.

Finally the mode of death in this case was characteristic of cases of grave injury to the cervical spine. It came suddenly by failure of respiration. Involvement of the centre for the phrenic nerve by the process of softening probably accounts for this. In injuries so high as the fifth cervical vertebra the phrenic may escape at first, because its first and second branches of origin come from the fifth and sixth cervical segments, which are slightly above this level, and in such a case the breathing will be purely diaphragmatic, the thoracic nerves arising below the lesion, and hence being paralyzed. But as the process of necrosis spreads, as it usually tends to do, the phrenic centre is invaded and death comes quickly, even suddenly. I have seen this mode of death in a number of these cases.

Treatment. The treatment for these cases is primarily surgical, and yet surgery cannot always bring these patients relief. It is not my province to discuss here, except briefly, the indications for laminectomy. The chance in all cases of fracture and dislocation is that the worse injury has been done to the cord at the time of the accident. Surgery obviously cannot repair a cord which is crushed, or which is disintegrated by a medullary hemorrhage, or which is bound down by a pachymeningitis. In saying this, I do not intend to discourage any rational operation; but I think the tendency has been too great to regard simply the injury to the spine and to forget, until too late, the worse injury to its more vital contents, the spinal cord. In cases with total anæsthesia, abolished knee-jerks, rapidly forming bed-sores, and diaphragmatic breathing, the operation of laminectomy is contraindicated, because these are the signs of a totally transverse lesion, such as a crush, a hemorrhage, or an area of softening, or a combination of all three of these lesions.

SPINAL CARIES.

After what has been written on the subjects of pachymeningitis and leptomeningitis, but little remains to be said from the standpoint of neuropathology on the subject of spinal caries. This disease affects the membranes and the cord secondarily, so that there result by infection a meningitis and myelitis, which have been described sufficiently under their appropriate headings. It ought always to be remembered that bone-pressure is of secondary importance in the vast majority of cases in causing the cord-symptoms.

Spinal caries is usually a disease of early life, but cases occur sometimes even in middle life. I have seen it occur in a man about forty years of age. According to Gibney,[1] 87 per cent. of cases begin before the fourteenth year. Sex is an unimportant factor: the disease occurs about equally in boys and girls. Statistics differ as to the seat of the disease. Taylor, quoted by Bradford and Lovett, found the favorite seats of the disease to be in the following order: Sixth and seventh cervical, eighth dorsal, second and third lumbar vertebræ. The least liable are from the first to the fourth dorsal, the eleventh and twelve dorsal, and the two extremities of the spinal column. Other observers have reached rather different conclusions. In my own observation among crippled children the dorsal and the dorso-lumbar vertebræ have been seen to be affected much more commonly than the cervical.

The surgical conditions accompanying spinal caries, such as the deformity and the abscesses, and the various internal pathological states, such as secondary tubercular infection of the lungs, spleen, kidneys, and mesenteric glands, are not to be described here.

Symptoms. The nerve-symptoms of spinal caries are those of a gradual involvement of the membranes and the cord. An early symptom sometimes is pain along one nerve-trunk, or in a limited area, simulating neuralgia. This is caused by irritation of the nerve-root. The symptoms of inflammation and compression (by inflammatory products) of the cord are similar to those that occur from other slowly advancing lesions of that organ. They consist of paralysis of motion below the lesion in most cases. If the lesion is cervical, the arms are the seat of muscular atrophy and paralysis due to involvement of the cervical enlargement, and there is spastic paraplegia. If the lesion is dorsal, a spastic paraplegia, without muscular atrophy, results. In severe cases the bladder and rectum are paralyzed. If the lesion is lumbar, a muscular atrophy or the symptoms of a multiple neuritis in the legs, with or without knee-jerks (according as the lumbar enlargement is or is not invaded), results. Sensory symptoms are usually much less conspicuous in spinal caries than motor. The reason for this is in the fact that the membranes are usually invaded from the body of the vertebra, hence the anterior and lateral aspects of the cord are usually most affected by the pachymeningitis and the compression from resulting inflammatory deposits. When these deposits, however, become great enough to cause total transverse compression, or in the rare instances in which such compression is caused by the diseased bone, the sensory paths are involved, and anæsthesia results.

In cervical cases the sympathetic is involved sometimes, causing alterations in the pupils either by irritation or paralysis, and causing also sweating. Respiration also may be affected in grave cervical cases. All the symptoms sometimes are suddenly aggravated by changes or progress in the lesion. The disease in many cases is extremely chronic, and some patients with spinal caries go for many years with marked angular deformity without involvement of the spinal cord. In fact, there is no necessary connection between the degree of deformity and the nerve-symptoms; these latter, in the vast majority of cases, depend, as already said, not upon compression by bone, but upon the invasion of the membranes by the infectious disease.

The following case occurred in the services of Dr. De Forest Willard and the writer at the Home for Crippled Children. It illustrates several pathological and surgical points of great interest.

H. R., aged four years, had caries of the fourth dorsal vertebra, the hump involving the fourth to the eighth dorsal vertebra. He developed gradually the symptoms of spinal pachymeningitis. He had spastic paraplegia, in which the symptoms were much more marked in the left leg. This leg was the more paralyzed, and the knee-jerk and ankle-clonus were much more marked in it. He lost control of his bladder and rectum, so that he lay constantly bathed in his urine and soiled with occasional discharges of feces. This condition aggravated a bed-sore, which formed in spite of every precaution. The elongated prepuce became inflamed, and in one place ulcerated quite through, so that it was necessary to circumcise the child. Sensation was involved, although not completely. It was discovered that the patient had analgesia, by the fact that he made no complaint and evidently felt no pain when the stitches were removed from the foreskin. There was anæsthesia of the legs, but accurate tests were difficult because of the patient's age. A psoas abscess was feared; an abscess formed over the left hip, and tubercular thickening of this joint was observed. In order to try to give some relief to this suffering a laminectomy was decided upon, and the operation was performed by Dr. Willard. When the laminæ and spine of the fifth dorsal vertebra had been removed a large tubercular nodule, pressing on the posterior and left side of the cord, was found. (See Fig. 171.) This was connected in front with a large tubercular cheesy mass, which was the *débris* of one or more bodies of the vertebræ. Such destruction had occurred that a finger could be passed down into the cavity past the cord. The mass pressing on the cord was dissected off from the dura, and the carious bone scraped out from its position in front of, or ventrad to, the cord. During this procedure one or two of the posterior nerve-roots, apparently diseased, were severed. The inflammation had been confined almost to the exterior of the dura (a pachymeningitis *externa*). When the parts had been cleaned of exudate the dura was found not perforated and, to a certain extent, healthy. After the cavity had been cleared out a drainage-tube was passed directly around the cord in order to drain the cavity in front of, or ventrad to, the cord. The appearance now was remarkable, as the spinal cord, encased in its dura mater, extended across this cavity like a great cable. The ends of the drainage-tube were brought out through openings in the muscles and integuments of the back, and the wound was closed. The child did not endure the operation well, and died subsequently from shock and exhaustion.

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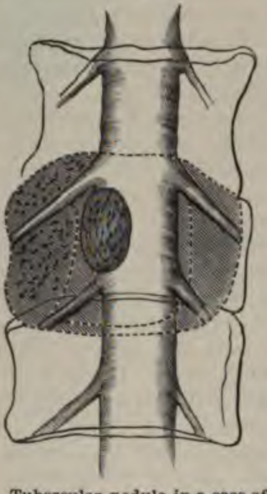
[1] Quoted from Bradford and Lovett: A Treatise on Orthopedic Surgery, New York, 1890.

FIG. 171.



Tubercular nodule in a case of spinal caries. The shaded space indicates the area exposed by the operation. (Home for Crippled Children.)

This case illustrates the extensive destruction of bone and the grave involvement of cord and membranes that occur in some of these cases. The tubercular nodule was similar to the solitary tubercle sometimes found in the spinal cord, independent of caries, which acts, as this did, practically as a cord tumor. The inflammation of the dura was not so extensive as was anticipated, and many of the symptoms, especially the unilateral features, were, no doubt, due to this nodule. The microscope, however, has revealed extensive myelitis with ascending and descending degenerations. The clearing out of the cavity of the diseased bodies of the vertebræ and the passage of a drainage-tube all the way around the cord were procedures, I believe, original with Dr. Willard. They furnish precedents which, I believe, must be followed in future cases, if the real focus of the disease is to be attacked and the cord relieved entirely from the noxious processes that environ it.

Diagnosis. Spinal caries may be mistaken for rheumatism, intercostal neuralgia, gastralgia, tumor of the spinal cord, thoracic and abdominal aneurism, the various forms of myelitis and meningomyelitis, and hysteria.

The disease could be miscalled rheumatism by only a careless observer, who was in the habit of attributing all obscure pains about the back and joints to "rheumatism." Intercostal neuralgia is sometimes closely simulated in the first stage of spinal caries; this is due to the fact that irritation of a sensory nerve-root causes a localized pain. Gastralgic and obscure abdominal pains also may be caused in the same way by beginning caries of the body of a vertebra. In all such cases a careful inspection of the spine, the detection of slight beginning deformity, of localized tenderness, of stiffness and resistance on movement of the back, and, perchance, if the case had advanced to involvement of the membranes, of paraplegia, or paraparesis, would suffice to distinguish the true character of the affection.

Tumor of the spinal cord, especially a tubercular nodule similar to the one reported above, might simulate the nerve-symptoms of spinal caries. The deformity of the spine would be the best distinguishing mark of caries. Carcinomatous and sarcomatous growths of the cord or spine are very rare, and not likely to appear in early life, when spinal caries is most common. There might be much difficulty in distinguishing a case of malignant growth, in which the vertebræ were involved, softened, and deformed, from a case of spinal caries in an adult. In malignant growth more severe pain would probably be present; the progress of the disease would be more rapid; secondary deposits might occur, and the familiar cachexia would be seen.

Erosion and pressure by an aneurism are to be distinguished by physical exploration of the chest or abdomen and the detection of the characteristic signs of aneurism.

From all other forms of myelitis and meningomyelitis the deformity alone could distinguish spinal caries.

The diagnosis of spinal caries in its early stages from hysteria is not always easy, especially in children. The disease itself may induce hysterical symptoms in neurotic children, and these may mask the true nature of the affection. Again, a slight trauma may induce an hyperæsthetic spine in an hysterical child and thus simulate spinal caries. It is important to distinguish correctly in either case. This can only be done by careful inspection of the back for the detection of the objective signs of caries, and by an examination for hysterical stigmata. When in doubt it is better to treat for incipient caries, and thus gain time, than to announce hastily what may be a premature judgment. In one such case in a child, in whom localized spinal tenderness followed a fall, I once applied a plaster-jacket in order to await developments. The patient made a prompt recovery.

Treatment. The treatment for spinal caries is, of course, largely surgical. To the neuro-pathologist, who is familiar with the morbid anatomy of the disease, and knows how imminent in every case is danger from infection to the cord and its membranes, as well as to other organs of the body, the usual treatment by braces and expectancy seems often inadequate and unsatisfactory. The difficulties and dangers, however, of the operation of laminectomy are not to be ignored, and they have been indicated already in the report of the above case. The greatest of these difficulties is in the fact that the carious process is in the vertebral body and consequently ventrad to the cord. Hence, it is almost inaccessible to the surgeon. Without reaching and eradicating this focus of infection it is not apparent how an operation, in many cases at least, can do permanent good. The procedure of Dr. Willard in the above case may supply a precedent for draining this cavity in more favorable cases. Another difficulty lies in the fact that in some cases the inflammatory exudate reaches far up and down the anterior aspect of the cord, often intimately adherent to the dura. To remove this, in some cases that I have examined post mortem, would seem to have been practically impossible. Still, with all the difficulties fairly stated, there is much opportunity for this most useful and rational operation. It should not be postponed too late. It is far better to do it before abscesses have formed in the psoas and iliacus sheaths, and dense inflammatory exudates have bound down the cord. Conspicuous symptoms of commencing involvement of the cord should be sufficient to indicate and to justify this conservative operation.

SOFTENING OF THE SPINAL CORD.

Acute softening of the spinal cord is discussed in most text-books as identical with, or as one of the terminal stages of, inflammation. Varieties of it even are described as the red, yellow, and white. There can be no doubt that many of the cases thus described are true examples of myelitis, and that the various colors or tints upon which such stress has been laid are simply the effects of various degrees and stages of extravasation of blood into the tissue. The question whether an acute, non-inflammatory softening, analogous to what occurs in the brain, ever takes place in the cord is still, perhaps, an open one. Most authorities, on what seems to me to be insufficient evidence, claim that there is no such softening. Strümpell says that it is still undecided whether there is such a softening, as a result of an obstruction of the vessels by a thrombus or an embolus. He states, however, rather contradicting himself, that he has seen one—but only one—spinal cord from such a case, in which the substance was changed into a soft pulp, which contained nothing but the remains of the nerve-tissue and fatty granular cells. I can add to Strümpell's the record of a case of my own, in which, as in his, a small but totally transverse softening was found in the dorsal region, without thickening of membranes, extravasation of blood, or any other sign of inflammation. Unfortunately, no microscopic study was made of this tissue.

It is not possible, of course, to decide such a purely pathological question on *a priori* grounds, but these seem to be the grounds so far upon which most writers base their arguments. Upon such grounds it seems that an equally valid argument can be maintained in favor of acute softening. In the first place, it is known that the spinal bloodvessels can suffer from infective and degenerative processes, which might very readily impair their walls and conduce to thickening of the coats, and hence to obstruction. Syphilis can so act. It is not certain that atheroma of the vessels may not occur. The argument of Gowers, based upon the anatomical arrangement of these vessels, does not appear adequate to disprove this. That degeneration of vessels in the cord does occur seems to be proved now by the cases of undoubted hemorrhage into its substance which are occasionally reported.[1] Finally, we must recast to some extent our ideas of inflammation in the spinal cord. It has been too much the custom to call most, if not all, disorganizing processes in the cord inflammatory. Our conception of the idea of inflammation, and our use of the term, must be more strictly guarded now in the light of bacteriological science. If the term is to be confined to the processes, such as congestion, migration of leucocytes, pus-formation, etc., that arise in tissue in antagonism to some morbific germ or germ-product, then it is certain that all examples of softening of the spinal cord are not inflammatory. The softening that occurs after trauma, and even after shock, is not thus always inflammatory. It is more truly necrotic, and probably depends primarily upon lesions of the vessels or capillaries, causing minute hemorrhages and thrombi. Even the thickening of membranes found in these cases, as in hypertrophic pachymeningitis cervicalis, has not the essential characteristics of an inflammatory process. It is exceedingly chronic in some cases; there is no pus-formation, and the results to the cord are gradual damage by degeneration rather than by inflammation. In such cases the thickening of membranes and proliferation of neuroglia are the results probably of the attempts of the tissue at repair.

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[1] See section on "Hemorrhage into the Spinal Cord," etc., in the present treatise.

This is largely as yet a pathological question. Clinically these cases will no doubt continue for a long while to be grouped with the cases of myelitis; and as, therefore, to describe them clinically would involve a mere repetition of the section on myelitis, this will not be done.

HYPERÆMIA AND ANÆMIA OF THE SPINAL CORD.

It was the custom formerly of some writers to erect an elaborate pathology and symptomatology upon the entirely hypothetical basis of hyperæmia and anæmia of the brain and spinal cord. It is almost needless to say that this custom, which never had a scientific warrant, is now falling fast into disrepute. I do not believe that we have any clinical types whatever that can be attributed exclusively, or even in a minor degree, to such slight variations in the circulation as would be indicated by these terms. It requires but a glance at the elaborate descriptions formerly in vogue to see that they include chiefly the symptoms of hysteria, neurasthenia, dyspepsia, and minor degrees of ill-health which were often poorly described and are now quite unrecognizable. There is no proof that sexual excess induces an hyperæmia of the spinal cord which can persist long enough and with such characteristic symptoms as to constitute it a distinct clinical entity. There is no reason to believe that intense brain-work and worry can cause an anæmia of the cord. There is absolutely no justification for the claim that special regions of the cord, as the anterior horns or the lateral columns, can be congested or depleted apart from the rest of the organ. Congestion of the cord may occur as a preliminary stage or as a result of some well-recognized process, as inflammation or strychnine-poisoning; or anæmia may result from excessive hemorrhage, as in typhoid fever or the puerperium, but in such circumstances the hyperæmia or anæmia is but a concomitant and not a separate disease. In grave forms of general anæmia, as pernicious anæmia, the spinal cord and the tissue of the nervous system in general suffer doubtless with the rest of the body. Some of the symptoms, as muscular weakness, hyperæsthesia, etc., may be due in part to the impaired functions of the cord and nerves. It is impossible, however, to regard such symptoms apart from the general symptoms of the disease, or to say how exclusively they are caused by involvement of the cord. It must be remembered, too, that organic changes in the cord have been found in pernicious anæmia. Posterior sclerosis has been reported by a number of observers. Hence, symptoms referable to the cord are not due simply to anæmia of the cord-tissue, but to actual organic changes. Such organic changes are due presumably to some irritant in the blood rather than to a mere decrease in the amount of any of its constituents.[1]

On the whole, it would be better to abolish entirely the terms hyperæmia and anæmia as designations for special forms of diseases of the spinal cord.

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[1] Since the above was written I have made a study of the anatomical changes in the spinal cord in a case of pernicious anæmia which occurred in my wards at the Philadelphia Hospital. Extensive changes were found in the posterior columns and beginning changes in the lateral tracts. The case will be reported in the forthcoming volume of the Hospital Reports.

CHAPTER XIX.

DISEASES OF THE SPINAL CORD.

(CONTINUED.)

BY MORTON PRINCE, M.D.

MYELITIS.

MYELITIS is an inflammation of the spinal cord. Besides occurring as an acute, subacute, and chronic process, several varieties of myelitis are distinguished according to the distribution of the inflammatory process in the cord. For practical purposes it is only necessary to recognize: 1. *Transverse (or diffuse) myelitis;* 2. *Disseminated myelitis;* 3. *Central myelitis;* 4. *Poliomyelitis.* The last is commonly known in its acute form as infantile paralysis, and is described as a distinct affection.

ACUTE TRANSVERSE MYELITIS. Acute transverse myelitis involves the whole thickness of the cord, and vertically extends, as a rule, through one or two segments. It thus involves both white and gray matter, and abolishes more or less all the functions of the cord at the seat of the lesions, and interrupts volitional impulses from the brain to segments of the cord below.

Etiology. The most common cause is injuries producing a laceration or bruising of the cord, or hemorrhage within its substance. Fractures and dislocations of the vertebræ, gunshot- and stab-wounds, produce these results, though, more logically perhaps, myelitis from these causes being secondary, the resulting lesion should be classed as lacerations of the cord, etc.; but hemorrhage may occur independently of trauma, and even when minute in amount is often a cause. Hemorrhage may follow injuries even when no recognizable injury to the vertebræ occurs. Myelitis has been known to follow violent contraction of the muscles of the back, and in these cases has been ascribed to an inflammation spreading from the connective tissue to the membranes and cord itself, but it is possible that in such cases there has been a temporary dislocation of the vertebræ, with spontaneous reduction. The cord would necessarily be bruised by the dislocation. I have known it to follow falls upon the back without demonstrable lesion of the bones. This seemed the most probable explanation, in view of the course of the symptoms. The statement that concussion is a cause of myelitis is frequently made. Such statements are quoted from one writer by another, and seem thereby to obtain more validity than should properly be ascribed to them. That concussion, pure and simple, can be a cause of myelitis may be properly questioned. It is more probable that when such seems to be the case there has been a direct blow upon the back at the time which during the excitement of the accident was not noticed by the patient, and which may have caused injury to the vertebræ, or hemorrhages within the cord itself.

Compression of the cord, as from tumors or caries of the vertebræ, is a common cause. Many cases are clearly traceable to exposure to cold, which is particularly regarded as an exciting cause. Thus, a person after working in the water and becoming thoroughly chilled, was attacked by myelitis. Several toxic agents may give rise to the disease. Thus, it sometimes follows or occurs in the course of various infectious diseases, especially measles, smallpox, typhus and typhoid fever. Syphilis is so frequently present that it must undoubtedly be regarded as an exciting cause, though it more commonly gives rise to the subacute or chronic form. The exact nature of the connection has not been accurately determined. Myelitis has been known to follow influenza, and two well-marked cases followed gonorrhœal infection. (Reynaud.) Myelitis has been found experimentally in animals poisoned by arsenic, and probably occurs in severe cases of arsenical poisoning with general neuritis in man.

The disease also is sometimes due to meningitis, the inflammation spreading to the cord. Various other causes of the disease have been described, such as gout, alcohol, overexertion, mental emotion, etc., but the effect of such agents in directly producing the disease must be regarded with considerable suspicion. Myelitis sometimes follows the caisson disease, and is then due to the disturbances of the circulation induced by high atmospheric pressure. In many cases, no satisfactory cause whatsoever can be obtained.

Anatomical Changes. On examining the cord the inflamed area may be detected by the cord being swollen at that point and reddened from injection of the membranes if death has taken place early in the disease. The cord is also softer. On section these changes are still more apparent. The distinction between white and gray matter is lost. The softening may be so great that the cord has a creamy consistence. If there have been much injection and extravasation of blood, the softened substance has a reddish color (red softening).

In other instances where the extravasation has been small in amount, the color may be yellow (yellow softening). This may also be the case when the disease has existed for some time, owing to the original red color having faded to yellow.

If there has been little injection of the vessels, the color may be white (white softening).

The axis-cylinders of the white fibres at first are swollen and the myelin becomes broken up and later disappears. Nothing remains of the fibres but the axis-cylinders, which in turn become degenerated, and finally are destroyed.

The cells of the gray matter likewise become degenerated; they become swollen and their contents granular. Within them may be seen highly refracting granules which have all the appearance of fat globules. Their processes also are shrivelled or lost. Vacuoles may be formed in them, but there is some difference of opinion regarding the interpretation of these. The intermediate gray matter is infiltrated with cells, round and angular, and the substance itself has a granular look.

The connective tissue of the white substance becomes filled with amorphous material and numerous cells (nuclei). Besides, there are many so-called Deiter's or spider cells to be seen in the neuroglia, which is thus much increased.

In old cases there may be a development of fibrous tissue in the form of trabeculæ.

The vessels are dilated, filled with blood, and have their walls studded with an increased quantity of nuclei. This is observed in both capillaries and arteries. The walls of the arteries are thickened in consequence, and their sheaths are packed with cells of different kinds. In some places extravasations of blood from the capillaries are conspicuous.

In severe cases, when the cord is much softened, nothing of the normal structure can be made out; but in the field of the microscope is found only a mass of detritus, consisting of swollen axis-cylinders, globules of myelin and fat, round cells, "spider cells," amorphous matter, red blood-cells, and peculiar bodies called "corpora amylacea." These are probably altered myelin. In such cases it is impossible to make sections of the diseased segment, which may be so softened as to flow like thick cream. When the destruction is not so severe as this the pathological changes may be distributed fairly evenly through a transverse section of the cord, or they may be more intense in certain spots in the gray or white matter, or in the lateral columns, etc. It must be remembered, however, that a portion escaping at a given level may be involved in a section just above or below, and thus a complete transverse myelitis may be produced.

The proportion in which the various elements—neuroglia cells and fibres—suffer varies in different cases.

In the later stages of many cases which survive absorption of the detritus takes place, the bloodvessels become thickened, and a great development of connective tissue takes place, so that the cord becomes hardened and gray; in fact, a condition of sclerosis develops. Finally, by a further extension of this process into the adjoining portions of the cord, an acute myelitis becomes transformed into a chronic myelitis.

An important fact, also, from a clinical point of view, is that the inflammation may extend up or down the cord, along the different special tracts; *i. e.*, up or down the pyramidal tracts or anterior horns. This extension of the inflammation does not take place in a direction corresponding to the physiological conduction, as do the secondary degenerations, but may be in either direction, up or down.

In disseminated myelitis reddish foci are scattered through the cord. They may be widely distributed. Histologically the same changes are found as in transverse myelitis. The microscope will reveal numerous small foci which cannot be detected by the naked eye. The distribution of these foci may be irregular, affecting a piece of the cord here and another there. Clinically, this is of importance. In central myelitis a crowding of nuclei will be observed round about the central canal, in addition to the other changes in the gray matter.

An important secondary effect which takes place in severe cases is the degeneration which occurs in some cases along the various white tracts, upward along the posterior columns and direct cerebellar tract, downward along the pyramidal tracts.

The nerve-roots may become involved in the inflammation and show changes similar to those in the cord. The motor nerves become affected with secondary degenerative changes when the gray matter with which they are connected is diseased.

Symptoms. The most characteristic symptoms of acute transverse myelitis, that is to say, those which constitute the salient features in the clinical picture, are rapidly developing paralysis of motion and sensation in the legs, or legs and arms, loss of control over the bladder and bowels, and alteration in the reflexes (increased or diminished) of the paralyzed portion of the body, to which may be added atrophy of and changes in the electrical reaction of those muscles which are directly connected with the diseased segments of the spinal cord. Nutritive changes in the skin, such as bed-sores and bullæ, later enhance the picture.

Such would be the general appearance presented by a well-developed case. It will be noticed that these symptoms, with the exception of the nutritive changes (which are not essential), are, for the most part, only the expression of the impairment of abolition of function of the spinal cord (negative symptoms), and that the grouping of the symptoms indicates that this abolition of function extends through the whole thickness of the cord.

But besides the grouping of the symptoms, that which distinguishes this form of myelitis from other varieties of spinal disease, is the manner of outset and development of the symptoms.

Careful observation of the latter is quite as important for the recognition of the disease as the grouping of the symptoms, especially as considerable variations occur in the symptoms in individual cases, according as the functions of the cord are impaired in a greater or less degree.

With variations in the localization of the inflammatory process, variations which may be longitudinal along the cord or lateral through its diameter, still further differences occur in the clinical picture.

Though the distinctively characteristic symptoms are those due to interference with the functions of the cord, other and general symptoms are sometimes, though not as a rule, observed at the onset of the disease. These are feelings of malaise, headache, fever of a mild degree, pains in the limbs and back, sometimes chills, and in children convulsions.[1]

These general symptoms may appear as prodromata or may accompany the special spinal symptoms. It is possible that in some cases at least they are to be ascribed to the exciting cause of the disease and not to the myelitis itself.

It will be noticed that these premonitory symptoms, when they occur, are not at all indicative of the development of a cord disease, and not until the patient begins to complain of some weakness of the muscles or impairment of sensation, such as numbness and "pins and needles" feeling, or, perhaps, in some cases, a difficulty in passing or retaining the urine, are we likely to suspect the true nature of the disease.

The order of development of the symptoms differs in individual cases according as the disease first attacks the motor, sensory, or other portions of the cord.

The rapidity of onset of the spinal symptoms varies sufficiently to enable us to distinguish three varieties, viz., the apoplectiform, the acute, and the subacute. This distinction is arbitrary, but has some clinical value.

In the acute variety the symptoms develop in the course of a few hours or a few days. The patient notices, perhaps, while walking or on getting up in the morning, that his legs feel heavy and numb. He finds difficulty in moving them and they tend to give way beneath him when walking. If he has been exerting himself, he ascribes the symptoms to tire, and rests for an hour, but finds on attempting to walk that instead of feeling better this weakness has increased. He is tempted to rub them to remove the numb feeling, but without benefit. The weakness increases until in the course of a few hours he cannot stand without support and is obliged to take to his bed. In bed he can move his legs up and down, but in a day or two even this power leaves him, and such control as he has becomes limited to feeble movements of the toes. The numbness also increases and the patient complains of various paræsthesiæ, which he describes as tingling, "pins and needles" feeling, and even pains in the legs. If tested, it will be found that sensation is blunted. With the progress of the disease this loss of sensation increases until it may be complete. If the disease is in the dorsal region, a characteristic symptom generally develops early, namely, a painful feeling of constriction, as if a tight band were bound round the waist, the so-called girdle sensation. This follows the distribution of the nerves arising from the level of the lesion. When the disease is in the cervical or lumbar region this sensation is replaced by pains in the arms and legs, respectively.

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[1] Convulsions have been observed in adults (Gowers).

Early in the course of the disease the patient finds he has lost control over his bladder and bowels. Retention or incontinence of urine is then present. The more rapid the onset the sooner the paralysis of the bladder and bowels develops, though naturally it may not be called to the attention of the patient for some hours after he has noticed the motor or sensory symptoms. Only in mild cases of limited extent do the sphincters escape. The exact character of the loss of control depends upon the seat of the lesion, whether it involves the centre for the bladder and rectum or is above them.

The loss of power and sensation in the legs may attain its height in the course of a few hours, or two or three days or a week may elapse before the maximum of injury has occurred. Sometimes the paralysis, notwithstanding a decidedly acute onset, at first is mild in degree and only increases slowly, so that ten days or more are occupied before the final stage is reached. The paralysis may then be complete.

As has been said, the order of development of the individual symptoms varies; sometimes the motor, sometimes the sensory are the first to be noticed, or it may be that a difficulty in passing the urine is the initial spinal symptom. The weakness of the legs is naturally more likely to be noticed by the patient, but careful inquiry is necessary to elicit the first symptom, as not only sensory loss is apt to be overlooked, but patients are not quick to distinguish between the sensory feeling of numbness and the motor feeling of heaviness, often mistaking one for the other.

The reflexes, deep and superficial, show various changes. Those immediately connected with the diseased segment of the cord are abolished. Those originating below it are increased, but during the first few days there may be a temporary diminution or loss of these, presumably from "shock."

The constitutional symptoms are not conspicuous, but there may be fever during the first few days, particularly in the severer cases. The thermometer may then record 102° or even 104°. Similarly anorexia, general malaria, headache, and perhaps chills, may be noted.

At first all the paralyzed muscles are toneless, and in the course of a few days the muscles directly in connection with the inflammatory focus waste. Those whose nerves originate below this focus do not waste, but regain their tone.

The electrical contractility of the wasted muscles usually exhibits the reaction of degeneration.

In this general picture of acute transverse myelitis, the paralysis of the legs has been principally referred to, as this is the most usual distribution of the paralysis and of the sensory loss, owing to the fact that the dorsal and lumbar portions of the cord are most commonly the seat of the disease; but when the disease is higher up, the functions of all the motor and sensory nerves originating at or below the lesions are impaired. If the disease is in or above the cervical enlargement, the arms are affected as well as the legs.

In severe cases the nutrition of the skin suffers and bed-sores develop with great rapidity; all the functions of the body subserved by that portion of the cord situated above the lesion are performed as in health. The intellect remains clear, the pulse and respiration are within normal limits. It is sometimes striking to see a patient lying perfectly helpless in bed, unable to move hand or foot, not even knowing the position of his arms or legs, yet fairly comfortable, conversing with natural ease and eating and sleeping as if almost without an ailment. It seems as if a live head were attached to a dead body. Owing to the anæsthesia such a person is not conscious of what in another would be discomfort, or of painful sensations, even when bed-sores or incontinence of urine and feces are present.

In the subacute cases the same group of symptoms is eventually developed, but the individual symptoms are much slower in developing. The paralysis or anæsthesia may occupy ten days or a month or more before reaching its height, and then the final degree of disability will generally be much less in degree. Sufficient power may even be retained to enable the patient to move about, and he may never actually take to his bed.

Individual symptoms may be absent, such as bladder paralysis, or may be present in only a mild form. But in such cases a history of a distinct and sharp onset, in the midst of health, on a particular day may be obtained, though the initial symptoms may have been mild in degree. In this respect the subacute cases may be clinically distinguished from the chronic variety.

In the apoplectic form complete paralysis comes on suddenly in the course of fifteen minutes or half an hour. The paralysis may be so sudden as to be suggestive of hemorrhage, and it may be even impossible to determine whether hemorrhage has occurred in such cases or not; but in the majority of such instances we should probably be safe in inferring the existence of some hemorrhage.

Sometimes a patient wakes up after a night's sleep to find himself paralyzed. Under such circumstances, as it is impossible to determine the duration of the onset, it is often difficult to decide whether or not hemorrhage has occurred.

It remains to consider the individual symptoms more fully in detail.

Sensation. There are three phenomena of disturbed sensibility which should be looked for: First, a "girdle pain;" second, a zone of hyperæsthesia corresponding to the upper level of the lesion; and, third, a loss or diminution of all forms of sensibility, or the retention in a greater or less degree of either touch or pain, and the loss of the other of these two.

The well-known "girdle pain" or painful sense of constriction is due to the irritation of the nerves passing through the upper edge of the inflammatory focus. Its seat necessarily varies with that of the lesion. As the dorsal cord is most commonly affected, its most common seat is somewhere between the ensiform cartilage and the waist (crest of the ilium).

Pains of an analogous character may be present in the arms and legs when the cervical and lumbar cords are respectively only partially involved. When these enlargements are totally affected the pains cease in the limbs.

The pain may be felt about the perineum and anus. A zone of hyperæsthesia will usually be found at the same level as the girdle pain and corresponding to the same portion of the cord. It is usually about two inches wide, and should be carefully looked for. Both these phenomena are important from the fact that they indicate the upper limit of the disease. The girdle pain is also a valuable, though not absolute, evidence of organic disease in the cord. The degree to which sensation is lost varies in different cases. The loss may be absolute or slight only, though, as a rule, the perception of touch and pain and temperature is impaired to a very marked degree. As in the peripheral nerves, the sensory fibres seem to have a greater resisting power than the motor fibres, and it is not uncommon to see extreme paralysis of the muscles, with a slight or moderate amount of anæsthesia. There is always, however, some loss of sensation, or, in mild cases, paræsthesia, otherwise the lesion is not transverse. Pain and touch may be unequally affected, and one may be abolished without the other.

The anæsthesia extends from below upward to the upper level of the lesion, and consequently is of value as a guide in determining this limit, especially in the dorsal region. The line of division between the sound and affected portions of skin is not, however, sharply defined, and some skill is necessary in determining the boundary. The anæsthetic line may not be at symmetrical heights on the two halves of the body.

Paralysis. The paralysis may be absolute, so that the affected muscles cannot be moved in the slightest degree, or more or less power of motion may be left; or some movements of a limb may be lost completely and others retained. Aside from differences in the intensity of the process, these variations depend in part (not wholly) upon the fact that the different groups of muscles are represented in more than one level of the cord, so that the destruction of any one segment may not completely abolish the corresponding movement which may be affected by the next adjoining spinal segment above. But in acute cases it is usual for the degree of paralysis to be such that the affected limbs are of no practical use.

When the paralysis is not absolute the flexors are said to be more affected than the extensors—a fact difficult to explain, if true. The distribution of the paralysis depends upon the vertical position of the lesion. When the lumbar enlargement is alone involved, the paralysis is limited to the legs and sphincters. In the dorsal region the corresponding intercostal and abdominal and back muscles are included in the paralysis.

In cervical myelitis the arms, as well as the legs and body, are paralyzed. If the lesion is as high as the third and fourth cervical segments, the diaphragm (phrenic nerve) is also paralyzed, and death takes place. In rare cases where the myelitis was of limited extent the diaphragm escaped, though the neck muscles were paralyzed and wasted.

The pupils may be affected in cervical myelitis. This may be explained by the fact that nerves for the radiating fibres of the iris pass down the cord from the nucleus of the third nerve to reach the lowest cervical or first dorsal nerve, by which they join the cervical sympathetic, and then, ascending with the internal carotid artery, pass to the ciliary ganglion. Paralysis of these fibres would lead to contraction of the pupils.

Atrophy and Electrical Reactions. In consequence of the damage to the cells in the anterior cornua the muscles supplied by the nerves arising from the diseased segment become flaccid at once, and waste. In the course of ten days or a fortnight electrical examination will show the reaction of degeneration in these wasted muscles. This degenerative atrophy must not be mistaken, as it too often is, for the simple wasting of muscles occurring in any paralyzed or immobile limb. In mild cases, when the atrophy is slow, only diminished contractility will be found. But muscles connected with the cord below the seat of the disease do not atrophy or exhibit electrical changes. So that in lumbar myelitis the legs, or particular muscles of the legs, waste; in cervical myelitis the atrophy is limited to the arms, though the legs are paralyzed.

Reflexes. The reflexes that pass through the diseased segment are necessarily abolished; consequently, in lumbar myelitis all the reflexes—deep and superficial—of the legs (knee-jerk,[1] cremasteric, etc.,) are lost. In dorsal myelitis this loss is confined to the trunk reflexes, and in cervical myelitis to the arm reflexes.

The reflexes that are subserved by the segments below the level of the lesion may be (not always) temporarily lost (or diminished), presumably from shock. These reflexes may be recovered in the course of a few hours, and later become exaggerated; or they may remain absent for days and weeks. In traumatic cases with crushing of the cord they are especially liable to be lost, and may continue absent for days or weeks, and in rapidly fatal cases may not return at all.[1]

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[1] The knee-jerk and other tendon phenomena are spoken of as reflexes, although, in accordance with the observations of Gowers, they should more properly be regarded as conditions of muscular (myotatic) irritability. But as myotatic irritability is dependent on the integrity of the reflex arc, it is a quasi-reflex phenomenon, and clinically is more conveniently considered as a reflex.

The exaggeration of the superficial reflexes is due to the cutting off of the normal inhibitory influences from the brain. That of the deep reflexes (myotatic irritability) principally depends upon the secondary descending degeneration in the pyramidal motor tracts, but not entirely upon this, as there is reason to believe that loss of inhibition will also cause an increase in these reflexes, and that this is a factor at first. Therefore, in cervical myelitis with the loss of the arm reflexes, and in dorsal myelitis of corresponding trunk reflexes, there are ankle-clonus and increased knee-jerk, cremasteric, plantar, and other reflexes.

With the development of the descending degeneration in the pyramidal tracts a spastic condition of the legs develops. Certain exceptions to this statement occur, the explanation of which must be considered in doubt; viz., according to Bastian, when the transverse lesion is *complete* the reflexes subserved by the cord below are not increased, but lost. Bastian's observations have been corroborated by others (Herter, Leyden, Miles, Babinsky), but the interpretation of them is still an open question. That of Gowers, that the loss of reflexes is due to the inflammation having extended downward to the lumbar enlargement, is undoubtedly correct in certain cases, though the post-mortem findings have sometimes only shown degenerative changes in the contents of the cells, without diffuse inflammatory softening, but the coexistence of atrophy and loss of faradic contractility of the muscles shows that some sort of degenerative change must have been present in these cases. But it remains to be proved that a descending inflammation or some other kind of degeneration always is present in such cases.

Bladder and Bowels. In complete transverse myelitis there is necessarily always loss of control of the sphincters, but when the myelitis is only partial they may occasionally escape. Paralysis of the sphincters may be the first symptom, and, on the other hand, when recovery takes place control over the sphincters may be regained, while there is still considerable paralysis. But in mild cases only a slight weakness of the bladder may be present, rendering it difficult for the patient to retain or expel his urine.

There are two forms of incontinence of urine which may be present according to the seat of the lesion, and according as there is paralysis of the sphincters or the detrusor. When the lower portion of the lumbar enlargement is diseased the sphincter is paralyzed and the urine dribbles away as fast as it is secreted. When, on the other hand, myelitis is above the lumbar cord, the vesical centre being intact, the sphincter holds, and there is a full bladder. The bladder may then empty itself at intervals spasmodically, or, as the wall becomes weaker and the bladder empties itself more and more imperfectly, an overflow becomes established—overflow incontinence. It is important to determine which of these two conditions is present, inasmuch as a persistently full bladder may lead to disease of the kidneys. Similar differences in the form of the rectal paralysis occur. In lumbar myelitis there is incontinence of feces from paralysis of the sphincter. This, however, may not be obvious on account of the constipation which is usually present, and which is a symptom of myelitis and dependent upon the connection between the sympathetic system and the cord. In dorsal and cervical myelitis there may be involuntary evacuation of the rectum at intervals.

There is a tendency for the urine to become alkaline. This has been at-tions of skin is not, however, sharply defined, and some skill is necessary in determining the boundary. The anæsthetic line may not be at symmetrical heights on the two halves of the body.

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[1] Necrotic changes in the cord caused by injuries of this kind are not, strictly speaking, myelitis although they are followed by inflammation.

Paralysis. The paralysis may be absolute, so that the affected muscles cannot be moved in the slightest degree, or more or less power of motion may be left; or some movements of a limb may be lost completely and others retained. Aside from differences in the intensity of the process, these variations depend in part (not wholly) upon the fact that the different groups of muscles are represented in more than one level of the cord, so that the destruction of any one segment may not completely abolish the corresponding movement which may be affected by the next adjoining spinal segment above. But in acute cases it is usual for the degree of paralysis to be such that the affected limbs are of no practical use.

When the paralysis is not absolute the flexors are said to be more affected than the extensors—a fact difficult to explain, if true. The distribution of the paralysis depends upon the vertical position of the lesion. When the lumbar enlargement is alone involved, the paralysis is limited to the legs and sphincters. In the dorsal region the corresponding intercostal and abdominal and back muscles are included in the paralysis.

In cervical myelitis the arms, as well as the legs and body, are paralyzed. If the lesion is as high as the third and fourth cervical segments, the diaphragm (phrenic nerve) is also paralyzed, and death takes place. In rare cases where the myelitis was of limited extent the diaphragm escaped, though the neck muscles were paralyzed and wasted.

The pupils may be affected in cervical myelitis. This may be explained by the fact that nerves for the radiating fibres of the iris pass down the cord from the nucleus of the third nerve to reach the lowest cervical or first dorsal nerve, by which they join the cervical sympathetic, and then, ascending with the internal carotid artery, pass to the ciliary ganglion. Paralysis of these fibres would lead to contraction of the pupils.

Atrophy and Electrical Reactions. In consequence of the damage to the cells in the anterior cornua the muscles supplied by the nerves arising from the diseased segment become flaccid at once, and waste. In the course of ten days or a fortnight electrical examination will show the reaction of degeneration in these wasted muscles. This degenerative atrophy must not be mistaken, as it too often is, for the simple wasting of muscles occurring in any paralyzed or immobile limb. In mild cases, when the atrophy is slow, only diminished contractility will be found. But muscles connected with the cord below the seat of the disease do not atrophy or exhibit electrical changes. So that in lumbar myelitis the legs, or particular muscles of the legs, waste; in cervical myelitis the atrophy is limited to the arms, though the legs are paralyzed.

Reflexes. The reflexes that pass through the diseased segment are necessarily abolished; consequently, in lumbar myelitis all the reflexes—deep and superficial—of the legs (knee-jerk,[1] cremasteric, etc.,) are lost. In dorsal myelitis this loss is confined to the trunk reflexes, and in cervical myelitis to the arm reflexes.

The reflexes that are subserved by the segments below the level of the lesion may be (not always) temporarily lost (or diminished), presumably from shock. These reflexes may be recovered in the course of a few hours, and later become exaggerated; or they may remain absent for days and weeks. In traumatic cases with crushing of the cord they are especially liable to be lost, and may continue absent for days or weeks, and in rapidly fatal cases may not return at all.[1]

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[1] The knee-jerk and other tendon phenomena are spoken of as reflexes, although, in accordance with the observations of Gowers, they should more properly be regarded as conditions of muscular (myotatic) irritability. But as myotatic irritability is dependent on the integrity of the reflex arc, it is a quasi-reflex phenomenon, and clinically is more conveniently considered as a reflex.

The exaggeration of the superficial reflexes is due to the cutting off of the normal inhibitory influences from the brain. That of the deep reflexes (myotatic irritability) principally depends upon the secondary descending degeneration in the pyramidal motor tracts, but not entirely upon this, as there is reason to believe that loss of inhibition will also cause an increase in these reflexes, and that this is a factor at first. Therefore, in cervical myelitis with the loss of the arm reflexes, and in dorsal myelitis of corresponding trunk reflexes, there are ankle-clonus and increased knee-jerk, cremasteric, plantar, and other reflexes.

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There is a tendency for the urine to become alkaline. This has been attributed to an alteration in the secretion, apart from the decomposition which is liable to follow the retention. This decomposition, which is common, is often, however, if not always, due to the introduction of unclean catheters, and can frequently be avoided.

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As a result of the decomposition cystitis may develop. This, like the bed-sores, is probably facilitated by the cord lesion impairing the nutrition of the bladder and its resisting power. Cystitis may lead to ulceration and perforation of the bladder, peritonitis, etc., and retention of urine to disease of the kidneys.

Priapism is common when the disease is above the lumbar enlargement.

Optic neuritis has been observed in a few cases. It seems to be more common in the diffuse form, and there is reason to believe that it may be properly regarded as caused by some toxæmic agent, the same which has in these cases been the exciting cause of the myelitis.

Trophic disturbances are common. The temperature of the skin of the limbs is at first raised, at times even above that of the mouth. This may be explained by the interference with the functions of the sympathetic vasomotor nerves through their connections with the cord. Later the temperature falls below normal. The nutrition of the skin suffers, in that it becomes dry and harsh, especially when there is atrophic degeneration of the muscles, and the least pressure is liable to produce bed-sores. The resistance to the electric current under such circumstances is so great as to make it difficult to obtain muscular contractions without using a battery of high electromotive force, and to lead to erroneous observations unless careful electrical measurements are made. The most common seat of bed-sores is over the sacrum. These should be carefully watched for. These bed-sores are sometimes malignant in character. Sloughing appears suddenly under so slight a provocation, such as pressure of a hot-water bottle, as to appear spontaneous, but it may almost always be attributed in part to an external cause,[1] although such causes would have no such effect in healthy persons. Consequently such lesions are properly attributable to the central lesions. The sloughing may involve not only the soft parts, but even the bones of the spinal column. Such complications are to be dreaded, as in their malignant form they may lead to great destruction of the tissues, and even septicæmia and death. Cellulitis in the lower part of the body, especially about the bladder and rectum, has been observed with subcutaneous emphysema (Gowers).

Œdema of the paralyzed limbs occurs, and is presumably due to vasomotor disturbances.

Joint disease, resembling somewhat that occurring in tabes, has been observed[2] following traumatism. A case in which spontaneous fracture of the thigh, without pain, occurred has come under my observation. The myelitis in this case followed fracture of the spine. Such trophic lesions are rare. Why they should be common in such diseases as syringomyelia, Morvan's disease, and tabes, and so infrequently observed in myelitis, is not plain, unless it be that they are dependent upon changes in the nerve-roots.

Contractures and Spasms. The spastic condition of the legs, which develops in the later stage of the disease in consequence of the secondary descending degeneration, has already been referred to. Although ordinarily spasm of the extensors preponderates over the flexors, occasionally the reverse is the case. Under these circumstances, owing to the continuous flexion of the joints and the subsequent shortening of the muscles, contracture develops and the limbs become fixed in a flexed position.

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[1] In a traumatic case described by Hammond ("Diseases of the Nervous System"), three large sloughs over the sacrum and both hips occurred within six hours of the injury. The trophic lesions observed in other cord diseases, as syringomyelia, show that the central lesion alone may induce such necrotic processes.

[2] Goldthwait: Transactions of the American Orthopædic Assoc., 1892.

Course of the Disease. The disease in the acute form reaches its height in three or four days or a week. In the subacute form this period is, of course, prolonged. There may be a succession of distinct acute attacks, during which the paralysis becomes more profound and more extended. The disease may be limited to the segments first affected, or may extend up and down the cord. In the former case the disease remains stationary for a variable time, and then improvement begins. In mild cases, after a week some change for the better may be noticed. In severe complete lesions a year may pass without improvement, and none may take place at all. Between these two extremes all grades may be observed. When the disease extends beyond the segments first attacked the progression upward will be indicated by the paralysis of successive muscles, by an increase in the area of anæsthesia, or by the implication of more reflexes. Its downward extension can only be recognized by the condition of the reflexes, the state of nutrition and electro-contractility of the muscles, and the condition of the sphincters. After the disease has reached its height its course is slow, unless there has been an early extension of the disease into the centres for the respiratory muscles or into the medulla oblongata, when, of course, death occurs. When improvement begins some return of sensation is usually first noticed, and often recovery of control over the sphincters is among the earliest signs. Sensation may be nearly recovered with little or no gain in motor power, and control of the sphincters may be established while considerable paralysis persists. Sometimes recovery is complete, but months must be counted upon as necessary for this result, and sometimes it is not until a year or two have passed that the total amount of improvement that will be attained has taken place.

Though considerable improvement is not incompatible with a certain amount of increased myotatic irritability, a high degree of spastic condition is a bad omen, as it indicates descending degeneration in the voluntary motor tracts in the cord. Some cases pursue a relapsing course. This is, however, more common in disseminated myelitis.

When death takes place it is either from extension of the disease to the respiratory centres or to the medulla, or from exhaustion consequent on bedsores, cystitis, or impaired nutrition. In some cases there may be a general weakening of all the vital forces. Death may take place in consequence without special local cause.

Termination. If complete recovery does not occur, various secondary changes usually persist in the cord and produce characteristic symptoms. The principal are: 1. *Descending degeneration*, with resulting spastic paralysis of a greater or less degree. 2. *Ascending degeneration* along the posterior column, producing ataxia. 3. The disease may clear up from the white matter and leave permanent destructive effects in the anterior gray matter below. In such cases a certain amount of paralysis, with atrophy and loss of electrical reaction, will persist in certain groups of muscles, and present the appearance of an anterior poliomyelitis. In all these cases, if the patient is seen for the first time in the very late stages of the disease, only very careful inquiry into the history will enable us to recognize the origin of the spinal condition.

It should always be borne in mind that neither motion nor sensation may be completely recovered, and some loss of both and some weakness of the sphincters may persist during the patient's lifetime.

Localization. It is often important in traumatic cases and those secondary to processes outside the cord to determine the localization of the inflammatory focus, including its lower and upper limit. The main points to be relied upon have already been stated and need not be repeated. In cases of partial myelitis involving the lumbar or cervical enlargements, special groups of muscles may alone be paralyzed, according to the height of the lesion in the cord; similarly the anæsthesia may be limited to special areas. The reflexes may also be differently affected. A careful study of Figs. 195 and 196, Chapter XX., will enable one to locate the site of the lesion. But it must be borne in mind that our knowledge of the localization of spinal functions is only approximately correct, and we cannot count upon absolute accuracy. Furthermore, the evidence we have points to the fact that the various groups of cells corresponding to the different sets of muscles have their seat in several spinal segments, and the injury of one segment may only weaken without absolutely paralyzing the corresponding muscles.

The accompanying table shows the principal symptoms present in lumbar, dorsal, and cervical myelitis.

	Lumbar myelitis.	Dorsal myelitis.	Cervical myelitis.
Paralysis.	Paraplegia.	1. Dorsal, abdominal, and intercostal muscles according to height of lesion. 2. Legs.	Neck muscles, diaphragm, arms, trunk, and legs.
Sensation.	Pains in legs, or girdle pains around loins; hyperæsthetic zone around loins; anæsthesia of legs, complete or uneven distribution.	Girdle pain and hyperæsthetic zone between ensiform cartilage and pubes.	Hyperæsthesia and pains in certain nerve distributions of arms; below this anæsthesia of arms, body, and legs.
Atrophy.	Of legs.	Of dorsal and abdominal (and intercostal muscles, not subject to examination) corresponding to height of lesion; sometimes mild and slow of legs.	Atrophy of neck muscles (rare) or more commonly of arms.
Electrical reaction.	R.D. in atrophied muscles; or in mild cases quantitative diminution.	R. D. in dorsal and abdominal muscles; slight quantitative changes only in legs when wasted.	R. D. in atrophied muscles.
Bladder.	Incontinence from paralysis of sphincter.	Retention, or intermittent incontinence from reflex action; later from overflow. Cystitis common.	Same as in dorsal myelitis.
Bowels.	Incontinence from paralysis of sphincter, disguised by constipation.	Involuntary evacuation from reflex spasm, or constipation.	Same as in dorsal myelitis.
Reflexes, superficial.	Lost.	Temporary loss, then rapid increase.	Same as in dorsal myelitis.
Reflexes, deep.	Lost.	Temporary loss, then slow increase.	Same as in dorsal myelitis.
Priapism.	Absent.	Often present.	Often present.

Diagnosis. Typical cases of acute transverse myelitis are easily diagnosed. It is only in the mild or partial form that there may be a question of the diagnosis. Great difficulty may then be met with. Chief reliance must be placed upon a rapidly developing paralysis, with loss of sensation and loss of control of the sphincters. Further than this it is not necessary to repeat what has already been said. Here can only be mentioned the diseases with which transverse myelitis is most likely to be confused and a few of the principal symptoms which will serve as the most useful guides. A knowledge of the diseases themselves will alone enable us to make the diagnosis in doubtful cases.

The principal diseases from which acute transverse myelitis is to be distinguished are: Central myelitis, poliomyelitis, disseminated myelitis, hemorrhage, embolic or thrombotic softening, multiple neuritis, neuritis of the sacral plexus, Landry's paralysis, spinal meningitis, and hysterical paralysis.

The distinction from central myelitis is difficult. The latter is the most malignant form that occurs, and, being an inflammation of the gray matter and extending up and down the cord for a considerable distance, produces more diffuse symptoms.

It may be suspected when symptoms indicative of an intense myelitis develop, attended by fever and by signs of damage to the trophic centres of the muscular system through a considerable vertical extent of the cord. A focal central myelitis sometimes follows trauma. In such cases there may be the dissociation of sensation, combined with limited atrophy of certain muscular groups, such as is observed in syringomyelia, or the atrophy and paralysis may alone be present without sensory loss. I have observed two instances of such a focal myelitis, and a few have been reported in the journals.

In poliomyelitis sensation is not affected, the sphincters escape, and there is no girdle pain or bed-sores. Hemorrhage need only be confounded with the apoplectic form. In cases of such sudden onset it is difficult to make the distinction, but we shall rarely go wrong if we assume the occurrence of some hemorrhage; further, in hemorrhage severe pain is much more prominent, and when present would be sufficient to make the distinction.

The distinction from disseminated myelitis must be based upon the fact that the symptoms in this form are more mixed and indicative of foci of inflammation scattered through the cord. Instead of the compact symptoms indicative of disease of one or two segments, the sensory, paralytic, and other phenomena will be more scattered and appear as paralysis of distinctly separated groups of muscles, patches of anæsthesia, more or less irregularly distributed. When the foci of inflammation are numerous, so as to produce a coalescence of the symptoms, the differentiation may be difficult.

In multiple neuritis there is rarely paralysis of the sphincters, nor is there a girdle pain, hyperæsthesia, or bed-sores. Loss of sensation is never so profound as is sometimes the case in myelitis. The paralysis is less rapid in development, and in some forms of neuritis the grouping of the paralysis is characteristic; that is, the extensors are principally affected. On the other hand, in myelitis, there is no tenderness of the muscles or nerves, and the pain is not so excessive. Neuritis of the sacral plexus is distinguished by the severity of the pain and tenderness of the plexus to the touch on vaginal or rectal examination. The sphincters are less likely to be affected. The history of traumatism, such as labor with or without instrumental delivery, or coexisting pelvic disease, is an important aid to diagnosis. When due to the former cause the neuritis generally involves the lumbo-sacral cord, and the paralysis is generally limited to the muscles supplied by the external peroneal nerve.

In Landry's paralysis there is an absence of sensory disturbances of all kinds, the sphincters are not involved, there is no atrophy, and all the reflexes are lost.

In meningitis there is usually muscular spasm, pain and fever are more prominent, and the sphincters are not affected.

Hysterical paralysis is liable to be mistaken for myelitis only when it is of the paraplegic type. It may be distinguished by the absence of atrophy, of changes in the electrical reactions of the muscles, and of trophic changes in the skin. Sensation is not generally affected in hysterical paraplegia, though the reverse is the case in hysterical hemiplegia. Marked changes in the reflexes are less likely to be observed, and hysterical stigmata may be found elsewhere.

Prognosis. Almost any case of myelitis must be regarded with considerable apprehension, from the point of view of recovery, though not necessarily so, so far as danger to life is concerned. The more complete the paralysis and loss of other functions of the cord the less likelihood of complete recovery, and, *vice versa*, the milder the paralysis the greater the possibility of recovery. Complete recovery is not, however, incompatible with total abolition of function of a diseased spinal segment. A tendency for the inflammation to spread up or down the cord is an unfavorable sign. Complete recovery is not infrequent, though, as a rule, some motor weakness or other disturbance of function is apt to remain. The longer the paralysis has lasted and the more extensive the inflammation longitudinally in the cord, the less the chance of recovery; but all hope of recovery should not be abandoned until a year or more has elapsed. The danger to life is greatest when the myelitis is in the cervical region, on account of inflammation extending to the respiratory centres, when death necessarily ensues. In other cases, death may occur from exhaustion, especially when bed-sores and severe cystitis are present. Disease of the kidneys, due to retention of the urine, is also an unfavorable and dangerous condition.

A favorable symptom is the early return of slight voluntary power.

Treatment. The treatment of acute myelitis is, for the most part, empirical. Individual cases vary so widely in the intensity of the inflammatory process, and consequently in the amount of the resulting necrosis, in the cord, that it is impossible to make a comparative estimate of the value of any special treatment. When such varying conditions are present it is impossible to obtain comparative statistics, or to determine with certainty the amount of benefit derived from any particular treatment. When the necrosis is extreme and the cord totally destroyed through its whole transverse extent, recovery under any treatment must be hopeless. When the inflammatory process is slight, there is a tendency toward spontaneous recovery. Between these two extremes there are all grades. The difficulty is enhanced from the fact that from the degree of paralysis and other symptoms present, we cannot always infer the amount of actual destruction present. Various remedies have been recommended, according to the theories maintained as to the nature of the necrotic process. Ergot, wet cups to the spine, and counter-irritation have been recommended on the principle that the disease is congestive in nature from the outset. They may be of value at times, but the success derived from their use has not been such as to give confidence that they affect the course of the disease. In cases clearly traceable to cold they may be of some value. When cold is the cause, the recommendation that the patient should at once be placed in a warm bath is rational, and should be tried. To be of any value it must be used early during the congestive stage. The bath should not be too hot, slightly under 90°, and the patient left in long enough to produce a decided effect on the peripheral circulation. It is recommended that the bath should be given daily for several days in succession, but it is doubtful whether any good that might be accomplished would not be neutralized, after the first day or two, by the necessary disturbance of the paralyzed patient. Spitzka has suggested that as acute myelitis frequently follows exposure of the lower extremities, and not of the back, to cold, and as the spinal cord is therefore vulnerable to influences affecting the distal distribution, that we may with plausibility affect the cord for good through the same channels. He therefore recommends the use of derivation, counter-irritation, etc., to the legs rather than to the back. He thinks he has obtained good results by this method. Both cold and hot applications to the spine have been recommended by different writers. Cold is applied in the form of Chapman's ice-bags, and if employed should be applied continuously for a number of hours at a time.

Strychnine has been highly recommended by some, while others deny that it has any effect upon the disease. In severe cases, where there is considerable necrosis, it would certainly be futile to expect it to have any favorable influence; while in mild cases it is difficult to determine how much of the recovery is due to the drug. In the last class of cases only could we expect any benefit, and if given should be administered in large doses, sufficient almost to produce its physiological effect.

But if the beneficial effect of drugs is problematical, we can at least do much to prevent the disease being aggravated by injudicious care. The patient should be at once put to bed, and not be allowed to sit up or walk about, even if he retains sufficient muscular power to do so. Physiological and physical rest is to be absolutely enjoined, and the patient not subjected to unnecessary movement. It has been urged that the back should not be the lowest part of the body, and, therefore, that the patient should either lie on his side or in the prone position. Whether the results obtained by keeping the body in these postures are sufficient to offset the discomfort to the patient is a question. Lying on the side is the more easily accomplished, and much assistance may be derived by a board supporting the back. In cases due to syphilis iodide of potash should be given, although it is true that in a minority only of them has it a specific effect. When a gumma is present it is of value, as it is elsewhere. It should be given in increasingly large doses until one or two drachms are taken three times a day. Under these heroic doses the poisonous effects should, of course, be carefully watched for.

When the myelitis is due to caries of the spine, extension should be applied according to surgical principles. Under this treatment even extreme cord symptoms usually subside and excellent results are obtained. Guttmann reports two cases of pressure myelitis from caries treated by suspension with great relief and improvement. Extension and rest in bed, however, are preferable.

The necessity of taking early measures to prevent the formation of bed-sores and cystitis cannot be insisted upon too strongly. By neglecting precautions to this end until these complications have developed, the physician will only increase his own care, as well as the suffering of his patient. Bed-sores and cystitis are often the cause of a fatal issue. The portions of the body on which pressure comes should be kept scrupulously clean and guarded as much as possible from pressure. The skin should be carefully watched for the first sign of necrosis, and daily washed. Bathing with alcohol and water is useful. When bed-sores have developed, they should be thoroughly and systematically treated, according to surgical principles. Antiseptics should be employed with a view to both healing and the prevention of septicæmia. Stimulant lotions, such as myrrh-wash and iodoform, are often of value. Hammond's galvanic plates deserve a trial. A water-bed is one of the best appliances; it should be covered with a sheet. The paralyzed parts should be kept warm, but care should be taken that a hot-water bag and bottles should not be at too high a temperature, as a degree of heat which would be harmless to a sound skin will sometimes produce a slough. I have known severe sloughs to be caused unwittingly in this way.

Retention of urine requires equal care. The urine should be drawn regularly with a catheter, extra care being taken to see that the instrument is thoroughly aseptic. When constant incontinence is present a urinal may be employed with males, although care must be used, as the friction of the vessel sometimes causes sloughs. Gowers recommends the use of large pads of absorbent cotton or oakum instead of a urinal. The wool or cotton should be changed as often as saturated. If cystitis occurs, it must be rigidly treated on surgical principles.

In the later periods of the disease the patient's general strength and nutrition should be built up with tonics, nutritious food, etc. At this period of the disease, when repair is going on, the return of sensation is often hastened by the judicious stimulation of the sensory nerves by a gentle faradic current. The condition of the muscles, when soft and flabby, may be improved by massage and electrical stimulation.

CHRONIC MYELITIS. Chronic myelitis may be the sequel of a previous acute process, which, instead of subsiding, takes on a chronic course, or it may originate in the chronic form. The first type may for convenience be termed secondary, and the latter primary. Like acute myelitis, it may be transverse, disseminated, or more or less diffused. The transverse is the most common form.

Antomically chronic myelitis differs from the acute process in the greater prominence of the growth of interstitial tissue, the absence of much vascular change, and the comparatively slight amount of softening of the substance that takes place, at least in that form which is chronic from the beginning. In consequence of the development of interstitial tissue the disease is sometimes described as sclerosis of the cord, and resembles in its anatomical appearance the system scleroses, with the difference that, instead of the process being limited to certain tracts in the cord, it invades the whole substance, spreading from a primary focus to the adjoining structures.

As a primary disease chronic myelitis is as comparatively rare as the system scleroses are common. During an experience of over ten years in the clinic for nervous diseases at the Boston City Hospital I can recall but few cases of the primary form which could be properly diagnosed as chronic myelitis.

Etiology. The most common causes are a previously acute myelitis, compression (caries, tumor, etc.), hemorrhage, and a traumatism which has caused a bruising of the cord or membranes. As a primary disease it may be due to syphilis, which induces sclerosis of the vessels and interstitial inflammation.

Repeated exposure to cold is generally acknowledged to be an effective cause. The disease may be excited by the extension of inflammation from adjoining structures; hence, it may follow chronic meningitis. Under such conditions the inflammatory process extends into the cord, along the processes of the pia mater, after which the myelitis may take on an independent course. We thus have a *meningo-myelitis*. By some, chronic myelitis is thought to be the direct sequel of an ascending neuritis, as, for example, a neuritis of the sacral plexus following injury in childbirth. Traumatism, or so-called "spinal concussion," is said to be a cause; but in such cases the myelitis is probably always secondary to a small hemorrhage within or around the cord, or to a bruising of the cord itself by temporary dislocation of the vertebræ with spontaneous reduction. Dislocations of this kind are more common than is generally supposed. Alcoholism is a common cause, and some cases have been ascribed to the poison of infectious diseases and gout (Gowers).

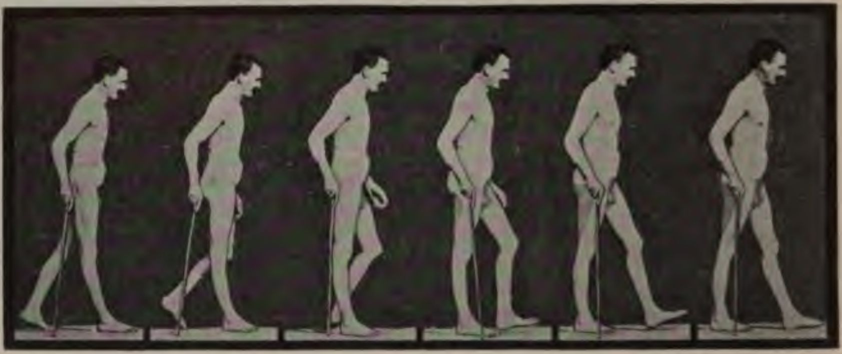
Pathological Anatomy. The morbid process consists, in the main, of a destruction of the nervous elements and a proliferation of the interstitial tissues. In the white substance the myelin of the nerve-fibre disappears, while at first the axis-cylinders persist and appear imbedded in the proliferating neuroglia tissues. The nuclei of this neuroglia are much increased in numbers, and among other varieties of cells "spider cells" are found with their long processes extending into and contributing to the formation of the connective-tissue growth. The trabeculæ of connective tissue become thickened and the normal network of interstitial tissue enormously increased in amount. This may appear as fibrillary or amorphous material. Later the axis-cylinders disappear and nothing is left of the nervous structures, but in their places is found shrunken connective tissue. The bloodvessels take part in the process; their walls become studded with nuclei and thickened, and their lumen becomes diminished and, it may be, occluded.

In the gray substance changes practically of the same kind take place, and result in the destruction and disappearance of the cells. The pia mater may take part in the process if the disease reaches the surface of the cord, or it may be the starting-point of the inflammation—meningo-myelitis. The pia is then thickened over the diseased areas and trabeculæ of the connective tissues extend into the cord from the surrounding thickened membrane. The consequences of these changes in the microscopical appearance of the cord are to give to the naked eye a gray and discolored look to the cut surface of the affected part, which is harder than normal. The cord is shrunken, sometimes not being more than one-half its normal size, and its contour misshapen. The whole thickness of the cord may be more or less involved, though the whole diameter is not affected with equal intensity, while vertically the disease may be limited to one or two segments. Secondary ascending and descending scleroses develop according to the usual laws and produce their characteristic symptoms.

Symptoms. The symptoms of chronic myelitis are similar in their grouping to those of the subacute or acute form. They differ principally in the fact that they are slower in their development, and therefore a much longer time elapses before the disease has attained its typical development, and the affection may in the early stages bear more or less resemblance to other spinal diseases.

Instead of a rapidly developing paralysis which compels the patient to take to his bed, he is able to go about for a long time pursuing his ordinary vocation, and complaining only of weakness and heavy feelings in the affected limbs. He notices that his legs feel heavy and are easily tired, and this may exist for months, or even years, before the paresis has developed to so great an extent that he cannot get about. (See Fig. 172.) Sensory disturbances are also complained of, but they are not, as a rule, so marked as the loss of power. There may be nagging pains, not sufficiently intense to be a source of great suffering or to call for opiates, but sufficient to be annoying and a source of discomfort. Various forms of paræsthesiæ also occur in the form of numbness, pin-and-needle feelings, etc. Later, loss of sensation develops, which may at first be confined to limited areas of the limbs and body. The intensity of the anæsthesia varies, but it is rarely as great as in the acute form, in which it is as prominent as the accompanying paresis. When the myelitis extends forward into the anterior cornua, atrophy of the muscles is added to the symptom group. This atrophy is usually irregular in its distribution, and is likely to be limited to certain groups of muscles arising from one or two segments of the cord into which the myelitis has extended. The reflexes passing through the cord below the lesion are increased, while those subserved by the diseased area are diminished or lost. Some loss of control over the bladder and bowels is likely to occur; the patient may experience a difficulty in retaining the urine or completely emptying the bladder. Some ataxy may be present from damage to the posterior columns. Sooner or later there are grouped together all the symptoms that point to the involvement of the different structures of the cord, motor, sensory, trophic, etc., and which indicate that the disease is not limited to any one system, as in the other chronic degenerative diseases. As the disease is slow in its development, it is evident that for a time the inflammatory process will be limited to certain parts of the cord, and thus the disease may temporarily simulate the different system diseases, especially primary spastic paralysis and chronic poliomyelitis. When the pyramidal tracts are principally implicated there will be marks of spastic paralysis, with the usual exaggeration of the reflexes, etc.; but a careful examination will show that the sensory and other elements are more or less affected, and the later progress of the disease will make it clear that the cord is transversely affected. In the disseminated form, where there are a number of diseased foci scattered through the cord, all possible combinations of symptoms may be present, and when the symptoms of the different foci fuse together they may present the picture of a transverse lesion. When atrophy of the muscles is present, electrical changes will be found, usually a simple diminution of contractility to both faradic and galvanic currents. Some qualitative changes may sometimes be found, or with the loss of faradism galvanic contractility may be maintained or diminished.

FIG. 172.



Gait in a case of chronic myelitis. From instantaneous serial photographs of a patient of Dr. Dercum, by Muybridge.

For a further description of the symptoms the reader is referred to the account of acute and subacute myelitis, bearing in mind that the chief difference is the more gradual development of the symptoms.

Erb has described, under the name "syphilitic spinal paralysis," a group of symptoms which he considers pathognomonic of myelitis when due to syphilis. For a description of this affection the reader is referred to the section on Spinal Syphilis in Chapter XXIV.

Course and Termination of the Disease. As has been said, the course of chronic myelitis is usually slow, extending over many years, but it is generally progressive, though there may be stationary periods. It may cease in its progress at any period. The periods of cessation may be followed by periods of comparatively rapid increase of the symptoms. It may remain stationary for a long period of time. Death may take place from involvement of the medulla, in rare cases from bed-sores, more commonly from intercurrent disease.

Prognosis. It must always be difficult to forecast the future in any individual case, at least without watching its progress for some little time. In the great majority of cases, so far as complete recovery is concerned, the prognosis must be unfavorable. After sclerosis has been established the impossibility of removing it is apparent, but there is always a possibility of the inflammatory process stopping at any time, even if we cannot say of being arrested by treatment, and when this has taken place it is possible that there may be an amelioration of the symptoms and considerable restitution of the lost functions. We can easily understand how this can occur by supposing restoration of the myelin in the fibres which still retain their axis-cylinders, and a regeneration of the axis-cylinders which have undergone only the first stages of atrophy. It may also be assumed that nerve-cells that are only degenerated, not destroyed, may recover their normal condition, but such nerve structures as are absolutely destroyed and have their places replaced by hypertrophied neuroglia tissue are lost forever. It is possible also that the functions of the destroyed fibres may be more or less carried on by other tracts, and in this way a certain amount of restitution of the functions be obtained. We know this obtains in some cases of posterior sclerosis, in which there has been a disappearance of ataxy and other symptoms, although the autopsy has shown the persistence of the sclerosis of the posterior columns.

The disease is not necessarily progressive, and long stationary periods occur in which little or no change can be discovered in the symptoms. It may lead to death in the course of years, varying in individual cases from three to twenty (Spitzka). When the disease is comparatively intense in its development, even though it is of limited extent, the prognosis is worse than when the disease is more extensive in its distribution and mild in its course. It is also more dangerous to life when it is located in the lumbar enlargement, where it is more likely to occasion bed-sores, cystitis, etc.

When in the upper cervical enlargement there is danger to life from involvement of the respiratory centres; and in any case when the lesion has become transverse, or approximately so, there is always danger from the exhaustion which usually, sooner or later, supervenes in such cases.

In cases due to syphilis (Erb's type) there is a tendency toward improvement under specific treatment.

Diagnosis. The principal and more common diseases from which chronic myelitis must be differentiated are spastic paralysis, chronic poliomyelitis, progressive muscular atrophy, compression from caries or tumor. It is only in the early stages that the disease is likely to be mistaken for primary spastic paralysis. Sensory, atrophic, and other symptoms indicative of involvement of other parts of the cord than the pyramidal tracts will clear up the diagnosis. When the chief incidence of the inflammatory process falls upon the anterior cornua the disease may resemble chronic poliomyelitis, which by some is considered a special variety only of chronic myelitis, while others consider it a distinct affection. The atrophy in chronic myelitis is more irregular in distribution and associated with sensory and other symptoms. The development of paralysis in parts below the level of the cord lesion, with spastic symptoms showing the involvement of the pyramidal tracts, will be a sufficient indication that the disease is not limited to the anterior cornua.

Progressive muscular atrophy is more likely to be confounded with the disease under consideration, but the indication of damage to other structures in the cord, such as pain, loss of sensation, and paresis of the sphincters, will sooner or later afford a sufficient basis for distinction.

In compression from caries the presence of spinal deformity will usually be sufficient, though with caries there is apt to be some myelitis, which takes on a chronic course. With tumors the pain is much more intense, and in a large proportion of cases there is marked spasm or rigidity of the muscles. These symptoms of irritation are apt to precede those indicative of damage.

At times symptoms similar to those of myelitis may be caused by chronic pachymeningitis and leptomeningitis and ataxic paraplegia. The reader is referred to the description of those diseases for the purpose of diagnosis.

Treatment. We may set before ourselves the fact that we cannot remove the interstitial tissue that has been formed, nor restore nerve structures that have been destroyed, but the possibility of arresting the disease and thereby saving such neural structures as have not been destroyed, and giving an opportunity to those only partially disabled to recover their normal condition, is one that should always be entertained. At the outset it is desirable to search for such causes as seem likely to have in any individual case incited the disease, and may be still acting, and to remove the patient from their influence, even though we may not have scientific certainty that they are the etiological agents. In a disease of such gravity, and in which the consequences are so disabling, it is not wise to neglect every precaution in the absence of absolute certainty of the cause. If the patient continues to be constantly exposed to cold and the hardships of weather, he should be warned against the danger, and a change of occupation or habits insisted upon. Complete and absolute rest in bed at first is desirable for a while, but in a disease of such long duration it is not practicable or desirable to continue it beyond a few weeks. Overuse of the damaged nerve structures should be avoided, and, therefore, the patient should be cautioned against overexertion and physical fatigue. The daily use of warm baths is recommended by some, with the view of acting upon the peripheral nerves and circulation and thereby indirectly affecting the circulation and nutrition of the cord. In other words, it is thought that warm baths have an effect contrary to a cold one. They are certainly agreeable to the patient, seem to be palliative and remove uncomfortable symptoms, and therefore seem to benefit. The temperature should not be too high, as extremes of heat and cold in many respects seem to have the same physiological reflex effects on the circulation and nervous centres.

When possible it may be desirable to send the patient in the winter to a warm climate. It is generally considered that a cold climate has a deleterious effect on the disease and a warm one a favorable influence. Spitzka considers every month spent in a cold climate as so much time lost. When syphilis is present or there is reasonable grounds to suspect its existence, antisyphilitic treatment should be energetically pushed, for, besides the possibilities of cure, if syphilis can excite the morbid process, it is possible that the same influence may prevent its subsiding. We have not a sufficient knowledge of the mode in which syphilis induces myelitis to justify us in neglecting any precaution, nor can we always be certain that some gummatous growth may not be present. As was said above, some cases of Erb's type are cured and many are improved by specific treatment. (See also Chapter XXIV.)

The mixed treatment should be given and the iodides should be pushed in large doses at first; later they should be diminished and given in smaller doses during a long period. The individual symptoms should be treated as in other destructive diseases of the cord. Blisters and the cautery are often useful for the relief of pain when this is present in a sufficiently intense form to be annoying. One of the best modes of relieving pain is the electric battery. Faradism, galvanism, or static electricity may be used; sometimes one is more palliative and sometimes another. The application should be continuously repeated, either daily or three times a week. It must not be expected that electricity will directly affect the cord, but it is of great value in keeping the patient comfortable and rendering unnecessary a resort to anodynes.

Faradism has the advantage that a battery can be placed in the patient's hands and can be used daily at home. Careful instructions should be given as to its use, otherwise it is likely that it will be very inefficiently applied. The best method for the relief of pain is to brush large surfaces of the skin, particularly the areas supplied by the nerves rising from the seat of the lesion, using a moderate current. When bed-sores are present they should be looked after with the greatest care, the usual surgical means being employed for this purpose. The method of Hammond, viz., using two plates, one of zinc and one of silver, connected by insulated wires, has been used with success. The silver plate is placed directly upon the bed-sore; the zinc plate is covered with a piece of flannel or absorbent cotton wool, and is placed upon the adjoining sound skin. The whole is worn continuously. This is said to give good results. See also the method of feeding bed-sores described in Chapter XXIII.

The general health of the patient must, of course, be built up by tonics, food, and a hygienic course of life. There is no drug known that has any specific influence upon the disease and the use of drugs cannot be recommended. They have the disadvantage of diverting the mind of both physician and patient from the importance of other measures effecting the daily life of the patient such as have been described, and thus indirectly lead to procrastination and neglect. If paralysis of the bladder exists, the importance of guarding against the dangers of retention and cystitis cannot be too urgently insisted upon.

THE CAISSON DISEASE, OR DIVER'S PARALYSIS.

Persons who have been exposed to high atmospheric pressure in a caisson or diver's apparatus are liable, on returning to the outer air, to be attacked with paralysis (generally paraplegia) and other nervous symptoms. The symptoms are attributable to the sudden change of atmospheric pressure, for if precautions are taken to make the change of pressure sufficiently gradual they may be avoided. The increased pressure must be, as a rule, over one atmosphere, for when it is less than this the symptoms rarely ensue. They come on shortly after returning to the outer air, when they may immediately ensue or they may be delayed for an hour. They have occurred in the lock of a caisson where the atmospheric pressure is gradually lowered. The symptoms are both cerebral and spinal. The former consist of feelings of faintness, prostration, headache, nausea, vomiting, giddiness, double vision, incoherence of speech, and even coma, any one or all of which may precede or accompany the paralysis, which, in the severer cases, is the most obtrusive symptom. Vomiting is frequently prominent.

Neuralgic pains are present in the majority of cases and are generally one of the earliest symptoms. These pains are of all degrees of severity, being sometimes mild and sometimes so excruciating as to demand large doses of morphine; they usually occur in paroxysms, with intervals of remission. They may be "tearing" in character. They are generally felt in the legs, but may be located in the arms, back, or any part of the body. Epigastric pain is common. These pains may still continue, even though complete anæsthesia is present in the painful parts—*anæsthesia dolorosa*. The large joints, especially the knees, are frequently the seat of pain. It is interesting to notice that the pains have much similarity with the irritative root or cord pains which occur with tumors of the cord, and are probably likewise due to irritation of the sensory cord fibres.

In mild cases the symptoms may be limited to these pains, with nausea, vomiting, giddiness, or one or more of the symptoms just mentioned.

In severe cases, shortly after the onset of the pain, the patient notices that his legs feel heavy and weak; this weakness increases and may develop into a complete paraplegia. Occasionally all four extremities are paralyzed, and in rare cases there may be a hemiplegia. With the development of paraplegia sensation is diminished or lost in the legs. In the more severe cases the sphincters are affected and there are a retention of urine and constipation. In other cases the onset is marked by a sudden but temporary loss of consciousness, lasting about fifteen minutes, and followed by the above paralytic symptoms. Hemorrhage from the mucous membranes has been sometimes noted.

The cases vary in severity from mild weakness, prostration, with one or more of the other symptoms just mentioned, up to absolute paralysis and impairment of the functions of the cord.

In cases of more violent onset the patient may fall down unconscious soon after emerging into the outer air, and death may take place in a few hours without recovery of consciousness. In one case, for example, a man came out from a caisson and washed himself, and then fell down and died in fifteen minutes.[1]

In another instance, a man complained of not feeling well about one hour after coming out of the caisson and went to his lodging-house a few rods distant. As he passed through the lower story of the house on his way to his own room, which was on the story above, he complained of pain in the abdomen. While ascending the stairs he sank down insensible, and was dead before he could be laid on his bed.[2]

In these apoplectiform cases there may be delirium and convulsions. A man who rapidly became unconscious developed a muttering delirium, but no paralysis, and died the next day.[3]

The majority of cases recover. In mild cases, and even in cases where there has been temporary loss of consciousness, succeeded by paralysis, with loss of control over the sphincters, etc., recovery may take place in a few days or even hours. The course of the symptoms and the mortality vary much according to the conditions of exposure. Of 110 Brooklyn bridge cases which were of sufficient severity to come under treatment three proved fatal. At St. Louis, out of a total of 352 men employed, 30 were seriously affected and 12 died. Paralysis occurred in 61 per cent. of the St. Louis cases (47 out of 77 cases observed by Jaminet), but in only 15 per cent. of the New York cases.

In the severer cases the disease is more protracted, and if recovery does not occur within the first six or eight days typical symptoms of a dorsal or lumbar myelitis develop, and the case pursues the usual course of that disease. In such cases, in addition to the loss of power and sensation, bed-sores, cystitis, pyelitis, muscular wasting, etc., may occur. In some cases bloody urine has been noted. In the fatal cases, when death does not take place within a few hours, the symptoms deepen and death occurs after several days or it may be after weeks (eight or ten) from myelitis. When recovery occurs it may be complete or only partial.

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[1] Pol, B., et Wattelle, T. I. I: Ann. d'Hyg. Pub. et Méd. Légale, Paris, 1854, 2 S., t. i. pp. 241-279. Quoted by Van Rensselaer.

[2] A. H Smith: The Physiological, Pathological, and Therapeutic Effects of Compressed Air, 1886.

[3] E. A. Clark: Med. Archives, St. Louis, 1870-71, v. pp. 130-295-300. Quoted by Van Rensselaer.

Etiology. While the exciting cause of the disease is exposure to high atmospheric pressure, there are several conditions which may be regarded as predisposing causes. There seems to be a predisposition by which certain persons are more likely to be affected than others. This is only another way of saying that the vascular system of one person has a greater capacity to accommodate itself to altered conditions than that of another.

Alcoholism, heart and kidney disease, old age, in fact anything tending to lower the vitality of the body, predisposes to the affection. It is generally thought that fulness of habit and obesity are predisposing causes. Smith states[1] "that during the progress of the work on the East River Bridge, there was among those taken sick a remarkable preponderance of men of heavy build with a tendency to corpulency. Of thirty-five men of this build only three escaped sickness, while of fifty-three lank and spare men twenty-five men escaped." The mode in which obesity acts will be stated in the discussion on pathology.

One of the most potent factors is inexperience in the work. In the report of the construction of the great St. Louis Bridge, Captain Eads states that nearly all the deaths occurred among the inexperienced workmen. Several of the fatal cases were men who had worked but one watch of two hours. Those who had begun to work with the commencement of the operation, and thus had become accustomed to the increasing pressure at lower depths, were unaffected throughout, and no cases of importance occurred after the watches were reduced to one hour, although the work was finally carried on at a depth of ninety feet. In the Wyoming (Pennsylvania) cases (reported by Charles P. Knapp) 10 per cent. of the old hands and 35 per cent. of the new were affected.

Severe muscular exertion and chilling of the body after leaving the lock are thought also to be predisposing factors. Jaminet considers that entering the caisson fasting has a similar influence.

Pathology.[2] In considering the patholoy of this affection, it is necessary to take certain facts into consideration. The symptoms develop, not while the individual is exposed to the high pressure, but only after returning to the normal atmosphere; other things being equal, the longer a person has been exposed to a high pressure and the greater the pressure the greater the danger. In practical works it is customary to take advantage of this and to limit the length of time during which the workmen are exposed. The danger may also be lessened or averted by gradually diminishing the pressure before leaving the caisson. For this purpose locks are provided. Constant exposure protects, and workmen become acclimated, so to speak, within certain limits. Inexperienced persons are more liable to be attacked than others.

It has also been found that anything that lowers the vitality of the body, such as alcohol and old age, predisposes a person to the disease. Obesity likewise seems to have this effect.

Many theories have been proposed regarding the nature of the affection; some of them are very fanciful. It is only necessary to consider two of them at this time, viz.: the gaseous theory and the theory of congestion with subsequent stasis.

According to the gaseous theory the blood under the high atmospheric pressure becomes supercharged with oxygen, which is retained in the blood so long as the high pressure continues. When the pressure on the body is relieved there is a tendency for the oxygen to escape from the bloodvessels, much as it tends to escape from a bottle of aërated water after the cork has been drawn. The lungs cannot remove the excess of gas sufficiently fast to relieve the internal pressure, consequently the gas escapes from the blood and is carried along in the vessels as bubbles, or escapes from the walls of the capillaries into the surrounding tissues. In the soft parts this escape can probably take place into the perivascular spaces and surrounding tissue without causing damage of consequence, or at least of such a nature as to produce marked symptoms; but in an organ like the spinal cord it is presumed that the bubbles of gas escaping from the blood would act as emboli, obstructing the capillaries, while the air that escaped from the latter would exert considerable pressure upon the nerve elements. These conditions would tend to interfere with the functions of the cord. In extreme cases it would seem probable that more or less structural damage might be caused and a myelitis set up. As the gas is gradually expired from the lungs and the pressure in the vessels diminished there would be a tendency to resorption of the gas from the tissues, and removal of the emboli by the current of the circulation. With the removal of the gas a recovery of the spinal functions would ensue.

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[1] Pepper's System of Medicine, vol. iii. p. 187.

[2] The most complete discussion on the pathology of the affection is by Van Rensselaer "The Pathology of the Caisson Disease," a prize essay. Philadelphia, 1891. In this paper will be found a complete bibliography with the post-mortem reports in nearly all the fatal cases. A free use of this essay has been made in this account of the affection.

Aside from theoretrical considerations, there is some evidence in support of this theory in the main. In the first place, in some of the autopsies that have been made gas has been observed escaping from the tissues and in the vessels. Numerous experiments have been made upon animals, which have apparently shown the presence of gas in the tissues. The most valuable of these are those of Catsaris, of Athens, who experimented by shutting dogs up in a diver's apparatus and letting them down to various depths in the sea. The animals developed the usual symptoms. The dogs were then killed, and Catsaris observed, with the aid of a magnifying glass, bubbles of gas in the vessels and in the tissue itself in the lumbar region of the cord. In the case of one animal which was killed on the second day after the paralysis was complete, there was found advanced softening of the cord in the lumbar region. The lateral and posterior columns were colored a yellowish-gray. A little below this region the gray matter was red; above, the gray matter was red, but without softening; there was said to be no congestion. Bubbles of gas could be seen in the middle of the soft portion and escaping from the vessels surrounding this part.

The theory of congestion followed by stasis, proposed by Smith, supposes that under the high pressure the blood is driven from the periphery into the internal organs, and particularly the cord and brain, which being enclosed in a hermetically sealed and resifting bony canal, are not directly subjected to the increased atmospheric pressure. At the same time the bloodvessels of the cerebro-spinal system, receiving no support from the counter-pressure which is given to them in the other organs of the body, will be abnormally dilated. Furthermore, a greater quantity of blood would probably be forced into the cerebro-spinal vessels by the force of the heart's action alone, as the blood would seek the easiest channels, which would be those vessels which are not compressed by the external atmospheric pressure. By the overdistention thus produced it is presumed that the walls of the vessel are more or less paralyzed. Thus far, however, the hyperæmia is purely an active one, and the blood flowing without impediment, no symptoms are produced. When, however, the external atmospheric pressure is removed, the theory supposes another condition of affairs to follow. The blood rushes to the periphery of the body through channels which had been depressed and largely deprived of their blood. Within the spinal cord the blood pressure would in consequence be relieved, and the circulation would be retarded. The walls of the vessels being partially paralyzed from overdistention could not contract, and thus help on the blood current. There would therefore be a comparative stasis of the circulation of the brain and cord.

Nixon and Birmingham have pointed out that the anatomical arrangement of the bloodvessels supplying the lower portion of the spinal cord are such as to render precarious the blood supply under conditions causing a sudden diminution of the blood pressure; such a diminution would probably lead to a reduction of the blood supply.

The pathological findings at autopsies have varied somewhat, and have not afforded the information that might be expected, owing to the incompleteness of most of the examinations. Thus far a post-mortem examination has been reported in twenty-nine cases,[1] but in only four of these was a microscopical examination of the cord made. Inasmuch as in protracted cases myelitis develops, the condition of the cord in such cases would not indicate the nature of the pathological process in those cases in which the symptoms are of an ephemeral nature, or which have a rapidly fatal issue. It is just these cases that represent true caisson disease, and in which the nature of the pathological process is in doubt. Unfortunately, in the four autopsies in which the cord was examined microscopically (Leyden, Schultze, and Van Rensselaer, Sharples), the subjects lived sufficiently long to allow of the development of a myelitis, viz.: two weeks, two and a half months, five weeks and nine weeks, respectively. In the apoplectic form, or rapidly fatal cases, which would afford the most light on the subject, the post-mortem examinations have been incomplete. Studying the reports as given, however, we find that out of twenty-nine cases death took place within forty-eight hours in ten. In none of these was the cord examined microscopically, and in only four of the ten cases was a macroscopical examination made, but in all four cases it was found congested. In one case there was an extensive effusion of blood pressing on the cord in the lower dorsal region, and in another clots of blood were found on different parts of the dura. The brain, or meninges, or both, were reported as congested in five. In six some one or all of the internal organs were reported as congested (in one no report); an extravasation of blood in the kidneys was reported in two cases.[2]

An effusion of serum under the arachnoid and dura of the cord (?) was found in one case. Subcutaneous ephysema was observed in two cases. It will thus be observed that the cord was found congested in all of the cases where it was examined. In many of the cases that pursued a chronic course softening of the cord was found, as might be expected. Taking all the cases (twenty-eight), the acute and chronic, it may be said in general that the most constant appearances are congestion of the spinal cord and (generally) brain and internal organs in the acute cases, and softening of the cord with similar congestion of the internal organs in the more protracted cases. The effusion of serum in the spinal canal in five cases is worth noting, as well as the extravasation of blood in the dura of the cord in four.

From all these considerations, it seems very probable that the pathological conditions underlying the earlier symptoms in caisson disease are some kind of disturbances of the circulation; that congestion is present would seem to be shown by the results of the autopsies.

The mechanism by which this congestion brings about the functional disturbances is not quite so plain. It is evident that the primary *active* hyperæmia is not the exciting factor, inasmuch as, if the theory of Smith be correct, the hyperæmia of the cord is most active while the subjects are exposed to the high barometric pressure, whereas the symptoms do not develop until after the pressure has ceased to exist.[1] The exciting etiological factor must, therefore, be the supposed subsequent stasis of the blood, though of the occurrence of this we have no direct evidence, though it may be theoretically probable. On the contrary, it is possible that the sudden diversion of the blood from the cerebro-spinal system to the periphery might induce an anæmia of the cord, instead of a passive engorgement. Such an anæmia has been shown[2] to follow compression of the abdominal aorta and vena cava, and to cause a sudden paraplegia; such an anæmia, if long continued, leads to myelitis. It is very likely that a stasis and anæmia are practically the same in their results, excepting that a stasis would be more likely to cause an œdema of the tissues, which may be the chief pathological factor. The fact that serous effusion into the membrane has been found in some cases would lend support to this view. It is readily supposable that if the œdema were absorbed the symptoms would disappear, but that when it is excessive, in conjunction with the impaired circulation, it would lead to changes in nutrition and softening of the cord.

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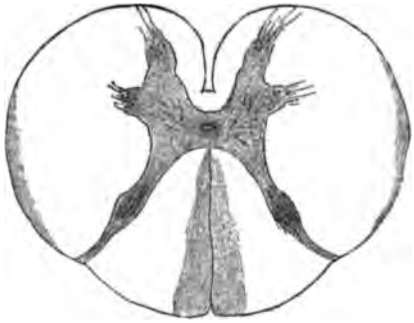
[1] Van Rensselaer's collection (1891) includes twenty-eight cases. More lately Sharples (Journ. of Nerv. and Ment. Dis., 1894, xix. No. 10, p. 636) has reported an autopsy with microscopical examination.

[2] This explains the bloody urine sometimes seen.

The other pathological facts which were mentioned above are more in harmony with the view that involves changes in the circulation than with the gaseous theory. The fact that repeated exposure protects, that the longer the exposure and the more sudden the withdrawal of the increased atmospheric pressure, and that obesity, alcoholism, and debility, heart and kidney diseases favor the development of the symptoms, are all in accord with the former theory, while these phenomena cannot be so well explained on the theory of the liberation of gas in the blood.

FIG. 173.



Figs. 173–178, sections of cord from Van Rensselaer's case, showing myelitis in dorsal region with ascending and descending degeneration. Gray substance normal. Fig. 173, section at level of II. Cervical segment.

In fat people the amount of blood in the periphery is greater, and, therefore, there is a greater amount of blood to be driven into the interior, while on relief of pressure the peripheral channels through which the blood may flow, are more numerous, and, therefore, stasis (or anæmia) would be more marked.

In the light, however, of the experiments of Catsaris and others, as well as the frequent findings of emphysema in the tissues of the blood and the air in the large vessels, in the recorded autopsies, it is impossible to exclude gaseous emboli as factors, at least, in the pathological processes. Such emboli would certainly tend to increase the œdema and stasis. Before reaching a final determination of the exact nature of the pathology of the disease, it will be necessary to have before us the results of microscopical examinations of the cord in the rapidly fatal cases. These have not yet been made.

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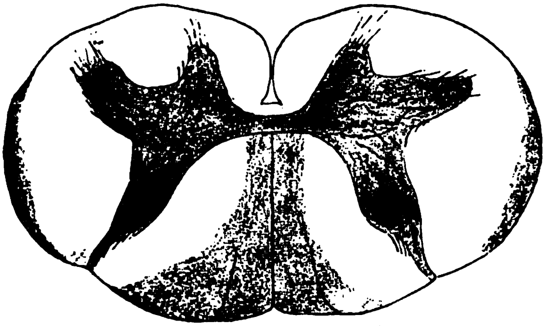
[1] Moxon (Lancet, 1881, i. p. 528) has pointed out the fallacy of the traditional belief that increased arterial blood supply without venous obstruction can cause nervous symptoms.

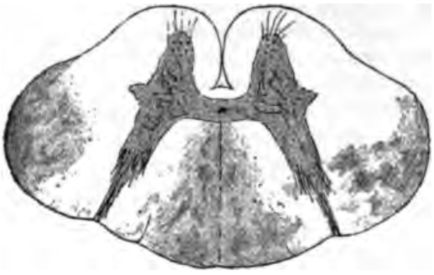
[2] Herter: "A Study of Experimental Myelitis," Journal of Mental and Nervous Diseases, April, 1889.

FIG. 174.



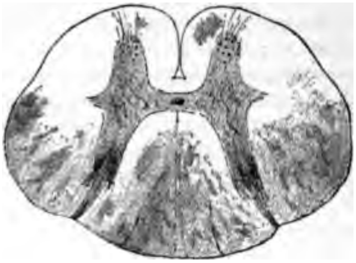
VIII. Cervical.

FIG. 175.



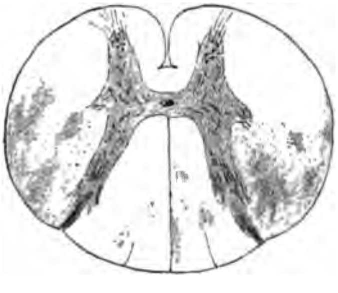
III. Dorsal.

FIG. 176.



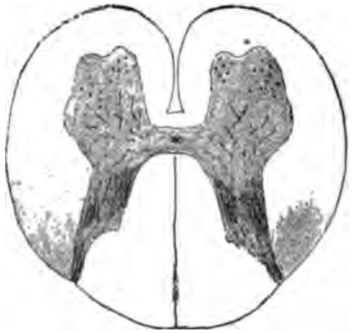
VII. Dorsal.

FIG. 177.



XII. Dorsal.

FIG. 178.



III. Lumbar.

In the three cases of Leyden, Schulze and Van Rensselaer, in which a microscopical examination of the cord was made, a diffusive parenchymatous myelitis was found, leading to degenerative changes.

The greatest amount of degeneration was found in the posterior columns and adjacent portions of the lateral columns. This would seem to account for the prominence of pain in most of the cases. The anterior columns were but slightly affected, the gray matter was "for the most part normal; even in the most intensely degenerated portion there is not total destruction of all the nerve substance. Above and below this area of this disease, in the lower dorsal region, are respectively the usual ascending degenerations of the columns of Goll, and the direct cerebellar tracts, and descending degenerations of the pyramidal tract. In the substance of the cord no evidence of hemorrhage could be found."[1] In Sharples' case practically the same changes were found, the disease being for the most part limited to postero-lateral white columns. It will thus be seen that the condition of the cord in the chronic cases corresponds with the symptoms, namely, a myelitis.

Treatment. Prophylaxis is extremely important for persons working in a caisson. No one should enter a caisson when sick. All workmen, before being employed, should be obliged to submit to a physical examination, and any found suffering from serious disease, especially kidney[2] or heart disease, should be rejected. Persons known to be addicted to or under the influence of alcohol should be refused employment. Very fat persons and those of advanced age should enter the caisson with caution. Inexperienced persons should in the beginning limit their first exposure to the shortest possible time practicable—one hour if the pressure is at all high—and pass a liberal time in the lock where the pressure is gradually reduced. The length of time during which workmen and others are daily exposed should be rigidly regulated. Collingswood's rule is that the length of time should vary inversely as the pressure; that is, if twelve hours be an ordinary day's work under normal pressure, under two atmospheres (or fifteen pounds additional pressure), it should be six hours; three atmospheres, four; four atmospheres, three hours.[3] While in the lock, where, in consequence of the lowering of the atmospheric pressure, the temperature is sometimes reduced to 30° and upward, great care should be taken against chilling the body by putting on warm clothes. It is difficult to induce workmen in a body to take these precautions, but individuals will do so.

Smith recommends that about five minutes should be occupied in the lock for each atmosphere; if practicable, even more time than this should be given for inexperienced persons.

At St. Louis those who commenced with the works when the pressure was slight were unaffected throughout. This acclimation of persons to high atmospheric pressure has been confirmed by general experience. It is, therefore, desirable that when works of magnitude are to be undertaken measures should be adopted which will secure the continuous employment of the same men throughout the work.[4]

Jaminet's view, that entering a caisson fasting predisposes a person to being attacked, is thought by Smith to be corroborated by some of his cases.

It is not always well to wait for a scientific demonstration before adopting a special mode of treatment, which, *per se*, would be harmless, but to act upon presumptive evidence; therefore, while waiting further evidence on this point, it would be well to enforce a rule, as has been done, that no one should enter a caisson while fasting.

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[1] Van Rensselaer.

[2] In one case a man with advanced Bright's disease was found unconscious in the lock. After regaining consciousness convulsions set in, and he died.

[3] While building the St. Louis Bridge it was found necessary to reduce the length of time of each shift to one hour at a depth of ninety feet. At New York a day's work was finally reduced to four hours, divided into two shifts, separated by a four hours' interval.

[4] Smith's important monograph contains valuable suggestions regarding the management of works, and should be consulted by those who have the supervision and construction of such undertakings.

After the symptoms have set in it has been recommended that a person should be returned to the caisson. Although this has been done with success it is not always practicable, and probably only in a few cases could be done within the first few hours, when it would be likely to be serviceable. More practicable would be putting the patient into a pneumatic cabinet, where he would be subjected to increased atmospheric pressure.

In large cities it would not be difficult to obtain the use of such a cabinet, and it would not be impracticable for contractors and others to have such a cabinet in readiness in case of accident during the progress of the work.

When large works are contemplated a temporary hospital, conveniently located, should be provided, containing a chamber or cabinet, in which patients could be again subjected to high pressure. This might be a proper subject for legislation.

For the intense pain sometimes observed, recourse to large doses of morphine must be had. Morphine also controls the vomiting.

Smith states that ergot, though not always successful, was very useful in a considerable number of cases. He says, for very severe pain, complete relief has been experienced within half an hour after the administration of a drachm of the fluid extract. Unsteadiness of the limbs also yielded promptly to one or two doses. This effect of ergot is confirmed by the experience of Knapp in the Wyoming cases. The drug should be given liberally—one drachm every hour or every two hours for several doses.

Various other measures have been recommended, such as cups, douches, electricity, etc., but we have no positive evidence that we can favorably influence the spinal circulation in this way.

It may be suggested that in the way of experiment it might be well to try bandaging tightly the limbs, perhaps the whole body, with an Esmarch or some other form of bandage, with a view to imitating the action of increased atmospheric pressure and driving the blood once more into the spinal cord.

Later, after the symptoms of myelitis have developed, the principles of treatment are the same as when that disease occurs under other circumstances.

ACUTE ASCENDING PARALYSIS (Landry's Paralysis).

This disease, which was first described by Landry in 1859, derives its name from the fact that in a large majority of cases it is characterized by a rapidly developing motor paralysis, which involves first the legs, and then, in close succession, the trunk, arms, and finally many of the muscular regions supplied by the medulla. Some authors describe also a so-called bulbar form, in which the last-mentioned regions are the first to be attacked.

In both forms death is the usual termination, if we are to judge by reported cases.

As a rule, there is little or no disturbance of sensation, no atrophy, no alteration in the electrical excitability of nerves and muscles, and the sphincters are rarely involved. The disease is not common.

Etiology. The predominating etiological factor in most of the reported cases is exposure to cold and wet, certain of these having the additional element of alcoholism. In other cases the victims were suffering at the time of onset from the weakening effects of some preceding infectious disease, such as smallpox, diphtheria, or typhoid. It has also been known to follow febrile diseases of obscure nature at an interval of one or two weeks, and has occurred after trauma when the wound has apparently healed.

Gowers has seen it follow an attack of pelvic cellulitis. I have seen one case apparently caused by some sort of septicæmic process, as it followed a false passage in the urethra made by an attempt to introduce a catheter. It has been observed frequently, too, in the subjects of syphilis, and has apparently been arrested by specific treatment. The form of rabies known as "paralytic rabies" causes a disease apparently identical with Landry's paralysis.

Males are affected with greater frequency than females, and the period of life at which the disease commonly occurs is between the ages of twenty and forty-five years, but it has been observed in young children and in old people.

Symptoms. Some cases present such premonitory symptoms as general malaise, dragging pains in head and back, and tingling in the extremities for some days, or, rarely, some weeks.

The two cardinal symptoms, and, in fact, almost sole symptoms of typical cases, are paralysis and loss of all reflexes. The contrast of profound and widespread paralysis, with little or no impairment of other functions of the cord or nerves, makes a picture which is characteristic. The paralysis begins in the legs, often one before the other, and usually rapidly increases in degree, until complete paraplegia results. A distinguishing peculiarity of the paraplegia is, as has been said, its ascending course. After the legs, the muscles of the trunk are successively involved—first of the pelvis, loins, and abdomen, then of the thorax. All this may occur in a few hours, so that the progressive course at first may escape notice, or two or three days may be occupied before the patient is helpless.

After the trunk the arms are affected, either both simultaneously, or, like the legs, one before the other. Whether the individual muscles of the arms and legs are successively attacked in an order corresponding to the longitudinal arrangement of the spinal centres remains to be determined; probably not, but sufficiently accurate observations have not yet been made to establish this point. The paralysis of the arms and trunk is less apt than that of the legs to be absolute. The arms may be only partially paralyzed. The final stage of the disease is marked by a still further extension upward of the paralysis, causing respiratory disturbances and symptoms of bulbar involvement. The respiration is then labored and difficult, and the diaphragmatic movements are weaker. The lips, tongue, palate, and throat become involved. In consequence, dysphagia results, and the power to articulate is impaired or lost. In certain cases partial facial paralysis and ocular muscle disturbances have been noted.

Sensation is not usually affected, although there may be a slight dulling to all forms of stimulation, especially in the extremities. In two or three cases there has been marked anæsthesia. The absence of pain is conspicuous. The reflexes, both cutaneous and tendon, are diminished or wholly lost, but in one case the knee-jerks were increased at the beginning, although abolished later on.

Although the paralyzed muscles become rapidly flaccid, they present in the large majority of cases neither wasting nor change in electrical reaction. The normal character of the reactions presented by such soft and flabby muscles is often striking. In some few otherwise typical cases a rapid loss of faradic muscular excitability and even polar changes have been noted. But it may be suspected that such cases, as well as those presenting marked anæsthesia, were really instances of multiple neuritis, certain types of which simulate Landry's paralysis, and cases of which have certainly been mistaken and wrongly reported as of this affection. Further, other apparent anomalies in the clinical picture are caused by the fact, as will presently be seen, that in cases which run a more protracted course, certain secondary changes in

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usual mode of death—in forty- rs. In other cases, again, the p
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takes place it is usually slow.

Pathology. In the majority he results of autopsies have
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been apparently typical, varie al findings have bee
It is exceedingly probable tha ry cause of the paralysis
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in the spinal cord or nerves, or b pothesis is supported by an
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the toxin of the Klebs-Lœffler bacillus upon nerve structures.

It is highly probable that the paralytic symptoms at the onset are due
the chemical affinity of the poison for the nervous motor elements, while the continuous action of the poison may cause various secondary changes in the nerves and cords, and perhaps in the muscles. This would be in harmony with the entire absence of anatomical changes, which is the rule, as well as the varied and dissimilar lesions found in other cases, such as neuritis, poliomyelitis, etc. Baumgarten, in one case, found in the spinal cord many rods like the bacilli of malignant pustule. In Curschmann's case the typhoid bacillus was found in the same region. Catanni found bacilli in the endoneural lymph-spaces of many of the peripheral nerves. Eisenlohr demonstrated various kinds of bacilli, and in one rather typical case, found staphylococci in various organs of the central nervous system. In some cases disseminated inflammatory patches or ecchymoses were found in the medulla, especially in the motor tract, and in others similar alterations in the cord. Particularly marked was the swelling of axis-cylinders in the crossed and uncrossed motor tracts. In still other cases, what was found corresponded to the lightest form of poliomyelitis. Pitres and Vaillard found nothing abnormal in the gray matter of the spinal cord, and few alterations in the spinal nerve-roots, but found the peripheral nerves degenerated to such an extent that in many fibres nothing remained but an empty sheath of Schwann. Watson found nothing but a congestion of the spinal meninges. Luigi Villa found in his case in "the peripheral nerves and in the nerve-roots the lesions of parenchymatous neuritis with preservation of the axis-cylinders, but no changes in the intra-vertebral ganglia. The axis-cylinders were surrounded by accumulations of a substance having a special morphology and micro-chemical character, which was also found in the transverse sections of the spinal cord, particularly in the periphery. The central canal

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The sphincters are rarely affected. The cerebral functions also are not usually involved, although in the rare cases, with high temperature and signs of general infection, there may be some stupor or mild delirium. In the overwhelming majority of cases there is no elevation of temperature. In a few cases a slight rise has been noted. Profuse sweatings and splenic enlargement are frequent. Irregular forms of the disease are sometimes met with, in which the arms are involved before the legs, or the diaphragm before the intercostals. When bulbar symptoms begin first, death may occur by involvement of the cardiac centre before the arms are affected.

The course of the disease is usually, though not always, rapid and, as a rule, fatal. Of four cases which have come under my own observation, and which presented typical symptoms of Landry's paralysis, two died. The mortality is very high, and many cases end fatally in a week. The paralysis may run a still more rapid course, attacking the cardiac or respiratory centres—the usual mode of death—in forty-eight hours. In other cases, again, the progress is slower, and the patient may live three or four weeks. When recovery takes place it is usually slow.

Pathology. In the majority of cases the results of autopsies have been negative, even in most competent hands. Again, in other cases that have been apparently typical, varied pathological findings have been reported. It is exceedingly probable that the primary cause of the paralysis is some toxic principle which has an affinity for the nervous motor elements, either in the spinal cord or nerves, or both. This hypothesis is supported by analogy with other morbid processes which are known to be caused by such influences, such as diphtheritic paralysis, which it is now known is due to the action of the toxin of the Klebs-Lœffler bacillus upon nerve structures.

It is highly probable that the paralytic symptoms at the onset are due to the chemical affinity of the poison for the nervous motor elements, while the continuous action of the poison may cause various secondary changes in the nerves and cords, and perhaps in the muscles. This would be in harmony with the entire absence of anatomical changes, which is the rule, as well as the varied and dissimilar lesions found in other cases, such as neuritis, poliomyelitis, etc. Baumgarten, in one case, found in the spinal cord many rods like the bacilli of malignant pustule. In Curschmann's case the typhoid bacillus was found in the same region. Catanni found bacilli in the endoneural lymph-spaces of many of the peripheral nerves. Eisenlohr demonstrated various kinds of bacilli, and in one rather typical case, found staphylococci in various organs of the central nervous system. In some cases disseminated inflammatory patches or ecchymoses were found in the medulla, especially in the motor tract, and in others similar alterations in the cord. Particularly marked was the swelling of axis-cylinders in the crossed and uncrossed motor tracts. In still other cases, what was found corresponded to the lightest form of poliomyelitis. Pitres and Vaillard found nothing abnormal in the gray matter of the spinal cord, and few alterations in the spinal nerve-roots, but found the peripheral nerves degenerated to such an extent that in many fibres nothing remained but an empty sheath of Schwann. Watson found nothing but a congestion of the spinal meninges. Luigi Villa found in his case in "the peripheral nerves and in the nerve-roots the lesions of parenchymatous neuritis with preservation of the axis-cylinders, but no changes in the intra-vertebral ganglia. The axis-cylinders were surrounded by accumulations of a substance having a special morphology and micro-chemical character, which was also found in the transverse sections of the spinal cord, particularly in the periphery. The central canal was filled with cellules and globules which completely obstructed it. The same condition existed in the brain and cerebellum, where the nervous elements were well preserved. In the muscles the fibres were numerous and the nuclei of the sarcolemma augmented."[1]

Ross, after an analysis of ninety-three cases, believes the disease to be a form of multiple neuritis. Oppenheim mentions a case in which the examination of a fragment of muscle, secured by means of the harpoon during life, showed waxy degeneration. It should be said that Landry thought a toxæmia to lie at the base of the disease, and this idea certainly acquires support from the fact that in it acute swelling of the spleen is common, and of the lymphatic glands not rare. If this view be correct, it still remains to be determined whether there is a single specific poison or whether there are several which can produce a similar effect upon the nervous system. After what has been said, a little consideration will show that our knowledge of the nature and pathology of this affection is in a very confused state, and the frequent occurrence of neuritis, which has of late been found, has only added to the confusion. Clinically, this uncertainty has undoubtedly led to numerous errors of diagnosis. Supposing that the toxic principle has induced a secondary neuritis, is the disease to be looked upon as essentially a toxic paralysis, or is it to be claimed as one of the numerous forms of multiple neuritis of a peculiar type (clinically)? If the latter, how is it to be distinguished from other varieties? If, on the other hand, the symptoms in a given case are plainly indicative of a neuritis, can we determine with positiveness whether or not we have to do with a toxic principle in addition?

At present we have only its course and termination to guide us. The fatality of Landry's paralysis is certainly excessive as compared with ordinary multiple neuritis, and points to another factor as being predominant. As the prognosis is so grave, the necessity of distinguishing this variety from other varieties of neuritis or myelitis is evident. It is highly probable that occasionally cases of diphtheritic paralysis have been mistaken for Landry's paralysis. It is well known that in the former it is not always possible to obtain evidence of a preceding throat affection, owing to the fact that the latter was very mild or that the primary lesion was confined to the nares. The similarity between the two varieties of paralysis is marked when there is little sensory disturbance in the diphtheritic form and the paralysis is widespread.

Diagnosis. This must be made from acute anterior poliomyelitis, myelitis, neuritis, diphtheritic paralysis, and paralytic rabies. The recognition of the disease described above must rest upon the peculiar ascending succession of the muscles involved in the paralysis, the character of the paralysis—almost entirely motor—the preservation of electro-muscular excitability and of almost unimpaired sensation, the absence of atrophy, and the (usual) exemption of the sphincters. In diphtheritic paralysis there is most commonly a history of faucial or nasal infection, and paralysis of the palate and accommodation as initial symptoms. Sensory symptoms also are more common. In paralytic rabies we also have usually additional aid in the history of a bite of an animal.

Prognosis. This is extremely bad, although, as has been said, recoveries may take place oftener than statistics would indicate.

Treatment. Owing to the rarity of the disease no extensive knowledge from the application of therapeutic measures has been gained. Probably no remedy thus far employed has any specific effect. Possibly a line of treatment similar to that found useful in diphtheritic paralysis would be most effective, especially alcohol and strychnine, the latter given subcutaneously. In the early stages of the disease a vapor bath has been recommended, if exposure to cold has been an etiological factor, though it is difficult to reconcile this treatment with the toxic theory. The bath should be followed by counter-irritation in the form of a long, narrow strip of mustard-plaster over the spine. The body should be kept in as perfect a state of rest as possible. Internally large doses of ergotin were thought to be successful in a case mentioned by Gowers. The other remedies used are iodide of potassium in doses of 10 to 20 grains, well diluted, three times a day; salicylate and benzoate of sodium; mercury, either in the form of the iodide, internally, or the ointment by inunction; belladonna, in the form of the fluid extract, 1 or 2 minims once or twice a day; and the sulphate of quinine, 2 grains three or four times a day. In cases that follow traumatic lesions, especially if there have been any indications of septicæmia, Gowers thinks that full doses of the perchloride of iron offer the best means of destroying the activity of the blood state.

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[1] Sajous' Annual, 1894.

SYRINGOMYELIA.

Syringomyelia is characterized by the formation of a tubular cavity or cavities in the substance of the cord, and the development of gliomatous tissue. Its symptoms are at first latent in their course and slow in progression, but finally in a certain proportion of cases present certain peculiar groupings which allow the disease to be recognized. In other cases they are atypical and present no definite grouping. A knowledge of the mode in which the cavities and gliomatous tissue are developed enables us to understand the relations between the symptoms and the anatomical changes in the cord.

It is generally agreed that gliosis is a congenital condition, due to a defect of development resulting in the persistence of embryological tissue with subsequent proliferation in the adult cord. Regarding the cavities there is still a considerable difference of opinion as to whether all have the same origin or whether there are several ways in which a cavity may be formed. There is no doubt, however, that there are at least two modes by which cavities originate, and all cavities associated with gliomatous tissue may be referred to one or the other of these two modes of origin. First, a cavity may be formed by the persistence of the primitive tube of which the embryonic cord is composed, and later be enlarged by the breaking down of the embryonic tissue forming its walls.

The second way in which a cavity originates is by the proliferation and subsequent breaking down of nests of gliomatous (embryological) tissue, which has become imprisoned in the posterior gray matter in the course of development; consequently some cavities may represent the original tube, and others may be entirely new formations. The developmental process by which this is brought about is as follows: In the beginning the spinal cord is a simple tube composed of embryonic tissue—a substance which is gelatinous in appearance—and resembles, if it is not identical with, gliomatous tissue. In the process of development the tube becomes flattened laterally and closes by approximation by its sides with the exception of the anterior (ventral) portion, which remains patent and persists as the central canal. From the walls of the primitive tube, by a process of proliferation and transformation, various structures of the cord are formed. When the tube fails to completely close a cavity is left, necessarily lying behind or corresponding to the central canal and extending up or down the cord a greater or less distance. When this is the case it is found that some of the surrounding embryonic tissue also persists, and later by a process of growth increases in amount. This proliferating tissue surrounding the cavity is liable to break down, and thus the cavity is enlarged. The walls of the cavity are formed of persistent gelatinous embryonic tissue of varying thickness.

It should be observed that the clinical phenomena are conditioned by the fact that the remains of the tube, that is, the cavity, necessarily occupies either the position of the central canal or lies in the posterior half of the developed cord between the posterior cornua. In the latter position the cavity may or may not connect with the central canal. Whether the primitive tube has closed or not, nests of embryonic tissue may become imprisoned in the posterior portion of the cord. These are later also liable to proliferate as gliomatous growths, then degenerate, and finally break down, forming cavities. By the proliferation of the gliomatous tissue a distinct tumor formation may result, more or less differentiated from the surrounding tissue.

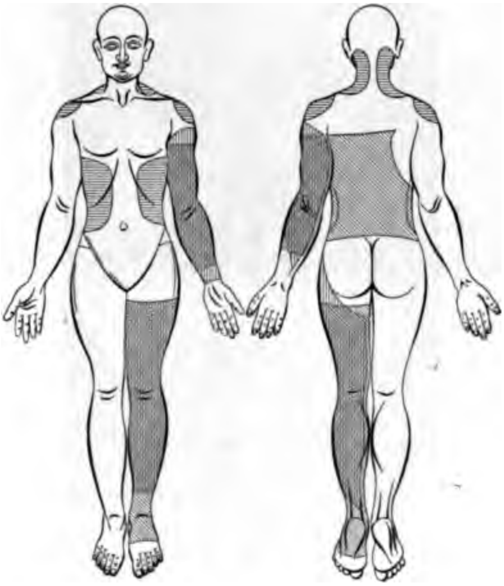
Syringomyelia is thus primarily a congenital disease due to mal-development, but it is a condition liable to secondary changes, cell-growth and disintegration, which occasion profound damage to the neighboring structures of the cord. It will be also seen that the cavity may represent an injured central canal or may be independent of it. It is to be noticed also that these new formations necessarily lie in the gray matter and in the posterior half. In consequence of the fact that the structures of the cord are not necessarily injured until the secondary changes, growth and disintegration, take place in the gliomatous tissue, the disease may lie latent long periods before it produces symptoms, and therefore it is that there may be no indications of such a cavity during life. The slow development of the symptoms after their onset corresponds to the slow growth of the gliomatous tissue and its disintegration. The fact that these formations are in the posterior gray matter occasions a peculiar grouping of the symptoms such as would be due to a central lesion. When, however, as is often the case, the development of the cavities encroach upon the neighboring white matter, other symptoms become added to or are substituted for those which are referable to the damage to the gray matter. The symptoms thus produced may not exhibit in their grouping any particular type, but may simulate various other destructive diseases of the cord. Therefore it is that syringomyelia sometimes cannot be diagnosed during life, even when clinical evidence of profound damage is present.

The extension of the cavity may injure the ascending and descending tracts in the white matter, thus producing paralysis and ataxic symptoms, or it may be into the anterior cornua, producing atrophic changes. While the majority of cases are unquestionably congenital from the presence of embryonic tissue, in others this origin must be inferred from the similarity of many of the pathological conditions present to those in the congenital cases. The following description of the symptoms applies to those cases in which the symptoms are so grouped as to produce a distinct type. The variations frequently met with will be afterward mentioned.

Symptoms. The symptoms come on insidiously, are slow in their development and at first are apt to be overlooked. They are most frequently observed between the ages of twenty and thirty, and therefore up to this period such congenital anomalies as may exist must be quiescent. In children, accordingly, symptoms are rare, and in some cases the manifestations of the disease have not made their appearance until middle life, or as late as the ages of fifty or sixty. The symptoms are almost always bilateral, but a few cases have been observed in which only one side of the body was affected. The most common situation for the lesion is the cervical enlargement, and hence the distribution of the symptoms is most commonly in the arms. The most common type, the one we are now considering, is that in which the most salient features are loss of perceptions of pain and temperature, with retention of the tactile and muscular senses combined with atrophy of the arms, similar to that observed in spinal progressive muscular atrophy. (See Fig. 180.) In this type usually the first symptom noticed is the loss of the sense of pain and of temperature. (See Figs. 179 and 181.) This dissociation of the different sensory perceptions was at one time thought to be pathognomonic of syringomyelia. It is now known that this is an error, and that this peculiar dissociation occurs in other diseases, as in neuritis, myelitis, and hysteria.

In syringomyelia this phenomenon is best explained on the theory that the paths for the conduction of the perception of touch and pain take different directions in the cord, and that those for pain cross in the posterior commissure to the antero-lateral ascending tract.[1] Consequently a lesion of the gray matter in the posterior half of the cord might obliterate the sense of pain alone. Further, the frequent association of the loss of the temperature sense with that of pain in other diseases than syringomyelia shows that the two paths probably lie close together. The sense of touch is not always preserved. In some cases it has been lost with the others, probably in consequence of the extension of the lesion into the posterior root zones.

FIG. 179.



The distribution of the areas of . . . ▥ *Analgesia*, ▨ *Thermo-Anaesthesia*, and general ▤ *Anaesthesia* in a case of syringomyelia. (LLOYD.) Excepting on the back, the "dissociated" sensory loss was confined to the left half of the body. The limits of the analgesia did not correspond with those of the thermo-anæsthesia.

Rarely the temperature sense alone is lost for a considerable time, while that of pain is preserved. The thermo-anæsthesia usually is not absolute for all degrees, but more commonly for mean temperatures only, but sometimes it is the extremes of temperatures that are not recognized, so that, for example, the perception of cold is preserved and that of heat is lost. In a case of Déjérine and Thuilant[2] the thermal sense was lost for all temperatures above 68° F., but preserved for all degrees below this point. The sensibility for touch and pain was preserved. If such patients are not carefully tested partial defects in the perception of sensation are liable to be overlooked. The loss of sensation is more commonly confined to the arms and adjacent portions of the trunk, corresponding to the cervical location of the lesion. Sensation in the legs is usually unimpaired in this type, in consequence of their sensory fibres having crossed below, and thus evaded the lesion in the gray matter above, but there may be much variation in the distribution of the analgesia. Sometimes it is general. It may, however, occur in the form of a hemi-analgesia.

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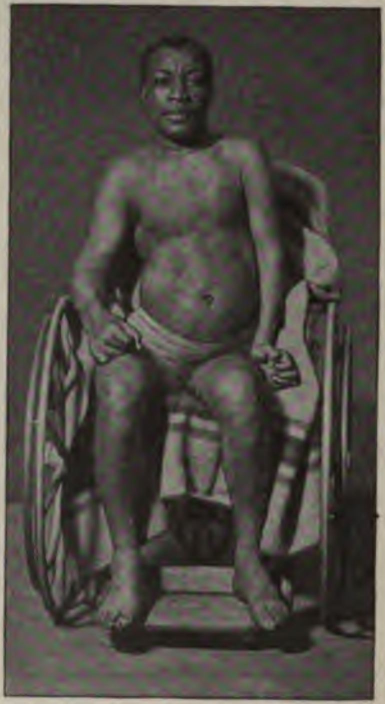
[1] Gowers.

[2] La Médecine Moderne, Feb. 5, 1891.

FIG. 180.



Syringomyelia presenting atrophy of muscles, scoliosis, and marked sensory changes. (Philadelphia Hospital.)

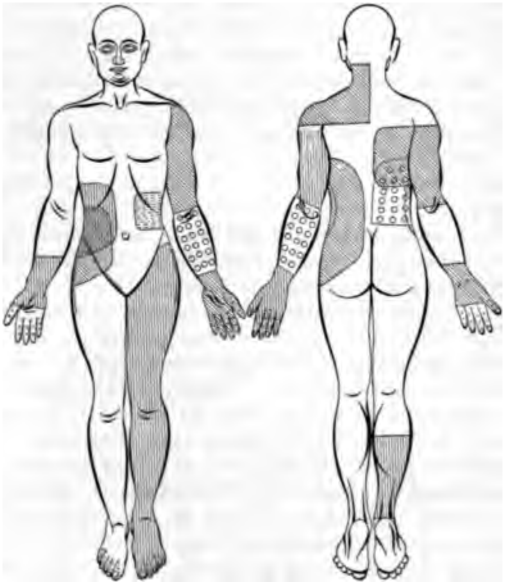
The areas of the loss of sensibility for pain and temperature may not correspond; one may be wider than the other, and there may be areas where one is lost and the other preserved. There may be analgesia on one side of the body and thermo-anæsthesia on the other.

According to Bruhl and Charcot the loss of sensation in the arms occurs in zones, extending in a circular manner around the limbs, as in hysteria, and does not follow the anatomical distribution of the nerves as in neuritis. This zone configuration is thought to correspond with the arrangement of the spinal centres.

Symptoms of sensory irritation are not infrequently observed, and may be the first thing complained of, such as paræsthesia of different kinds (numbness, prickling feelings, etc.), and pains referable to the arms, neck, and different parts of the body. These pains resemble in character those occurring in

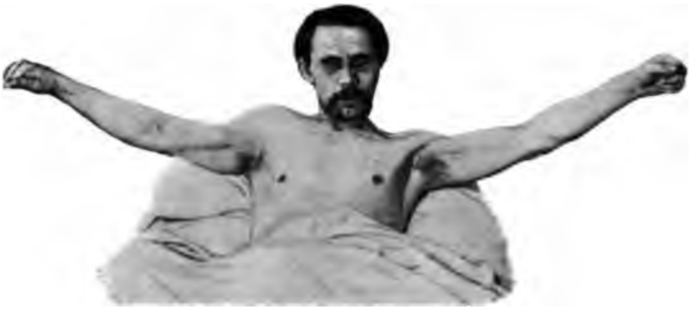
tumors of the cord, excepting that they are less intense, and are the occasion of annoyance rather than of suffering. They are very likely due to the same mechanism, viz.: irritation of the sensory fibres by the gliomatous growths or distended cavities. After symptoms of impaired sensibility have persisted for some time the extension of the lesion into the anterior cornua or the effect of pressure upon them begins to cause weakness and atrophy of the arms.

FIG. 181.



Sensory chart of patient shown in Fig. 180, showing areas of . . . . ▨ *Thermo-Anaesthesia*, ▥ *Analgesia* ▩ *Thermo-Anaesthesia, and Analgesia*, ▤ *Tactile Anaesthesia*, and areas in which the patients answer to tests of temperature showed reversal ⁂ *Cold-Hot; Hot-Cold.*

FIG. 182.



Syringomyelia. Extensive arthropathy of the right shoulder. (DERCUM.)

The atrophy is usually of the so-called Aran-Duchenne type. It may appear in the small muscles of the hand and gradually extend upward, involving

consecutively the forearm, upper arm, and shoulder muscles, or it may first appear in the shoulder and upper-arm muscles, and later descend to the hand. The difference depends upon whether the lower cervical gray matter is first affected with extension upward of the process, or whether the upper cervical enlargement, in which are located the centres for the shoulder muscles, is first affected. Corresponding with the atrophy there is naturally a weakness of the muscles which may go on to complete paralysis. The electrical changes in the atrophied muscles are usually limited in accordance with electro-physiological laws to quantitative diminution to both galvanic and faradic currents. But when the atrophy is rapid, some muscles may exhibit the reaction of degeneration.

Fibrillary contractions in the atrophied muscles are not rare as in progressive muscular atrophy.

Tremor may be present in the affected limbs, and rarely spasms.

The weakness of the spinal muscles produces scoliosis (see Fig. 180), which is a common symptom and quite characteristic. It is probable that a scoliosis of limited extent may be due to atrophic changes in the vertebræ similar to those that occur in the joints. As the disease progresses damage to the pyramidal tracts causes weakness of the legs, with spastic symptoms.

Trophic disturbances are common. Changes in the joints and bones very similar to those observed in tabes occur in about 10 per cent. of the cases (according to Ssokolow). Like the other symptoms they more frequently occur in the upper extremities, while those of tabes occur in the lower. The joint changes consist principally of enlargement of capsular ligaments, looseness of the joints, thickening of the capsule, changes of form in the ends of the bones, and development of bony spiculæ in the capsular walls. The further changes resemble those in tabetic joints. (See Fig. 182.)

Painless fractures of the bones may occur from very slight causes, as in a case of a man who fractured the radius while kneading dough (Schultze).

Various atrophic changes in the skin are frequent, such as herpes, eczema, and even deep ulceration and gangrene; in rare cases amputation of a hand may in consequence be necessary; or there may be simply vasomotor changes, causing lividity and coldness of the skin or the opposite, or œdematous swelling of the hands. There may be sweating or dryness of the skin. The nails may become dry, cracked and brittle, and may drop off.

An obtrusive symptom which is sometimes observed is the painless felon, similar to that which occurs in Morvan's disease. The felons occasion deep ulceration and necrosis of the distal phalanges of the fingers, so that they may drop off. Notwithstanding this extensive ulceration, the felons are painless, owing to the analgesia present. In consequence of the association of these felons with syringomyelia this disease and Morvan's have been considered by some as identical. This is probably an error, though it may be that these atrophic lesions may be due to the same pathological conditions in the two diseases, viz.: a neuritis, or possibly disease of the trophic centres. These conditions will be more fully considered in the section on Morvan's disease.

In some cases the disease takes on a less typical form. As has been said, tactile sensation may be lost with that of pain and heat. The secondary changes may be located in the dorsal or lumbar regions of the cord, producing corresponding variations in the symptoms. When in the lumbar region, the atrophy, loss of sensation, trophic and other symptoms usually observed in the arms occur in the legs. The sphincters are then affected with the usual secondary results. The disease may extend upward from the cervical enlargement into the medulla and pons, producing bulbar symptoms and paralysis of the cranial nerves, such as occur in other forms of bulbar disease.

Other changes in type are due to the irregular extension of the lesion transversely in the cord. One type is that simulating amyotrophic lateral sclerosis, from damage to the pyramidal tracts and the anterior cornua. Muscular atrophy and spastic paralysis are then the predominating symptoms. It may be that a spastic hemiplegia alone may be present, lasting for years before the development of other symptoms. A third type is that simulating Morvan's disease, in which the loss of sensation, atrophy, and felons are the most salient symptoms. It may not be possible to distinguish this type during life from Morvan's disease, and this has given rise to the view just referred to that the two diseases are identical. A fourth type may be called latent syringomyelia in consequence of the disease producing no symptoms whatever.

Besides the symptoms thus far described others sometimes occur. Very marked ataxy similar to that in tabes, has been found and is due to the secondary development of a posterior sclerosis.[1]

The pupils may be unequal in size, and contracted from injury to the fibres, innovating the radiating muscles of the iris. They respond to light and accommodation. Contraction of the bulb with narrowing of the palpebral fissure and slight ptosis have been observed. Contraction of the visual field of both eyes has been noted even where the lesion has been unilateral[2], though in the latter case the contraction was greatest on the side corresponding to the lesion. This symptom can only be regarded as a functional complication. Nystagmus sometimes occurs. The sphincters in most cases are unaffected, but may be involved in the later stages. In advanced cases bedsores and cystitis may develop as in other diseases, which cause transverse lesions of the cord. In some of these atypical forms it may not be possible to diagnose the disease.

The course of the disease is slow, usually extending over many years, unless there has been a distinct tumor formation of gliomatous tissue, or the development of a sarcoma has occurred, in which case the disease may be shortened, in accordance with the usual course of tumors, or the disease may be shortened by an acute destructive process in the gliomatous tissue. There are frequently periods of remission when the disease is stationary for long periods, and there may even be periods of temporary improvement. Death may finally be the result of exhaustion, bed-sores, cystitis, or the extension of the disease into the medulla; or life may be cut short by intercurrent diseases to which such patients seem to be peculiarly liable.

Pathological Anatomy. The most common seat of the disease is in the cervical and upper dorsal regions; hence the arms are most frequently affected. The cavities vary in length. They may extend the whole length of the cord and even upward into the medulla and pons. They may end in a solid mass of gliomatous tissue. There may be one cavity, or a secondary fissure may form from the disintegration of the tissue.

They may extend irregularly in a transverse direction backward into the posterior horn or forward into the anterior gray matter, and even laterally into the white substance, or the cavity may be—and this is the case especially in children—simply a dilatation of the central canal surrounded by a mass of gliomatous substance. The cavity may or may not be lined by cellular membrane, which is probably a rudiment of the original membrane lining the primitive embryonic tube.

The cavity sometimes is in connection with the central canal and sometimes is not. From an examination of 74 cases which he was able to collect, Chiari concluded that the cavity probably connected with the central canal in 45 (including those where the cavity was a mere dilatation of the canal). In 21 cases the cavity was thought to be due to destruction of gliomatous tissue alone.

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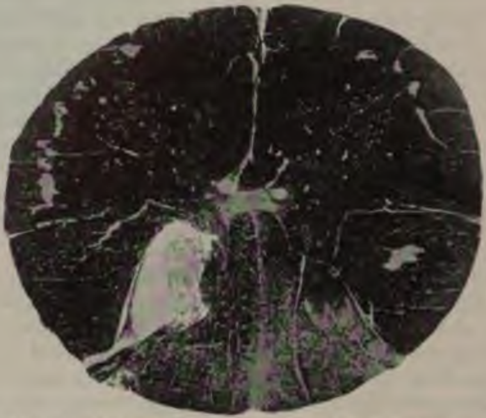
[1] Two cases are to be found in the literature in which, notwithstanding the presence of symptoms resembling those of locomotor ataxia, the posterior columns were not sclerosed.

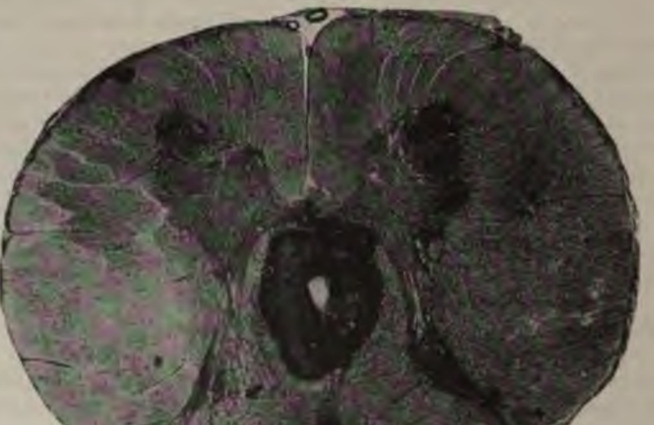
[2] Déjerine and Sotta: Compt. Rend. Hebd. Soc. d. Biol., July 23, 1893.

FIG 183.



Transverse section of the lumbar cord, showing gliosis of the posterior cornua and central gray matter. Weigert stain. (Figs. 183, 184, and 186 are from specimens kindly furnished me by DR. E. W. TAYLOR.)

FIG. 184.

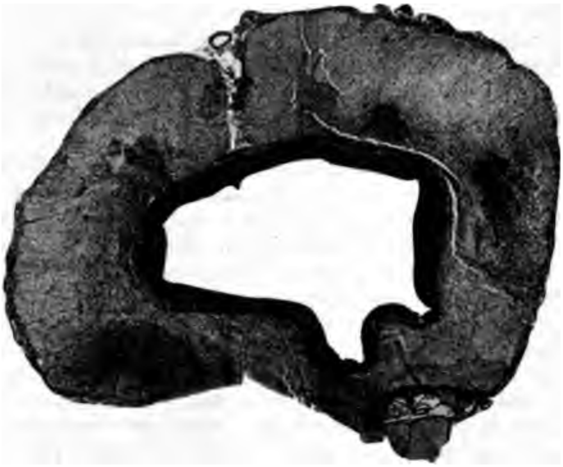


Transverse section of the cervical cord, showing circumscribed gliosis of the central gray matter and adjacent areas. The gliomatous tissue lies between posterior horns and posterior to gray commissure. A minute cavity is apparently just beginning to be formed in the centre of this tissue. Nigrosin stain.

It should not be overlooked that the clinical phenomena are not coextensive with the cavity or the distribution of the gliomatous tissue, but are related only to those segments in which the nervous substance has been injured by pressure, and, perhaps, secondary inflammation. So that even when the syringomyelia extends the whole length of the cord the symptoms may be limited to the arms.

The mass of gliomatous tissue varies largely. It may be present as a thick wall lining the cavity, or it may be so small as scarcely to be found, and its original presence must then be inferred from the nature of the disease. There may be a local proliferation so as to form a well-defined tumor or a glioma, which may have a considerable vertical extent. There seems to be a tendency to the development of localized tumors, which are either gliomata or sarcomata, and later not infrequently give rise to syringomyelia.

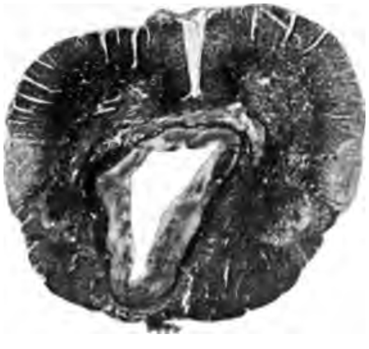
FIG. 185.



Transverse section of the cervical cord in a case of syringomyelia, showing gliosis with extensive cavity formation. The lateral tracts are also seen degenerated. Carmine stain. (From specimen loaned by DR. DERCUM.)

The gliomatous tissue has a gelatinous transparent appearance, and under the microscope it seems to be made up of small round cells with fine fibrous processes, and probably has much in common with embryonic tissue and normal neuroglia, if not identical with them. It is firmer than the surrounding tissue, and when circumscribed can sometimes almost be shelled out.

FIG. 186.



Transverse section of lumbar cord, showing gliosis with formation of cavity between posterior horns and posterior to central canal, the remains of which can be seen to be distinct from cavity. Degeneration of lateral tracts. Weigert stain.

The pressure of the growth and distended cavities may cause atrophy of the adjoining gray matter, particularly the anterior cornua, and even of the neighboring white substance. Hemorrhage, sometimes takes place into the cavities.

Cavities have been found in the cord in connection with other processes than gliosis, such as myelitis. In view of the researches of Van Giesen[1] who has shown how easily such cavities may be artificial productions as the result of carelessness in the removal of the cord, it is well to suspend our judgment before accepting such conditions as the result of a pathological process. According to Van Giesen several of the supposed cavities reported to have been found in the cords were only artificial products.

A fact of considerable importance connected with the trophic phenomena is the degenerative neuritis which has been found both in the sensory and motor nerves.

Diagnosis. The principal diseases with which syringomyelia is likely to be confounded are leprosy, Morvan's disease, focal myelitis, progressive muscular atrophy, cervical pachymeningitis, tumors, and neuritis. The other manifestations of leprosy are sufficient to make the distinction, although, overlooking this fact, some writers have claimed the identity of syringomyelia and leprosy, on account of the similarity in the sensory phenomena observed in both. Some cases cannot be distinguished from Morvan's disease until at least the disease has progressed so far as to produce symptoms which are clearly referable to the cord. A focal myelitis sometimes produces symptoms resembling a limited syringomyelia. Two cases have been reported by Charcot and Robinson, respectively, and I have myself now a case under observation; in these the symptoms resemble those of the disease now under discussion. All three cases immediately followed traumatism, which will serve as an important point in the diagnosis in similar cases. The course of the disease, the tendency to improvement rather than to the extension, the absence of the peculiar atrophic changes of syringomyelia, and the comparatively rapid onset of the symptoms must ordinarily be sufficient for diagnosis.

Progressive muscular atrophy may be distinguished by the absence of the sensory and atrophic disturbances. Chronic pachymeningitis pursues a more rapid course and causes much more severe pains, which are prominent in the disease. Trophic changes other than the muscular atrophy are absent, and the peculiar dissociation of the sensory symptoms is not met with.

The symptoms may resemble those accompanying tumors when there is a marked circumscribed growth in the cord, but in tumors symptoms of irritation are much more prominent and last a longer time, as a rule, before the development of symptoms of damage. The peculiar atrophic and dissociation symptoms of syringomyelia are not met with. Neuritis, in rare instances, simulates syringomyelia.[2] It is to be distinguished by its rapid development, the rarity of the dissociated sensory phenomena, the relative mildness of the anæsthesia, and the limitation of the analgesia to those nerves (or nerve-roots) which are distributed to the atrophied muscles. In syringomyelia the area of analgesia is more extensive, and, according to Bruhl and Charcot, envelops the whole circumference of the area instead of following the nerve distribution as in neuritis.

When no symptoms are present, of course syringomyelia cannot be diagnosed during life. Our knowledge of the relations between the cavity formation and symptoms is far from complete, and, as Déjerine[3] has pointed out, the cavities are often not located where they are supposed to be from the grouping of the symptoms, even when the disease has been correctly diagnosed.

The pathology of the atrophic disturbances is far from clear, although it is probable that they are dependent upon a neuritis; but, if so, it is not plain why the neuritis occurring in this disease produces such peculiar trophic changes, while they are absent in the ordinary forms of neuritis met with under other conditions.

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[1] N. Y. Med. Journal.

[2] Charcot: Archiv. d. Neurol., September 9, 1892.

[3] La Semaine Médicale, February 12, 1890.

Prognosis. The disease may last many years, from five to twenty. During intervals it may be stationary, and even some improvement may temporarily occur.

Treatment. There is no known way at present of affecting the disease. The only treatment is that called for by the skin eruptions, felons, paralysis of the bladder, bed-sores, etc.

MORVAN'S DISEASE.

The name Morvan's disease has been given to a group of symptoms which were first described by Morvan, of Lannelis, Brittany, 1883. The pathology of the disease is obscure, but it is probably a peculiar form of neuritis. In some of its clinical features it resembles syringomyelia so closely that some writers consider the two diseases identical. This is probably an error, but as cases of each are sometimes mistaken one for the other, and considerable discussion still continues regarding its pathology, it is described here. The disease is rare, but cases are being reported from time to time.

One of its chief characteristics is the occurrence of usually painless, deep-seated ulcerations of the terminal phalanges of the fingers, or felons; hence the name, analgic panaritium, by which it is also known. These felons lead to the destruction of the bones.

Symptoms. The first symptom usually complained of is neuralgic pains in one or both hands. In some cases the pains are distributed through both limbs of the same side of the body. They may be slight in intensity or severe, and in some cases may not be present at all. Later, successive felons appear. These are painless, owing to the loss of sensation, which examination at this time will show to be present. Occasionally ulceration precedes the loss of sensation, and in this case the felons are painful, but usually this is true of only the first felon.

The ulcerations are deep and sometimes require the amputation of the phalanges; by the successive development of these felons and resulting necrosis, one phalanx after another may be lost. The intervals between the development of these ulcerative processes may be weeks or years. The loss of sensation, which is always present, involves all forms of sensation in the great majority of cases. The perceptions of touch, pain, and temperature are lost. In some cases the dissociation of sensation peculiar to syringomyelia has been observed; that is, there has been loss of the sense of pain and temperature, with the preservation of that of touch. It is possible that in such cases continued observation would have shown the disappearance of the tactile sense in the later stages of the disease.

The loss of sensation, as a rule, is distributed over the whole arm, and it may include the adjoining parts of the body and even the face. Its area of distribution is generally wider than that of atrophy and paralysis, which follow and constitute the third cardinal symptom.

At this time atrophy and paresis of the muscles of the hand and of the forearm will be found. The muscular wasting does not seem to extend above the forearm (?). In this respect the disease exhibits a marked difference from syringomyelia. After a while the second hand may be affected in a manner similar to the first. The disease is therefore bilateral. In rare cases the legs may be attacked later in the course of the disease in a similar manner. The atrophied muscles lose their electrical contractility to both galvanic and faradic currents. The reflexes are unchanged,[1] and the sphincters are unaffected. Bulbar symptoms have been observed. Other trophic disturbances than those of felons are common and resemble those which are observed in syringomyelia; similar changes in the joints occur, and deep-seated ulcerations in the hands, much like the perforating ulcers of tabes, sometimes develop, as well as abscesses in the arms. The skin of the hands becomes cracked, the nails dry, brittle, and fissured. Vasomotor changes of a marked character cause the skin to be livid or pale and cold to the touch.

The progress of the disease is slow, extending over many years. During this time felons and other trophic disturbances occur so that one phalanx after another may be lost. The fingers become distorted by the contraction of the interosseous muscles. By reason of these changes, as well as the paralysis and atrophy, the hands may become useless.

Pathology. As has been said, the disease is thought by some to be identical with syringomyelia, others being equally positive that the symptoms are dependent upon a special lesion, viz., neuritis. In several cases,[2] which were thought during life to be typical of Morvan's disease, gliomatous cavities were found at the autopsy. It cannot be too strongly insisted upon that such evidence does not show identity. We might as well argue that because alcoholic neuritis and tabes have been confounded that they are therefore identical. Almost any disease may at times simulate another. It is possible, in fact probable, that while Morvan's disease and syringomyelia are distinct diseases, the lesion upon which the trophic disturbances depend is the same in each, viz., a neuritis.

As we have seen, there is reason to believe that the trophic changes in syringomyelia are due to a coincident and secondary neuritis; likewise in Morvan's disease a neuritis has been found in the stumps that have required amputation for the trophic lesions.

In Gombault's[3] case, which came to an autopsy, there was found a peripheral neuritis and a sclerosis of the posterior columns and of the posterior gray matter of the cord. The sclerosis included the vessels, causing an occlusion of them; there were no cavities present. The sclerosis of the cord observed in this case was such as might well be secondary to the changes in the peripheral nerves. A single case like this is of much more value in throwing light upon the pathology of the disease than many others in which, though the symptoms simulate Morvan's disease, the central changes of syringomyelia are found. Such a case demonstrates that the group of symptoms described by Morvan may develop from conditions other than syringomyelia and is entitled to independent recognition. The fact that so many cases, twenty in number, occurred in the practice of a single individual in a small fishing town of 5000 inhabitants, is strong evidence that this affection is not due to cavity formation. It is hard to believe that so many persons in a small community were affected by such a rare disease as syringomyelia, whereas it might be possible that a neuritis of obscure and infectious origin might easily affect a large number of persons. It has been too readily assumed by those writers who hold to the theory of neuritis that the infection was due to fish. If a poison from fish would cause a neuritis of this kind the disease should be common in fishing communities generally. Some writers[1] attempt to show the identity of syringomyelia and Morvan's disease by pointing out the gradual shading of the symptoms from one disease to the other in graded cases, while admitting the existence of characteristic types of each. Arguments of this kind are obviously fallacious. A judicial consideration of the evidence thus far submitted must, however, find that the pathology of Morvan's disease is not beyond a doubt, but that the evidence strongly leans toward the view that it is due to a peripheral neuritis.

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[1] In some cases the deep reflexes of the legs have been observed to be increased. In consideration of the fact that some cases supposed during life to be Morvan's disease have been shown at the autopsy to be cases of syringomyelia, it is probable that those cases, in which paresis of the legs with increase of the deep reflexes without atrophy was observed, were really cases of syringomyelia, as such symptoms indicate a lesion of the cord. The symptomatology of Morvan's disease needs careful revision in some of its details, as in view of recent autopsies the two diseases have undoubtedly been confused.

[2] Joffroy (La Semaine Médicalo, March 4, 1891) has reported a second case.

[3] Archiv. de Méd. Exp., 1891.

Prognosis. Though few cases have thus far died from the disease, the prognosis, as far as recovery is concerned, must be regarded as unfavorable, but the disease may remain stationary a long time, and improvement has been observed in a number of characteristic cases. If the theory that the disease is a neuritis is correct, there is theoretically no reason why great improvement or recovery should not occur in mild cases. Our knowledge is at present so limited and the difficulty of distinguishing individual cases from syringomyelia is so great that we cannot at present formulate positive views regarding the prognosis.

Treatment. All rational treatment must be limited to the surgical treatment of the felons and other trophic changes. All treatment directed toward the disease itself must be purely experimental.

TUMORS OF THE SPINAL ENVELOPES AND OF THE CORD ITSELF.

In the present state of our knowledge it is not possible to distinguish clinically tumors of the cord from tumors of the spinal envelopes; it is therefore customary to consider them together. Nevertheless various attempts have been made to distinguish one class of tumors from the other, and certain symptoms have been supposed to be characteristic of one or the other class. More extended knowledge has shown that we have very little that is trustworthy for this purpose.

The following account is based on an independent study of 133 cases, which include the collections of Horsley and Mills and Lloyd,[2] and 37 others.

Etiology. Various causes have been assigned as productive of tumors; but, aside from syphilitic, tubercular, and parasitic growths, we know almost nothing of the real cause of tumors. Traumatism, "cold," and emotional excitement have been assigned as adequate causes of tumor, and this statement has been copied from one text-book to another, but the evidence is insufficient to justify this conclusion. The etiological question is an important one, especially in connection with traumatism, as claims for damage are likely to be made and have been made on such statements. The chief reason for assigning traumatism as a cause is that symptoms have been known to develop immediately after a fall or blow. Such evidence proves too much, for tumors do not, like mushrooms, spring up in a night, and a tumor that has attained a size sufficient to produce symptoms must have existed in such cases for some time previous to the accident. If the traumatism occurred at a remote period it is equally difficult to trace the cause and effect. At most the occurrence of symptoms following traumatism shows that a blow or a fall may have influence in developing symptoms of pressure which otherwise would not have appeared till a later period. Evidence of the correctness of this explanation is found in those cases where a similar train of symptoms followed traumatism, and yet the nature of the tumor was such as to show that it was due to a specific cause. For example, No. 53 of Horsley's collection was assigned to traumatism as a cause, and the symptoms of No. 16 were intensely aggravated by a strain, yet the former was a tubercle and the latter an echinococcus cyst. A case of my own, in which the diagnosis of a gumma was supported by the results of anti-syphilitic treatment, was ascribed by the patient to a fall.

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[1] Kornfeld: Wien. Med. Wochenschrift, May 11. 1892.

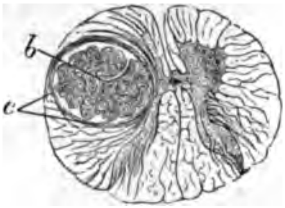
[2] Horsley's collection includes some of those collected by Mills and Lloyd. These, of course, are only counted once. The 37 additional cases have almost all been published since the date of Horsley's collection (1888).

There is a class of tumors which are probably really the consequence of traumatism, namely, cystic formations which develop from hemorrhage. According to Horsley some of the fibrocystic tumors reported as fibromata were really of this nature.[1] A hemorrhage first occurs in the membranes, and later undergoes secondary organization, finally resulting in a fibrocystic formation.

The effect of cold and emotional disturbances likewise is probably to excite at most symptoms of a previously existing tumor. An analogous effect of traumatism has been observed in other spinal diseases; for example, tabes dorsalis, in which it sometimes happens that the disease, which has been latent up to the moment of the injury, has suddenly given rise to marked symptoms. Gliomata, in many cases, and perhaps in all, are the result of a proliferation of embryonic tissue persisting in the cord.

Pathology. Tumors may grow from the cord itself (Figs. 187 and 189), or from any of its membranes (pia, arachnoid, dura) (see Fig. 188 and Plate III); or they may spring from tissues outside the dura mater, such as the fat lying between the dura and the vertebræ, the bones themselves, or the intervertebral cartilages. Tumors may therefore be classified as extra-dural, intra-dural, and medullary. The most practical classification for clinical purposes is:

FIG. 187.



Sarcoma of the lower cervical cord. (ADAMKIEWICZ.)

1. Medullary or tumors of the cord.
2. Extra-medullary or tumors of any of the envelopes.

This classification is based on the seat of the tumor, irrespective of the nature of the tissue from which it grows.

Tumors of the cord are far more rare than of the envelopes. Of 130 cases there were:

Outside dura	37
Between dura and cord	49
Outside cord, but exact location doubtful from report	11
Total outside cord	97
In the cord	19
Location doubtful from report	14
	130

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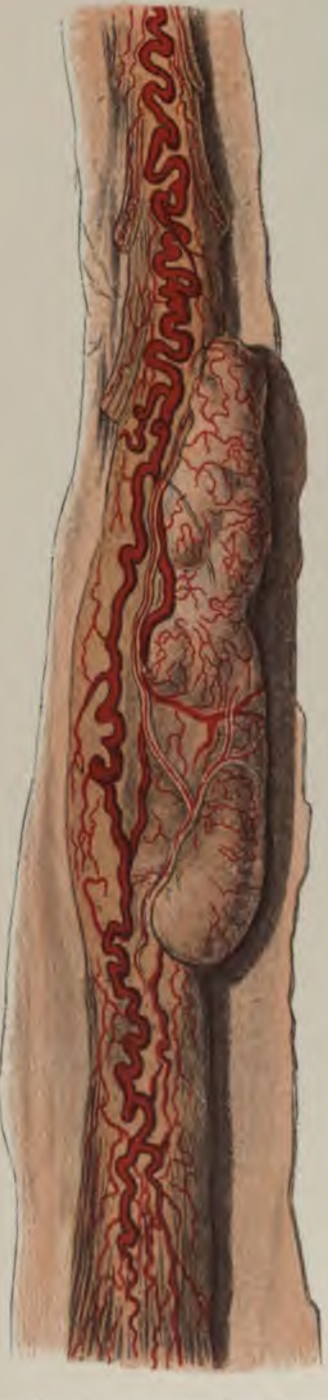
[1] Nos. 24, 28, 32, 44, 46.

PLATE III.



Cystic sarcoma of the arachnoid compressing the cord in the lower dorsal region. (Leyden.)

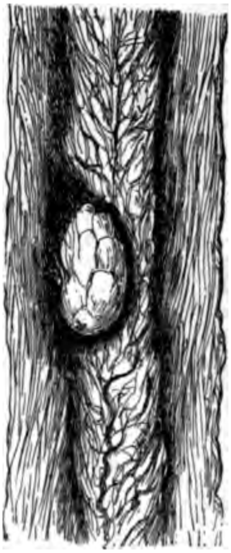
The different kinds of tumors met with are indicated by the following table. This table also shows, with probably fair accuracy, the relative frequency of each kind:

Sarcoma (different varieties)	36	Neuroma	2
Myxoma	15	Osteoma	2
Tubercle	15	Phlegmon	1
Echinococcus	10	Aneurism	1
Fibroma	8	Dermoid cyst	1
Gumma	8	Blood-clot	1
Psammoma	5	Angio-fibroma	1
"Cancer"	5	Lymphangeoma	1
Lipoma	7	Undetermined	4
Glioma	6		
Fibro-chondro-lipoma	1		130

Of the 19 tumors within the cord (included in these 130 cases), 5 were sarcomata, 4 gummata, 6 were gliomata, 2 were tubercles, and 1 was an echinococcus cyst (1 unknown).

Various mixed forms occur as in other parts of the body.

FIG. 188.



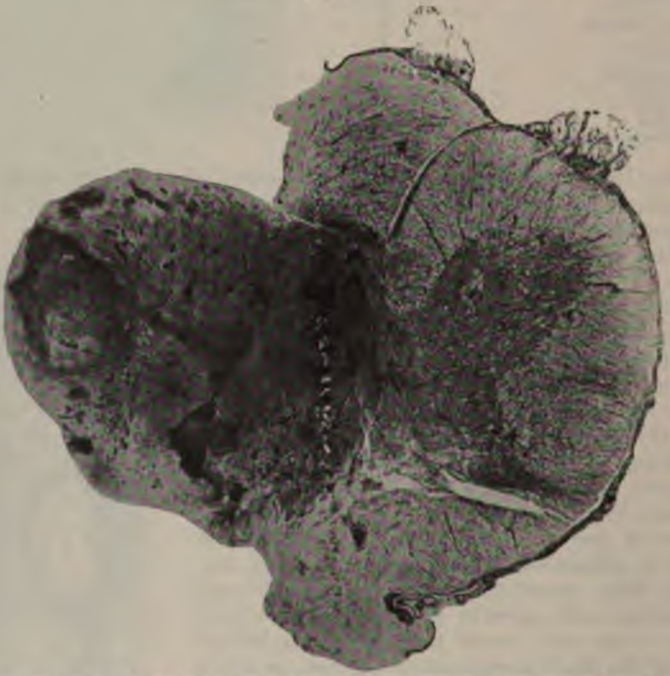
Sarcoma compressing the cervical cord. (E. LONG FOX.)

Tumors may be single or multiple. In the great majority of cases they are single. When more than one occur they are generally sarcomata or neuromata. Tubercular tumors are sometimes, though rarely, multiple. Spinal tumors growing within the canal do not attain to a large size, probably owing to the fact that before doing so they produce so much damage that death takes place. They vary from the size of a pea to a maximum diameter of two to three inches. One exception to this is gliomatous tumor springing from embryonal tissue about the central canal. Though in diameter such growths are small, in a vertical direction they may extend through a considerable length of the cord—sometimes throughout its whole length. They are often combined with syringomyelia, and then it is customary to consider them as a distinct affection. A diffuse sarcoma extending from the sixth cervical to the cauda equina has been observed.

The effect of tumors outside the cord is to compress its substance, producing atrophy, softening, or myelitis (pressure myelitis). An indentation of the cord corresponding to the tumor may be observed. The membranes in the neighborhood of the tumor may be œdematous or congested. The cord may be softened for some little distance above and below the tumor. This secondary inflammation may develop as an acute process, as indicated by the symptoms. In tumors of the cord substance the cord is enlarged at the seat of growth, and on cross-section the component structures of the cord will be observed to be pressed out of their normal positions, while the tumor itself will be firmer than the normal cord substance. The surrounding tissue will be softer. The central canal may be obliterated by pressure and dilated above the point of constriction. The association of gliomatous growths with cavity formations (syringomyelia) has already been referred to. Secondary degenerations usually occur, according to the well-known laws, *i. e.*, downward in the lateral columns, upward in the posterior columns.

Symptoms. The symptoms caused by tumors, whether they be within the cord itself or outside the cord, are of two kinds, namely, those due to irritation of the cord and the nerve-roots, and those due to destruction of the cord and nerve-roots or to impairment of their functions. The clinical recognition of these two sets of symptoms is important, as well as that of their order of development. The earlier symptoms are in the majority of cases those of irritation. At a later period symptoms of damage become associated with those of irritation; but this order is not always followed, as sometimes the initial symptoms are those of damage. The symptoms of irritation are pain and spasm; of damage, paralysis of sensation and motion, combined with the usual alterations in the reflexes, nutrition, condition of the sphincters, etc., found in transverse lesions according to the location.

FIG. 189.



Gliosarcoma of the cord. Nigrosin stain. (From specimen kindly furnished by DR. E. W. TAYLOR.)

Two stages can frequently be recognized: a first stage, in which symptoms of irritation predominate; and a second, in which those of paralysis constitute the principal objective feature, though pain still continues and aggravates the sufferings of the patient. Mills and Lloyd consider that these stages can generally be recognized.

The differentiation of stages by symptoms charcteristic of each cannot so generally be made, as some writers would have us understand. It is true that both irritative symptoms and damage symptoms occur in the great majority of cases, and that in a disease of this nature the closing scene must be one of damage; but the initial stage is not so very frequently one of irritation as is often asserted to be the case. In quite a large group of cases (25 out of 111) the first symptom is one of damage (paralysis or anæsthesia), and in others, again, the two sets of symptoms appear simultaneously, so that the cases may be arranged in two groups, a larger one in which irritation precedes damage, and a smaller one in which the reverse is the case; or both may develop together, or signs of irritation may be wanting. After symptoms of damage have developed both classes of cases pursue the same course and end in destruction of the cord.

The clinical features presented by individual cases of the larger class (as well as the smaller) vary much in the grouping, distribution, and mode of development of the symptoms. The symptoms are usually slow in their development, several months, as a rule, elapsing before decided damage to the cord has occurred; although in syphilitic tumors and tubercles this stage may be reached much earlier (three to eight weeks).

Initial Stage. The first symptom[1] complained of by a large majority of patients is *pain*, which may be referred to the distribution of the nerves arising at the level of the tumor, and in this case is due to the irritation of nerve-roots (root-pains), or it may be located in parts of the body below, and in this latter case is due to irritation of the sensory tracts within the cord (cord-pains). Pain is also persistent, as a rule, throughout the whole course of the disease. Out of 128 cases, it was present in 107 at some period of the disease. At the onset the pain is paroxysmal in character, with intervals of freedom. It is described sometimes as burning, sometimes as sharp, lancinating, and piercing. When the tumor is in the dorsal region, the pain may produce a feeling of intense constriction around the body (girdle pain). When in the cervical region, a similar sensation may be felt in the neck, causing a choking feeling. In severity the pain varies in individual cases; but while it may be slight, as a rule, it is severe, and is often so intense as to be insupportable without large doses of morphine. The intensity depends in part upon the nature and location of the growth.

The statement is frequently made that root-pains are more severe than cord-pains, and that in tumors of the membranes the pains are more likely to be due to irritation of the roots, and therefore more severe than in tumors of the cord. This needs considerable qualification. The question is an important one for differential diagnosis. From a careful study of the location of the tumors and of the character and distribution of the pains, in 69 cases available for the purpose, I find that in 57 the pains were described as "severe," "very severe," "atrocious," etc. Of these 57, in all but 8 the tumor was outside the cord, and yet the pains could only be interpreted as due to pressure on the cord itself in 18, on the nerve-roots in 18, and on both in 6. In 8 the report was not sufficiently detailed to enable the nature of the pains to be determined. In 3 the pressure was upon the cauda equina. In the remaining 12 of the 69 cases, the pains were of a comparatively mild character; in 3[2] the pains were due to pressure on the cord; in 5 to pressure on the roots; in 1 to pressure on both; in 2 doubtful; in 1 to pressure on the cauda equina.

Taking all the cases together, irrespective of the character of the pains, we have: cord-pains, 21; root-pains, 23; both, 7. In two additional cases the pain was local in the spine over the seat of the tumor, and, perhaps, should be classed with the root-pains. But these figures are probably only approximately correct, as it is not easy in every case to determine from the reports the origin of the pains.

From these facts it will appear that severe pains are quite as likely to arise from pressure on the cord as upon the roots, and that cord pains are as frequent in extra-medullary tumors as root-pains.[3]

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[1] It should be stated that in the later, and probably more accurately reported cases, pain is mentioned as the first symptom much more frequently than in the earlier cases, and it is possible that future observations will accord with the former.

[2] In one of these there were also pains which probably were root-pains.

[3] The fact is of importance in diagnosing the location of the tumor, as when the pain is due to pressure upon the cord it is impossible to determine from the distribution of the pain how much above the corresponding nerve-roots the tumor may be located.

Whether tumors of the cord itself are as likely to give rise to as severe pain as tumors of the envelopes, irrespective of their origin (cord-pains or root-pains), cannot be determined without a greater number of cases than has thus far been collected. It can, however, be said that both kinds give rise to severe pains.

In tubercle the pain is comparatively mild.[1] In tumors of the cervical and lumbar enlargement the pain may radiate down the arms and legs, respectively, involving distinct nerve-regions according to the nerve-roots or segment of the cord involved. In tumors of the cervical cord-pains in the neck are most commonly observed, but they are sometimes felt in the legs and in the back, and even a girdle pain around the waist may be present. When, at the onset they are felt in the neck or arms in the nerves arising from the cervical cord, they may at a later period as the tumor encroaches upon the cord substance appear in the legs and body below. In a few cases the pain has been felt in parts above the seat of the tumor.

It is not uncommon for the pain to begin on one side of the body, and then extend to the other side, after a distinct interval, or to begin in one arm and later appear in the leg of the same side or in the abdomen. In fact, it is not infrequent to meet with pains in one part of the body due to pressure on the roots and at the same time with pains in another part of the body from irritation of the cord. Sometimes the pain is increased or the paroxysms are excited by almost any movement of the body, such as jolting or jarring, or movement of the spine and limbs. Sometimes the pain is located in the spine, and there may be localized spinal tenderness. In some instances severe pains are felt in the joints. The slightest movement of the limbs may cause paroxysms of excruciating pain. In such cases the disease may be mistaken for rheumatism. At this stage of the disease the patient may suffer from pain alone for a long period before the development of other symptoms. The length of this stage varies according to the nature of the tumor, from a few weeks to one or two years.[2]

But early in the course of the disease another symptom of irritation—*spasm*—is apt to be associated with the pain. The spasm may take the form of simple stiffness of the muscles, or of clonic jerking of the limbs at intervals, or of contractures. Some spasm in the course of the disease is common, but it is not so frequent a symptom as pain. It was mentioned as present in one of its various forms in 73 out of 129 cases. Like pain, it may be due to irritation of the nerve-roots or of the cord itself. It occurs in intra-medullary as well as extra-medullary tumors. In tumors of the cervical region it may be observed as a rigidity of the neck, sometimes producing a form of torticollis. Spasm of the cervical muscles is more likely to be recognized than other spinal muscles, owing to the flexibility of the cervical spine. In tumors of this region the spasm may be observed in the fingers or arm; or, like pain, it may first and at an early period be noticed in the leg. When at the onset it is noticed in the upper part of the body it may later in the course of the disease appear in the muscles of the abdomen or legs. In tumors of the dorsal and lumbar regions spasm is necessarily limited to the muscles of the back, abdomen, or legs. Sometimes the spasms are very painful, and may, like pain, be excited by any movement of the limbs or body, according to their location.

Besides the pain already spoken of, patients may complain, in the early and middle stages, of still other sensory disturbances, such as paræsthesiæ, a feeling of numbness, prickling, and "pins-and-needle" sensations, and when pain is not present an examination may show the presence of the analogous symptom, hyperæsthesia. Sometimes, when sensory symptoms are not present in the initial period, this stage of irritation may still be recognized by the presence of some form of spasm.

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[1] Herter's 26 cases. Journal of Nervous and Mental Diseases, 1890.

[2] In one case excruciating pains alone persisted without other symptoms for eight years, when death took place apparently from exhaustion.

In some instances, in the earliest stages, the patient may notice a certain amount of weakness of certain groups of muscles. In a large minority of cases (25 out of 111), instead of symptoms of irritation, paralytic symptoms make up the initial stage. The disease begins then with gradually developing paraplegia, or, in a few instances, paralysis of one side of the body, or all four limbs. Occasionally the paralysis has come on suddenly after some exciting cause, such as exposure to cold.

Pain in this class of cases may appear later, though in most of them this is not recorded in the published report. It would, therefore, seem that in this class of cases the early development of the paralysis was dependent on the site of the disease. In a few cases, as was just mentioned, paralysis and pain appear together at the onset, or some form of spasm may be associated with the paresis. Occasionally, instead of pain, the initial symptom is muscular twitching or cramps, ataxia or vasomotor symptoms (œdema-redness of skin, etc.). The occurrence of paralysis as the initial symptom, or coincidently with pain, seems from the data at hand to be relatively more frequent with intra-medullary tumors, but the number of cases of this kind at our disposal is too small to make it safe to draw such inductions.

When a primary irritative stage has been present, as the disease progresses and the nerve-roots and the cord become more compressed, all the irritation symptoms increase in intensity, and may become more widely distributed. Symptoms indicative of damage are now added to the clinical picture.

Paralysis, if it has not already appeared, develops. One peculiarity of the paralysis, whether it follows a primary stage of irritation or is the central symptom, is that, with considerable frequency, it begins in one group of muscles, as of an arm or hand, and afterward extends to other muscles of the same limb, or the corresponding limb of the same side of the body. Later, with the growth of the tumor the other side of the body becomes affected. When, however, the tumor is centrally located, the paralysis may be bilateral from the beginning. This transference from one part to another, unfortunately for diagnosis, cannot always be traced, although it is likely that with more careful observation it would be recognized more frequently than the published reports in the past have indicated.

When the tumor is located in the cervical enlargement the arms may be, but not necessarily, the first to be paralyzed. Instead of the arms, the paralysis may first appear in the legs from damage to the pyramidal tracts.

The slower the growth of the tumor, the slower the development and the extension of the paralysis, unless a myelitis lights up, as is common. In the latter case the paralysis may come on rapidly, as in myelitis, due to other causes. Tumors arising outside the cord may cause paralysis of the nerves arising at the level of the tumor before affecting the cord. The final distribution of the paralysis depends upon the location of the tumor. When in the dorsal or lumbar region, paraplegia results; when in the cervical region, all four limbs are affected. One side of the body may be affected before the other; that is, a spinal hemiplegia or a Brown-Séquard's paralysis may result. Though the paralysis and other symptoms, when dependent upon compression, steadily increase, the same symptoms may, when due to the incited myelitis, largely subside with the inflammation, and thus a temporary improvement may occur.

About the same time with the paralysis, though more generally a little after, anæsthesia develops. The sensory fibres seem to be more resisting than the motor, and, therefore, the loss of sensation is not only apt to appear a little later, but may be comparatively slight, with great motor paralysis. With increase of compression the anæsthesia deepens. It will be found either in the course of the nerves, arising at the level of the tumor, or in parts of the body below from damage to the cord itself. In the former case isolated areas of anæsthesia may be found, and in any case the anæsthesia may occupy the same nerve lesions that are the seat of pains. This combination of anæsthesia and pain in the same area of the body is called *anæsthesia dolorosa.* It is due to destruction of the nerve fibres and irritation of their proximal ends.

The loss of sensation corresponds, as a rule, with that of motion, except that when the tumor is to one side and fairly high up (above the lumbar enlargement) symptoms of unilateral lesion, or Brown-Séquard's paralysis, just spoken of, may at first be present; that is to say, crossed paralysis of motion and sensation. Sensation is lost on the side opposite the tumor, and motor power on the same side. This is commonly explained by the theory that the sensory fibres cross soon after entering the cord, while the motor fibres do not do so until they reach the pyramids in the medulla; consequently, the sensory fibres of the opposite side and the motor fibres of the same side will be affected by any unilateral lesion. (As this anæsthesia does not quite reach to the level of the tumor, it is thought that the sensory fibres do not cross at once, but after ascending variable distances.) In unilateral lesion there may also be a zone of anæsthesia on the same side as the paralysis, corresponding to the nerves entering at the level of the tumor. The muscular sense, when this also is involved, is affected on the same side with the motor paralysis.

When the tumor is in the lumbar enlargement, although on one side, the anæsthesia is not crossed, but is on the same side with the paralysis, owing to the fact that the sensory nerves are affected before crossing. But it is not often that crossed paralysis is met with in a pure form, as the opposite side of the cord is generally more or less compressed, and some blunting of sensation and weakness of the muscles is generally observed on both sides, only the symptoms of unilateral lesion may predominate.

At this time, even after the paralysis and loss of sensation have become complete—that is to say, the lesion has become transverse—the pains not only generally continue, but may extend to other regions of the body and become intolerable. Painful contractures of the limbs may develop—the legs, for example, being held in flexion or extension, and any motion of the joints may excite excruciating pains. Sometimes any irritation of the paralyzed limbs may induce painful spasms.

With the symptoms indicative of the extension of the lesion transversely through the cord, paralysis of the bladder and bowels develops. The characteristics of this paralysis are the same as in transverse myelitis. When the tumor is in the lumbar region the sphincter of the bladder is paralyzed, and there is incontinence of urine; when above the lumbar enlargement, retention, with reflex evacuation or overflow. The same is true, *mutatis mutandis*, of the bowels.

At this stage there is a marked tendency for bed-sores to form. These sometimes commit frightful ravages. The necrosis may extend to the bone and open the vertebral canal. Septicæmia may result. Cystitis may also develop, and, secondarily, pyelonephritis. Œdema of the limbs sometimes occurs. Priapism is common as in other transverse lesions.

The reflexes follow the general law governing them in disease of the cord. Those arising from the diseased segment are sooner or later abolished; those from all lower segments are increased. The increase of myotatic irritability in the muscles below the lesion leads to the development in them of a spastic condition, and very frequently to contractures. An apparent exception has been observed in some cases in which, although the tumor was in the dorsal region, the knee-jerks were abolished; but such cases may be explained, as pointed out by Gowers and proven by Fancotte, by a descending myelitis[1] into the lumbar region. A careful study of the reflexes gives valuable information regarding the location of the tumor.

All paralyzed muscles do not atrophy, but only those whose nutrient centres in the anterior cornua or whose motor nerves connecting them with these centres are destroyed; hence the atrophy is limited to the muscles connected with the compressed segments or nerves, unless a more diffuse myelitis has been excited. The muscles connected with segments below are not affected with true atrophy, but may exhibit a certain amount of wasting from disuse. In consequence of the large number of nerves coming from the cervical and lumbar enlargements there may be much wasting of the arms when the tumor is in the former locality, and of the legs when in the latter. Likewise, of course, much atrophy of the leg muscles may occur in tumors of the cauda equina. When a descending myelitis has been superadded to the compression the atrophy is necessarily diffuse.

The electrical excitability of the wasted muscles will vary considerably according to the rate of growth and rapidity of destruction of the gray matter, or motor nerves. When the wasting has been slow, as is generally the case when myelitis has not been superadded, only a diminished reaction will be observed to both the galvanic or faradic current.

If an acute myelitis has been excited, affecting the anterior gray matter, the reaction of degeneration will be found in the acutely degenerated muscles. The excitability of the nerves that supply the atrophied muscles will be diminished to both currents; that of other nerves will remain unchanged.

When the tumor is in the cervical region, contraction or dilatation of the pupil sometimes occurs, the former being due to paralysis, the latter to irritation of the nerves supplying the radiating fibres of the iris through the sympathetic. A number of other symptoms are occasionally observed. Among them vomiting and headache have been noted in a few cases, and sometimes dyspnœa and dysphagia. Optic neuritis has been present in a few instances where the tumor was located in the cervico-dorsal region. Fever may occur as a consequence of bed-sores or cystitis, and vasomotor disturbances in the form of flushings of the skin, or œdema, are occasionally met with. Cough, dyspnœa, and pain in the chest have been noted in three cases of tumor in the cervical region.

The secondary degenerations which follow transverse lesions of the cord give rise to the usual symptoms. (See Chapter XX.)

Course and Duration. The course of spinal tumors has been sufficiently indicated. They necessarily lead to complete loss of those functions subserved by the cord below the seat of the lesion. They eventually end in death. The duration is variable, depending upon the nature and seat of the tumor. In tubercular disease death usually takes place in from six to eight weeks from the onset of the symptoms. (Herter.) In other forms of tumor the course is much slower, and the disease may last many months, or even several years (seven or eight years in extreme cases). In one case severe pains persisted for eight years, when death took place, presumably from exhaustion, before even the development of any motor or sensory paralysis. In general, it may be said that when pain is the first symptom, it may, though not always, persist for a long time before the development of symptoms of damage to the cord.

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[1] Later observations of the reflexes in myelitis seem to indicate that the knee-jerks may be abolished in these exceptional cases by inhibition.

The diagnosis of the location of the tumor often presents serious difficulties in the earlier stages of the disease. After transverse softening of the cord has occurred little difficulty will usually be met with, but at this time the practical importance of accurately locating the tumor will have largely ceased to exist in most cases.

Tumors of the meninges cannot be differentiated from tumors of the cord. As has already been explained in describing the symptoms, the distribution and character of the pain afford no aid for this purpose. The location of the tumors in the bone or inter-vertebral substance can be done with success only when there are external signs of deformity. This is easier of detection in the cervical than in the dorsal or lumbar spine. Tumors of the sacrum may often be detected by vaginal or rectal examination. The fact that pain is caused by movement is unreliable as evidence of the location of the growths in the bone. This symptom is quite as likely to be present when the growth is in the membranes. It should always be borne in mind, however, that tumors of the cord are relatively rare, and the chances are greatly in favor of any given tumor being extra-medullary. The vertical location of the tumor can be determined sooner or later, according to the number of symptoms presented. No one symptom is sufficient. The distribution of the pain, although an important sign, cannot alone be relied upon,[1] as it is not possible to distinguish between root pains and cord pains, and, therefore, it cannot be determined whether the growth is pressing upon the roots of the nerves in whose distribution the pain is felt, or upon the cord higher up. In exceptional cases the pain may even be felt in nerves above the tumor. The same uncertainty pertains to the indications offered by spasms. But when pain is felt in several nerve-areas, that corresponding to the highest spinal segment is more likely to be a root-pain and to indicate the position of the tumor. Among the most valuable localizing signs are the upper limit of the anæsthesia, the condition of the reflexes, and the limitation of the atrophy, and the abnormal electrical reactions to certain groups of muscles. The distribution of the paralysis may or may not be of value, according to its combination with other symptoms. The following are the chief peculiarities in the grouping of the symptoms liable to be met with in tumors of different portions of the cord:

In the cervical region rigidity of the neck muscles is common. When the arms are paralyzed careful observation may disclose that certain groups of muscles were affected before the others, and that the atrophy is limited to special muscular groups. For example, a growth in the lower cervical region (seventh and eighth) would cause atrophy with corresponding electrical changes limited to the lower arm[2] group of muscles, even though the body and legs were paralyzed. The shoulder and upper-arm muscles (with the exception of a portion of the triceps) would escape.

A growth in the upper cervical segment would cause atrophy limited to the shoulder and upper-arm group of muscles, though all the nerves of the arms and legs were paralyzed. If the legs are paralyzed before the arms, reliance must be placed upon the increased reflexes, deep and superficial, of the body and legs, and upon the height of the anæsthesia. If, in addition, pain and spasm occur in the distribution of the cervical nerves, the tumor is certainly in some part of the cervical cord. There is retention of urine with overflow or spasmodic incontinence in tumor of the cervical cord.

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[1] For example, in one case, where the tumor was successfully removed, the localization of the tumor must be regarded as due partially to good luck, as, according to the report, the distribution of the pain was largely relied upon, and the operation showed the tumor was above the nerve-roots supposed to be compressed, and that the pains must therefore have been due to pressure on the cord above. The highest localizing pains were in the distribution of the sixth and seventh dorsal nerves on the left side. The tumor was found adherent to the highest root of the left fourth nerve.

[2] The supinator region would probably escape, as its nucleus is in the fourth and fifth segment with that of the biceps groups.

In tumors of the dorsal cord there is paraplegia. The height of the anæsthesia and the reflexes must be the chief guides. The seat of the pain and spasms is of value as indicating a possible lowest limit, as, excepting in rare instances, the pain is never above the tumor. For example, if pain around the waist is present the tumor may be safely placed at the same level or above it. The same is true of abdominal spasm and paralysis. In tumors of the lumbar and sacral segments, we have to guide us the distribution of the atrophy, the condition of the reflexes, deep and superficial, and the paralysis of the sphincters, besides the peculiar distribution of the anæsthesia and paralysis, according to the exact segments compressed. A study of Figs. 195 and 196, pages 616 and 617 will be necessary for this purpose.

The symptoms of tumor of the cauda equina simulate those of the lumbar and sacral segments, and, as a rule, it is difficult to distinguish the one from the other. The rules generally given are inadequate. Only by carefully weighing the symptoms and judging of the probability for and against, can tumors of the two situations be differentiated from one another.

Diagnosis. The diagnosis of spinal tumors must depend upon the march of the symptoms; the more common order in which they appear being pain, spasm, or muscular rigidity, paralysis, and anæsthesia. The diagnosis may be aided by the fact of syphilis being known to be present, or tubercular disease existing elsewhere. The presence of tumors also in other parts of the body would point to a probability of the cord disease being due to a similar growth. The greatest difficulty likely to be met with is when the symptoms do not follow the usual order, but when paralysis appears first, and pain either is not present at all or comes on later. A careful study of all the conditions present will alone enable us to recognize the existence of tumors in such cases. The chief diseases with which tumors are likely to be confounded are, in the early stages, neuralgia, rheumatism, local neuritis, and hysterical neurasthenia; and, later, focal chronic myelitis, subacute myelitis, chronic pachymeningitis, caries and neuritis of the sacral plexus.

When pain alone is present, it may be, and often is, mistaken for that of neuralgia, rheumatism, and local neuritis. In the early stages it is not easy to recognize the true cause of the pain, and probably we shall have to wait until the development of other symptoms. The main grounds for suspicion would be its severity and continuous reference to the distribution of certain special nerve areas. A long-continued neuralgic pain, paroxysmal, and of great intensity, confined to a particular nerve, without evidence of special cause, such as traumatism or disease of the neighboring parts, is sufficient to justify the suspicion of irritation from pressure upon the cord or nerve-roots. Further, in neuralgia and neuritis we are likely to obtain, in the former, the various sensitive points, and, in the latter, tenderness along the course of the nerve. This absence of tenderness in tumor is an important point. The pains of neuralgia and rheumatism are also likely to be diffuse and to involve various nerve areas in different parts of the body.

Neurasthenia with hysterical symptoms sometimes exhibits considerable resemblance to the earlier stages of tumors. There may be long-continued pains, limited to certain nerve areas, and even spasm of muscles and muscular weakness, but careful inquiry will show the predominance of the neurasthenic conditions, the lack of a paroxysmal character to the pains, and often the dependence of these upon temporary emotional conditions. The muscular weakness should be easily distinguished from a real paralysis. The distribution of the pains and the muscular symptoms will not harmonize with any particular spinal localization. The presence of hysterical stigmata in other parts of the body will help to clear up the diagnosis.

In chronic focal myelitis there is much less likelihood of there being spasm or rigidity of the muscles; the pains are less severe and are more likely to be confined to the distribution of the nerves originating from the level of the lesion, while in tumor they are more likely to be found in nerves originating below. It is rare that chronic focal myelitis does not occupy a greater vertical extent of the cord, and hence a careful examination will reveal more extensive disease than occurs with tumor.

Subacute myelitis will only be a source of difficulty when it is secondary to a tumor. In this case a careful inquiry will enable us to recognize antecedent symptoms, referable to the tumor, which have excited the myelitis.

Chronic pachymeningitis, especially the hypertrophic form, is more likely to be confounded with tumor than any other disease. The symptoms in their grouping and development resemble very much those of tumor. It is, however, a rare disease. The chief points to be depended upon for differential diagnosis are that pachymeningitis affects a greater vertical area of the cord, and, as it usually completely surrounds the cord, its symptoms are bilateral from the beginning. The pains are usually due to irritation of the nerve-roots and not of the cord, and the most marked paralysis and anæsthesia are likewise due to the injury of the nerve-roots rather than of the cord. The paresis and other symptoms which are consecutive to pressure upon the cord are usually less prominent, and appear at a later stage than in tumor. The progression of the symptoms showing the successive affection of different nerve-tracts is less characteristic than in tumor.

In caries the pains are much milder, and rigidity of the muscles is rare. It is rare for the disease to have progressed sufficiently far to produce paralysis or anæsthesia without producing some changes in the spinal column, such as thickening of the tissues, or deformity.

Further, when paralysis and other cord symptoms appear, they are due to an incited myelitis, and, therefore, in their development present the clinical picture of that disease rather than of tumor. A careful examination of the spine will generally reveal the nature of the disease, and in doubtful cases treatment, including extension of the spine, would probably clear up the diagnosis.

Treatment. It is only in syphilitic growths that medicinal treatment can be of use. Where there is the slightest reason to suspect syphilis, specific treatment should be pushed with vigor. Even in syphilis, it is only in the early stages that treatment can be of value, because after the cord has been damaged by the pressure of the growth it will be too late to repair the injury; therefore it is desirable to begin specific treatment at the earliest possible moment. The iodides should be pushed, even to heroic doses, and, considering the hopelessness of the disease when left to itself, and of all other treatment, syphilitic treatment should be employed if there is the slightest possibility of previous infection. After the cord has been softened by pressure there is little chance of improvement, even though the gumma should be absorbed.

Surgical treatment alone offers relief in other kinds of growth. The autopsies show that a large number of extra-medullary tumors can be removed. Tumors within the substance of the cord, excepting in rare instances, probably cannot be removed without producing as much damage by the knife as is done by the tumor. Fortunately, however, the greater number of spinal tumors are extra-medullary. In the present state of our knowledge it is not possible to distinguish by the symptoms the two classes, and we can only tell by a surgical examination with which we have to deal. It is therefore desirable in every case, provided the disease has not so far progressed as to have destroyed the cord, to operate as soon as we can diagnose and localize the tumor. Quite a number of operations have been performed; several (Horsley and Gowers, Macewen, Abbe, Ray, Laquer) with successful removal of the tumor and recovery or improvement from the paralysis.[1] Further familiarity with the operation will undoubtedly render it less dangerous, and by operating earlier better results will be obtained.

Considering the hopelessness of every case when left to itself, and the sufferings and helplessness necessarily resulting from the damage to the cord, an operation would always be justifiable, provided the diagnosis were made sufficiently early. But, aside from the question of operation for the prevention of paralysis, or restoration of already damaged functions, in some cases otherwise hopeless, it should be considered whether it may not be justifiable to operate merely for the relief of pain, even when there is no hope of restoring the use of the damaged cord. The pain in some cases is excruciating, and the sufferings of the patient so intense that recourse must be had to continuously increasing doses of morphine. In such cases an operation with a view to removing all source of irritation, even though the cord be destroyed, is one that should be considered. I am inclined to the belief that in such cases an operation would be justifiable, and that by removing an extra-medullary tumor, or by substituting a surgical incision for a compressing tumor lying superficially in the substance of the cord, a relief from suffering, at least, would be given the patient.

All further medical treatment can only be directed toward making the patient comfortable and preventing many of the secondary consequences, such as cystitis and bed-sores. To relieve the pain, resort must be had to anodynes. In the early stages they should be given sparingly, but in the later stages with a free hand. The greatest attention should be given to the bladder to prevent the development of cystitis and its consequences, and the same attention should be employed with a view to prevent the formation of bed-sores. Great attention should be given to the cleanliness of the recumbent parts where exposed to pressure. Means should be taken to avoid the latter. After bed-sores have formed, antiseptics and other means should be used to prevent septicæmia. For further details the reader is referred to the section on Myelitis.

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[1] An interesting article by M. Allen Starr ("A Contribution to the Subject of Tumors of the Spinal Cord, etc.," American Journal of the Medical Sciences, June, 1895), published after these pages had gone to press, contains a collection of 19 cases of operation for the removal of spinal tumors, collected from medical literature. To these the author adds 3 personal cases, making 22 in all. Of these 22 the tumor was successfully removed in 13, while death followed the operation in 9. Of the successful operations, a recovery from the condition of paraplegia occurred in 6. As Starr says, in explanation of these unfavorable results, it should be borne in mind that in most cases the operation was not undertaken until so late in the disease that secondary damage had been caused to the cord. Starr reports 3 other cases without operation. I have been able to include these 6 cases in the total number of cases (133) analyzed for the purpose of this chapter.

CHAPTER XX.

DISEASES OF THE SPINAL CORD.

(CONTINUED.)

BY FREDERICK PETERSON, M.D.

STRUCTURE AND FUNCTIONS OF THE SPINAL CORD AND LOCALIZATION.

THE spinal cord, while it is a sort of cable carrying messages to and fro between the brain and the body, is at the same time to be looked upon as a series of thirty-one segments, piled one upon the other, each segment being connected with a pair of spinal nerves, and each representing a trophic, reflex, and vasomotor centre. These thirty-one segments occupy only seventeen or eighteen inches of the spinal canal, which is twenty-six to twenty-eight inches long, so that the spinal canal is some nine or ten inches longer than the cord. Thus the first segment of the cord is at the foramen magnum, and the thirty-first (or conus) is opposite the base of the first lumbar vertebra. The intraspinal course of the nerves after they emerge from the different segments is longer the lower we descend the cord, so that the pairs from the lumbar and sacral segments, for instance, form a large bundle (cauda equina) which occupies the remaining space in the spinal canal below the termination of the cord itself. If we take a cosmetic pencil it is very easy to draw on a thin person an outline of the spinal cord upon the back over the vertebral column, and in this way one obtains a clearer knowledge of these puzzling relations than by any amount of reading. (Fig. 190).

The eighth cervical and first dorsal segments lie immediately in front of the spine of the seventh vertebra. The lumbo-sacral enlargement of the cord begins just in front of the tip of the spine of the tenth dorsal vertebra. The conus lies at a level with the lower point of the first lumbar spine. Below this the canal is filled with the cauda.

From a pathological standpoint the most important parts of the spinal cord are the cervical and lumbar portions, or enlargements, the cervical portion being enlarged because it contains the sensory, motor and trophic centres for the arms (as well as the cilio-spinal, wrist-jerk, elbow-jerk, scapular, palmar, and hypochondrium reflex centres); the lumbar portion, because it contains similar centres for the legs (as well as the reflex centres for the bladder, rectum and sexual organs).

The cord consists of " columns " of white matter surrounding the H-shaped central gray matter. The columns, or tracts, contain the long fibres which carry peripheral and centripetal impulses and are variously named as in the diagram. (Fig. 192.)

The anterior columns are often called the columns of Türck, and contain motor fibres from the cortex. The posterior are designated as the columns of Goll and Burdach, the former nearest the median line, carrying tactile impressions upward to the brain. The lateral columns are made up chiefly of the long motor fibres from the cortex, the crossed pyramidal tracts. External to this on the periphery of the cord lies the direct cerebellar tract (carrying muscular sense impressions); and anterior to this also on the periphery of the cord is found Gowers's tract (conveying pain and temperature sensory impressions upward to the brain). These are the most important of the tracts or columns. What is known as Clark's column is a column of cells in the lower dorsal and upper lumbar cord (eighth dorsal to second lumbar nerves) lying in front of the column of Burdach in the inner part of the neck of the posterior horn of gray matter; and the Lissauer tract is a very small bundle of fibres near the periphery of the cord about the posterior extremity of the posterior horn.

FIG. 190.

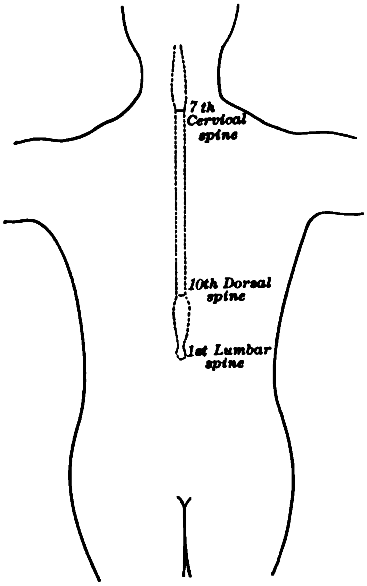
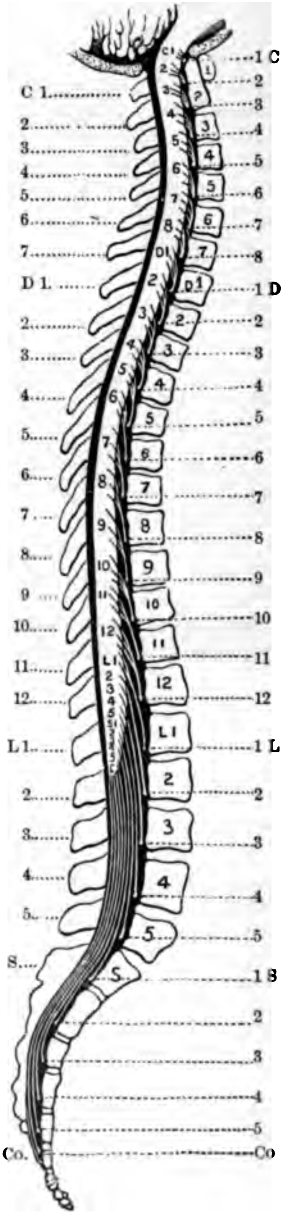


FIG. 191.

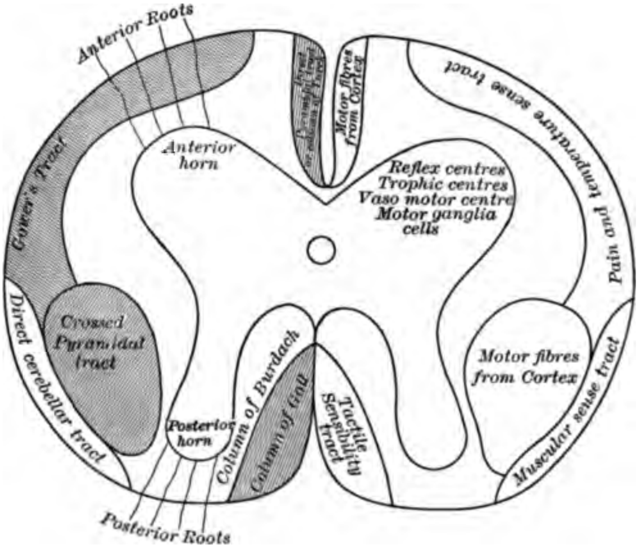


The mutual relations of the vertebral bodies and spines to the segments in the cord and to the exit of the nerves. (GOWERS.)

The reflex, trophic and vasomotor centres are wholly in the H-shaped central gray matter of the cord.

The white matter varies in amount at different levels, and diminishes in quantity from above downward. The gray matter varies also in shape and amount at different levels, being largest in the cervical and lumbar intumescences, especially the latter. To the practical physician a knowledge of the structure of the anterior horns of this gray matter is particularly serviceable. These contain the great motor ganglion cells connected on the one hand with fibres coming down from the motor area of the brain through the crossed pyramidal tract and column of Türck, and on the other with the fibres going out by the anterior roots to the muscles. Volitional impulses to the muscles pass through them, and they are also the trophic centres for the muscular fibres. These cells are arranged in groups or clusters in each anterior horn, but the arrangement varies at different levels of the cord. Thus in the following diagram of one anterior horn (Fig. 193) we see median, anterior, antero-lateral and postero-lateral groups.

FIG. 192.



Names of *tracts* in left half, of *functions* in right half, of diagram.

FIG. 193.

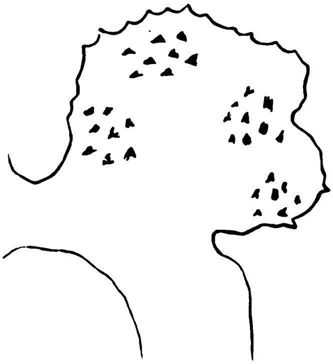


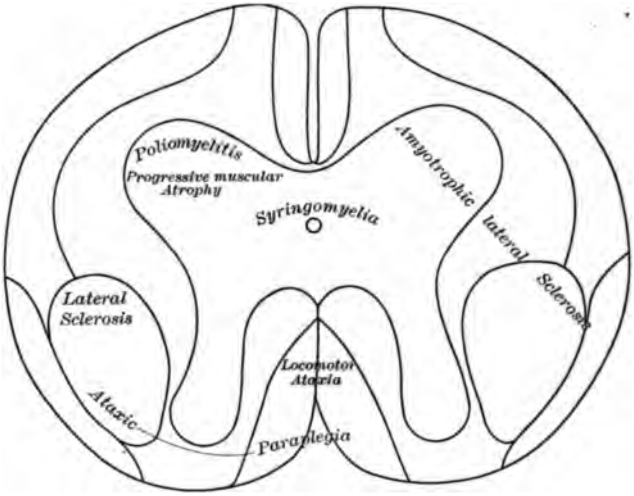
Diagram of clusters of ganglion cells in an anterior horn. Sometimes there is a central cluster. Doubtless these groups represent certain groups of muscles associated in function, but accurate knowledge is still wanting on this point.

In addition to our consideration of the cord as a whole with long tracts of ascending and descending fibres, and as a series of segments one upon the other, we must also remember that, like the brain, it consists of two symmetrical halves, with sensory fibres crossing from one side to the other throughout the whole extent, and fibres uniting the anterior columns and the anterior horns of opposite sides.

There are some diseases which attack certain columns in the cord, or certain portions of gray matter, and these are termed system diseases. These system-diseases, such as poliomyelitis, progressive muscular atrophy, amyotrophic lateral sclerosis, lateral sclerosis, ataxic paraplegia, and locomotor ataxia, are readily recognized by the peculiar symptoms brought about by injury to certain systems of fibres or cells. The localization of such pathological conditions is quickly apparent by a glance at Fig. 194.

The so-called focal lesions of the cord, lesions of limited extent in a particular segment or segments, due to neoplasms, pressure, hemorrhage, inflammation, spinal injuries, and the like, are not so readily recognized, for the symptoms produced (reflex, trophic, sensory, motor) are extremely variable, depending as they do upon the segment level of the lesion. Each separate segment of the cord has its own particular functions. For instance, the reflex centres for the bladder and rectum are situated in the fourth and fifth sacral segments and in the conus; the ankle-clonus in the fifth lumbar segment; the knee-jerk in the third lumbar; the wrist and elbow-jerks in the sixth cervical, and so on. Motor and trophic functions are distributed in the same manner, segmentally; so that, for instance, the intrinsic muscles of the foot are represented in the first and second sacral segments, knee flexors in the fourth and fifth lumbar, thigh muscles in the second and third lumbar, abdominal and spinal muscles in the second to the twelfth dorsal, thumb, and finger muscles in the eighth cervical and first dorsal, forearm muscles in the sixth and seventh cervical, shoulder muscles in the fourth and fifth, and neck muscles in the second and third. The apparent principle of the arrangement is seen to be that the lowest muscles in the body are represented in the lowest segments in the cord. The sensory distribution is also segmental, and has peculiarities of great diagnostic value. Thus, anæsthesia of the perineum, scrotum, rectum, vagina, and posterior surface of the penis is produced by a lesion in the lowest part of the cord, viz.: in the fourth and fifth sacral segments and conus. If the lesion is higher, say at the third sacral segment, the anæsthesia extends further out upon the buttocks and downward over the back of the thighs, over parts, in fact, which are touched by the saddle in riding (hence the so-called saddle-shaped area). If the transverse lesion be as high as the fifth lumbar segment, the anæsthesia includes, in addition to the preceding area, the outer sides of both legs, and even at times the outer surfaces of the feet. The

FIG. 194.



A diagram of the topographical situation of certain pathological processes in the cord.

higher up the lesion, the greater the area of anæsthesia. The lower part of the surface of the abdomen is not anæsthetic until the first lumbar segment has been implicated, though before this segment is reached all of the lower extremities and buttocks have been included in the anæsthetic areas. Above the first lumbar segment the anæsthetic areas are limited by circles about the trunk. The umbilical region is represented in about the tenth dorsal segment. When the eighth cervical segment is affected by a transverse lesion, the anæsthetic area is complete over all parts below a girdle about the nipples, and the inner sides of the arms and ulnar areas of the forearms and hands are included. Thus, from a careful study of the segmental representation of muscles, reflexes, and sensibility, we are enabled to arrive at a very accurate diagnosis of the exact position of a focal lesion; but it must always

FIG. 195.

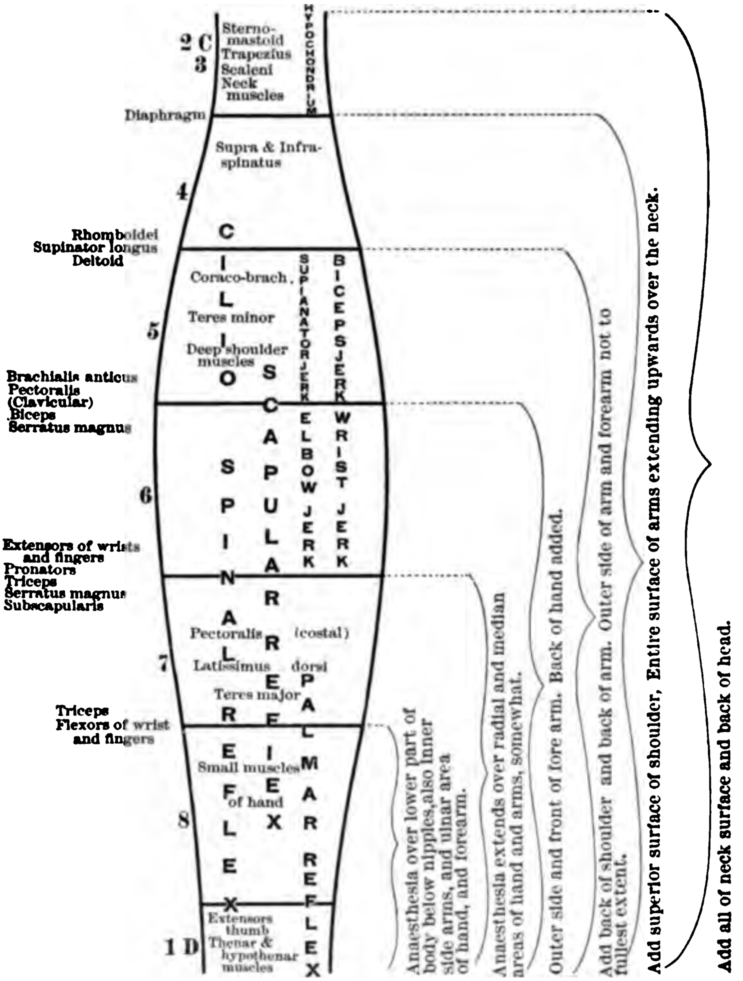

2 C
3
Sterno-mastoid
Trapezius
Scaleni
Neck muscles
HYPOCHONDRIUM
Diaphragm
Supra & Infra-spinatus
4
Rhomboidei
Supinator longus
Deltoid
CILIO SPINAL
Coraco-brach.
Teres minor
Deep shoulder muscles
SUPINATOR JERK
BICEPS JERK
5
Brachialis anticus
Pectoralis (Clavicular)
Biceps
Serratus magnus
SCAPULAR REFLEX
ELBOW JERK
WRIST JERK
6
Extensors of wrists and fingers
Pronators
Triceps
Serratus magnus
Subscapularis
Pectoralis (costal)
Latissimus dorsi
Teres major
7
Triceps
Flexors of wrist and fingers
PALMAR REFLEX
Small muscles of hand
8
Extensors thumb
Thenar & hypothenar muscles
1 D
Anaesthesia over lower part of body below nipples, also inner side arms, and ulnar area of hand, and forearm.
Anaesthesia extends over radial and median areas of hand and arms, somewhat.
Outer side and front of fore arm. Back of hand added.
Add back of shoulder and back of arm. Outer side of arm and forearm not to fullest extent.
Add superior surface of shoulder, Entire surface of arms extending upwards over the neck.
Add all of neck surface and back of head.

Diagram of cervical enlargement of the cord, showing the segmental representation of muscles, reflexes, and sensory areas.

be remembered to study in connection with segmental symptoms the effects upon the great tracts running through these segments between the brain and periphery. In connection with the facts just stated I reproduce here the diagrams of the cervical and lumbar enlargements first published by me in the New York *Medical Record*, November 12, 1892, and which many physicians and students have found practically useful as an aid in diagnosis (Figs. 195 and 196) as well as a help to the memory. As regards the dorsal portion of the cord, which it is not so important for some reasons to study as the two enlargements just described, it may be stated that the nerves represented in the second to twelfth dorsal segments are the intercosto-humeral, intercostals and dorsal posterior nerves; the muscles, those of the back and abdomen, and erectores spinæ, and the reflexes, the epigastric (fourth to seventh segments), and the abdominal (seventh to eleventh segments).

FIG. 196.

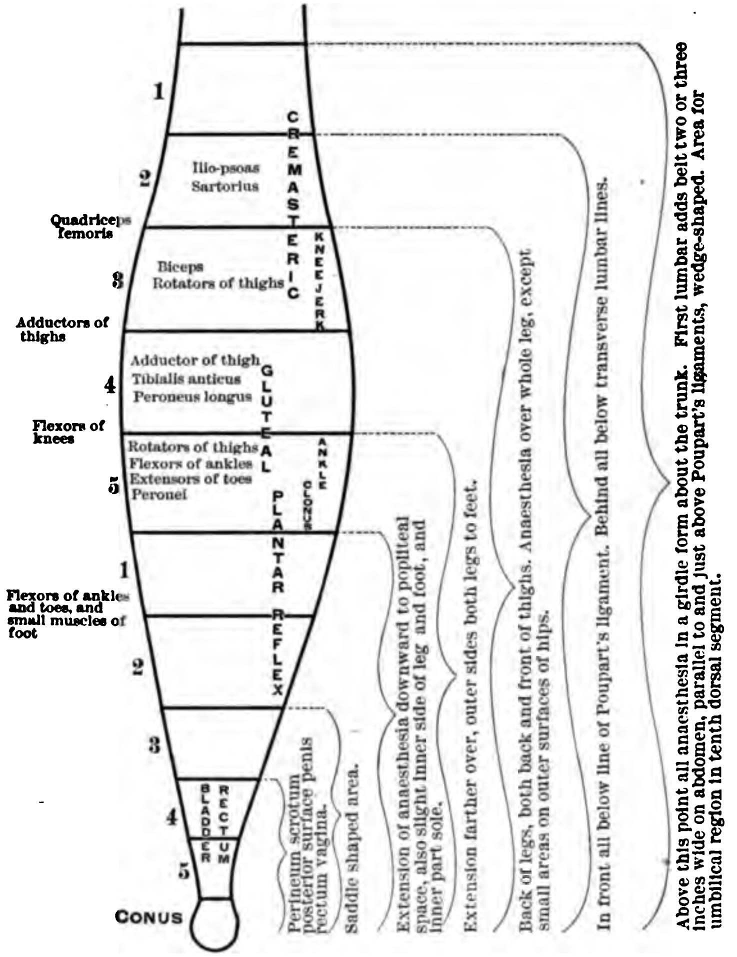

1
2
Ilio-psoas
Sartorius
Quadriceps femoris
3
Biceps
Rotators of thighs
Adductors of thighs
4
Adductor of thigh
Tibialis anticus
Peroneus longus
Flexors of knees
5
Rotators of thighs
Flexors of ankles
Extensors of toes
Peronei
1
Flexors of ankles and toes, and small muscles of foot
2
3
4
5
CONUS
CREMASTERIC
KNEE-JERK
GLUTEAL
ANKLE CLONUS
PLANTAR REFLEX
BLADDER
RECTUM
Perineum scrotum posterior surface penis rectum vagina.
Saddle shaped area.
Extension of anaesthesia downward to popliteal space, also slight inner side of leg and foot, and inner part sole.
Extension farther over, outer sides both legs to feet.
Back of legs, both back and front of thighs. Anaesthesia over whole leg, except small areas on outer surfaces of hips.
In front all below line of Poupart's ligament. Behind all below transverse lumbar lines.
Above this point all anaesthesia in a girdle form about the trunk. First lumbar adds belt two or three inches wide on abdomen, parallel to and just above Poupart's ligaments, wedge-shaped. Area for umbilical region in tenth dorsal segment.

Diagram of the lumbar enlargement of the cord, showing the segmental representation of muscles, reflexes, and sensory areas.

In the diagrams I have mapped out the various segments in such a way as to show the muscles represented in each, and in vertical letters the levels of the reflexes. The areas of anæsthesia produced by transverse lesions are shown in the brackets at the right, corrected from my first diagrams according to the recent studies of Thorburn (*Brain*, Autumn, 1893). Lesions of the cauda equina may give rise to symptoms very much resembling those of injury to the lumbo-sacral enlargement, because the cauda is composed of nerve-trunks derived from this part of the cord; but it is to be remembered that in cauda lesions there are comparatively extensive paralyses, while sensation is but slightly affected.

There are chronic lesions in the central gray matter of the cord, such as syringomyelia, which produce peculiar symptoms according to the segmental level of the lesion. Encroaching on the gray matter of the anterior horns, of course, the particular muscles of that segment are paralyzed and atrophied, and lose their reflex excitability. Encroaching around the central canal and toward the posterior horns, a peculiar anæsthesia is produced to pain and temperature, distributed, of course, according to the particular representations of skin area at the level of the lesion. This is because the fibres for pain and temperature sensibility lie in the central gray matter before entering their tract on the periphery of the cord (see Fig. 192) to reach the brain

Lesions of the motor tracts in the white matter of the lateral and anterior columns produce muscular weakness, or paresis, but without atrophy. This paralysis is naturally much more widespread than the paralysis caused by injury to the anterior horn. For instance, a very small lesion, say the size of a pea, in a lateral column will probably affect all of the muscles on the same side below that point, the lateral tract being the cable containing nearly all of the long fibres of communication between one side of the body and one-half of the brain. On the other hand, the same lesion in an anterior horn would produce a very restricted paralysis, affecting only the muscles innervated from that particular segment.

Another feature peculiar to paresis from lateral column injuries is the spastic rigidity with exaggerated tendon reflexes. Still another important point for differential diagnosis is the loss of faradic reaction in paralysis, due to lesion in the anterior horn or motor nerve, and the perfectly normal reaction of these muscles to faradism when the paralysis is due to lesion in the long fibres running from the cortex down the lateral and anterior columns to terminate in the large cells of the anterior horns.

Hence there are four absolutely opposite clinical conditions or symptoms which will serve to differentiate for us the two distinct types of spinal paralysis, the atrophic and the spastic; and these may be conveniently grouped as follows:

Atrophic Spinal Paralysis.	*Spastic Spinal Paralysis.*
1. Atrophy.	1. No atrophy.
2. Lost reflexes (such as knee-jerk, wrist-jerk, elbow-jerk, etc.).	2. Exaggerated reflexes.
8. Flabby relaxed muscles.	3. Rigid muscles.
4. No contraction to faradism. Lesion in the anterior horns.	4. Contraction to faradism. Lesion in the lateral columns.

This calls to mind the importance of a complete understanding of the knee-jerk by the general practitioner.

It is of the highest value among the reflex symptoms. The tap upon the tendon sends a sensory impulse to the reflex centre in the anterior horn of the third lumbar segment (*via* the sensory nerve from the knee-area and posterior root and posterior horn), whence an impulse is reflected outward (*via* the anterior root and motor nerve) to contract the quadriceps extensor. While the knee-jerk is, in truth, not a perfect representation of an actual reflex, yet for all practical purposes it may be so considered. (See also p. 27 *et seq.*)

Now, certain fibres from the cortex pass down by way of the lateral columns of the cord to enter the anterior horns, and these hold a rein upon the reflexes, so to say. They inhibit the jerk. Thus, under normal conditions, the knee-jerk is very moderate. If this inhibitory fibre is functionally impaired or destroyed the knee-jerk is no longer restrained, but becomes exaggerated. If any part of the reflex arc is destroyed (sensory nerve, posterior nerve-roots, posterior root-zone, posterior horn, anterior horn, or motor nerve), the knee-jerk is lost. Thus cerebral palsies, lateral sclerosis, exaggerate the knee-jerk; whereas neuritis, locomotor ataxia, poliomyelitis, and the like, destroy the knee-jerk. Fig. 197 is offered as a diagrammatic illustration of these points.

FIG. 197.

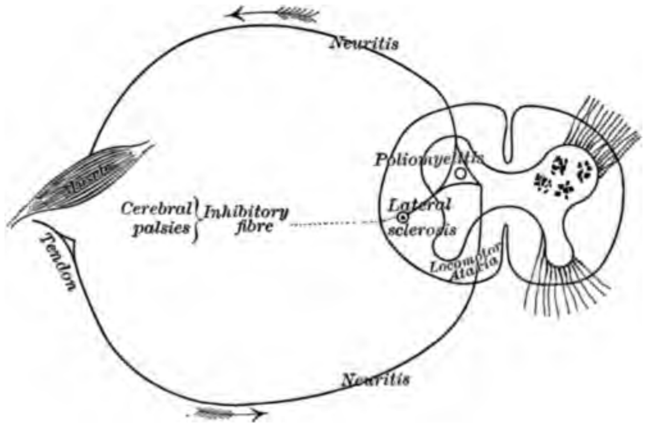


Diagram showing reflex arc and inhibitory fibre, and some of the diseases which exaggerate or destroy the knee-jerk.

In lesions affecting the inhibitory fibres anywhere all the tendon reflexes below the lesions become exaggerated. Thus ankle-clonus, not normally present, develops when the lateral column is affected above its centre in the fifth lumbar segment.

A unilateral lesion of the spinal cord gives rise to an interesting syndrome, generally termed Brown-Séquard's paralysis. Suppose that one lateral half of a segment of the mid-dorsal region is destroyed. The result seen in a typical case some time after the injury would be:

Same Side.	*Opposite Side.*
1. Atrophic paralysis of muscles innervated by that particular half-segment.	1. Loss of pain, temperature, and touch-sense below the lesion.
2. Spastic paralysis of the leg.	
3. Lost or impaired muscular sense.	
4. Exaggerated knee-jerk and usually ankle-clonus.	
5. At about the level of the lesion a half-girdle of hyperalgesia, and just below this a parallel zone of anæsthesia.	

The wrist-jerk, under normal conditions rarely present or just perceptible, becomes very excessive in lesions of the cerebro-spinal segment of the motor tract above its spinal centre in the sixth cervical segment.

In the earliest stage of an acute unilateral lesion of the cord the knee-jerk may be absent for a short time, which is also true of both knee-jerks in any complete transverse lesion above the third lumbar segment, but subsequently it takes on its overactive character. This is probably due to the fact that in an acute lesion there is irritation of the inhibitory fibres.

The reason that the muscular sense is impaired on the same side in a unilateral lesion is that the fibres for muscular sense do not decussate along the spinal cord as do the other sensory fibres.

In addition to the reflex centres already indicated, there are others whose precise localization has not yet been determined. For instance, there are the parturition, erectile, menstruation, and ejaculatory centres, and these are undoubtedly in the lumbar enlargement, for some of them have been known to be destroyed, and their functions lost, by lesions in this part of the cord.

The Membranes of the Cord. As is the case with the brain, three membranes enclose the spinal cord. The outer or dura mater is dense and fibrous, and separated from the bony canal by fat and a large plexus of veins. It is slightly attached by its anterior surface to the vertebral canal. There is therefore an epidural space. Filaments from the spinal nerves supply the dura with sensory nerves. The subdural space contains a small amount of serum. The middle membrane or arachnoid is completely separated from the inner or pia mater in the spinal canal, which is not the case in the cranial cavity. The pia sends septa into the cord.

The Blood-supply of the Cord. The cord receives its blood from branches of the vertebral, intercostal, and other arteries which enter with the spinal nerves and reach the cord along the anterior and posterior roots. Anterior branches pass to the anterior median fissure to form there the vertical continuous anterior spinal artery, from which a series of branches enter the fissure to supply most of the gray matter. Posterior branches enter the posterior fissure in a somewhat similar manner. Twigs are given off everywhere from the pia on the periphery of the cord to the white substance. The veins have very much the same distribution. There is a vertical continuity of bloodvessels both within and outside of the cord, though it is probable that the circulation is horizontal to a very great extent. The tortuosity of the arteries and the size of the venous plexuses serve to prevent overdistention, rupture, and the dangers of high blood-pressure.

POLIOMYELITIS.

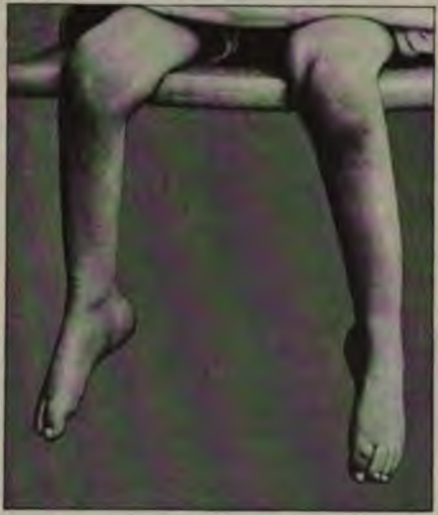
Poliomyelitis Anterior; Infantile Spinal Paralysis; Acute Atrophic Paralysis; Essential Paralysis of Children.

Poliomyelitis is a disease of the anterior horns of the spinal cord associated with paralysis and wasting of muscles, usually acute in onset, and accompanied with fever and other symptoms of general disturbance. The process is generally *acute*, but may be *subacute* and more rarely *chronic*. It may occur at any age, but is commonly a disease of childhood.

Etiology. The two sexes are about equally afflicted—more than one-half of the cases occur during the first three years of life. Ten times as many have their onset in the first decade as in all the others put together. After the age of forty years it is extremely rare. Heredity seems to have little if any influence upon the disease. Sinkler pointed out that it is more frequent in summer than in winter. Exposure to draught, overexertion in walking, infectious diseases, and falls have been given as antecedent causes, but it is doubtful if any of these are important factors. Most of the cases are suddenly attacked while in good health. In some instances a sort of epidemic character has been noted. By the best authorities it is now supposed itself to be a species of infectious disease, though nothing conclusive as to this has been determined.

Symptomatology. The onset is not sudden, though usually very rapid. As a rule, there is fever, and with it restlessness, headache, anorexia, general weakness, and at times vomiting and diarrhœa. There is widespread paralysis or paresis at first, though later on the majority of the muscles recover, leaving one or two limbs, often only a single group of muscles, permanently paralyzed and wasted. The constitutional symptoms often last but a few hours, but are occasionally protracted for several days. Rarely they are so slight as to escape notice. The paralysis is apt to be observed early in the attack, but may be overlooked if the general symptoms are severe. Pain referred to the limbs or back, and sometimes even muscular tenderness, is apt to be present. Convulsions may occur, and cases have been known to simulate to a certain extent cerebro-spinal meningitis. Incontinence of urine is rare. Very infrequently the onset may be such as to lead to the suspicion of spinal hemorrhage. In a few days usually the disorder has reached its height and improvement begins. But this period may be protracted for weeks. In from one to three months recovery has taken place in all but the parts which are to remain permanently disabled, and in these flaccidity and wasting are marked. At the end of a week from the onset the loss of faradic contractility in the most affected muscles will be noted. The deep reflexes are always absent in the paralyzed limbs, although with recovery in these parts there is a return of the reflexes. Cutaneous sensibility is almost never disordered. Retardation of growth of the whole member disabled occurs, so that as the child increases in stature the paralyzed limb does not keep pace with the others. Besides wasting of muscles and stunted growth of bones, the joints are often relaxed and singularly mobile through want of support by the tendons of paralyzed muscles passing over them. Subluxations of such joints are not uncommon. Permanent contractures often take place in normal opposing muscles, thus producing deformities. These are especially

FIG. 198.

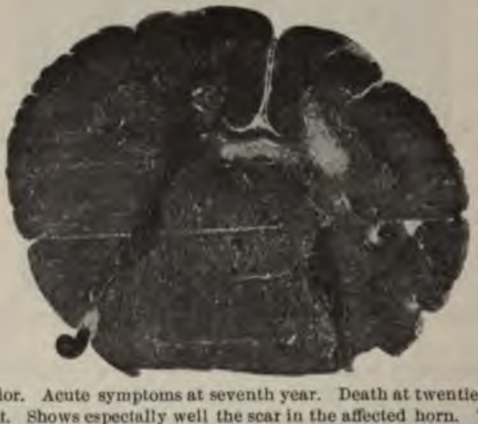


Showing atrophy and foot-drop in poliomyelitis of childhood, infantile paralysis. (Jefferson Hospital.)

common in the foot, where we have many varieties of club-foot, such as talipes calcaneus, talipes varus, talipes valgus, talipes cavus, talipes equinus, and talipes equino-varus. The permanent paralysis is generally in one of the legs. In sixty cases, of which I have notes, the leg below the knee was affected in 21, the leg and thigh in 15, both legs and both thighs in 11, one arm in 6, one leg and one arm in 5, and all four extremities in 2. The anterior tibial group is paralyzed most frequently of all. The left leg is twice as commonly affected as the right. There are few muscles of the body that have not been involved in some reported cases of poliomyelitis. Shoulder and trunk muscles, and even those of the face, have been known to be implicated. Weakness of the spinal muscles may cause spinal curvature. I have seen one instance in which the abdominal muscles of the left side were so paralyzed and wasted that the wall was very thin and the abdominal contents bulged out markedly to that side. Universal paralysis may cause death in the early stages, but death from poliomyelitis is very rare, though possibly cases fatal in the first day or two may go undiagnosticated. Relapses and second attacks are almost unknown. Possibly poliomyelitis creates a vulnerability of the spinal cord to sequelæ, such as progressive muscular atrophy and lateral sclerosis, for several such cases have been reported. The course of the disease in adults and children is much the same. In the subacute and chronic forms the general disturbances are naturally of a mild type or are absent altogether.

Pathology and Pathological Anatomy. The systemic disturbance found in most cases of poliomyelitis points to a blood-state, probably toxic in nature and due to a germ. The predilection shown by this toxæmia for the anterior horns of the cord is analogous to that exhibited by other poisonous agents for other portions of the nervous system. A sufficient number of autopsies have been made in the earliest stages to establish the fact that the disorder in the cord is inflammatory in character and that the inflammation may be either parenchymatous or interstitial. There are cases in which the inflammation extends further than the anterior horns, for meningeal and cerebral symptoms and peripheral neuritis have been observed in conjunction with this disease. In cases described there have been found congestion of the capillaries, with minute capillary ectasias in the gray matter, swelling of the ganglion cells and neurogliar elements, and infiltration of leucocytes.

FIG. 199.



Poliomyelits anterior. Acute symptoms at seventh year. Death at twentieth year. Seventh cervical segment. Shows especially well the scar in the affected horn. Weigert's stain.

Adjacent nerve-fibres may undergo degeneration. Sometimes only the ganglion cells are found affected. In later stages, months or years after recovery from the acute disorder the anterior cornu of the affected side is found shrunken, and its nervous elements replaced by connective tissue or shrivelled and diminished in number. Where the inflammation had evidently extended into neighboring parts, such as the antero-lateral or lateral columns, such parts are lessened in size and may show sclerotic changes. The affected half of the cord is smaller than the normal half, and the gross changes are visible to the naked eye. Descending degeneration is to be seen in the motor nerve-fibres corresponding to the segment or segments concerned. The muscular fibres innervated by such nerves undergo degeneration and ultimately disappear in severe types of the disease. Not all of the muscular fibres of any one muscle are affected, and often normal and even hypertrophied fibres are found in the diseased muscles.

Diagnosis. Error in diagnosis is very easy in the early stages of an acute attack. I have myself seen a case in which cerebro-spinal meningitis was diagnosticated by an excellent practitioner, and it was not until the subsidence of the severe symptoms and the appearance of a monoplegia with wasting that the true nature of the malady became known. Fever, vomiting, and diarrhœa, rheumatic pains and the like are concomitants of a great many other disorders, so that their presence has no particular significance. Widespread paresis is a more important indication, and when the early stage is past and the local paralysis remains, with evidence of atrophy of the member and reaction of degeneration in the muscles, the diagnosis is clear. Acute transverse myelitis is not found in children, and when it occurs in adults is almost always in the dorsal portion of the cord and not in the cervical or lumbar enlargement, as is the case with poliomyelitis. The absence of anæsthesia and other sensory disturbances should be evidence in favor of the latter disease. The differential diagnosis between peripheral neuritis and poliomyelitis is often difficult in adults, and sometimes, indeed, impossible, owing to the occasional prominence of sensory symptoms. Cerebral palsies are readily distinguishable because in these we have increased rather than lost reflexes, never reaction of degeneration, and never extreme muscular atrophy. There should never be confusion with spondylitis, pseudo-hypertrophic paralysis or spastic spinal paralysis, if the symptomatology of the poliomyelitis is carefully borne in mind. Congenital weakness of muscles or rickety conditions may in early infancy simulate poliomyelitis, but in these conditions the normal electrical reactions and normal reflexes should make the distinction apparent.

Prognosis. When the first period of the disease is past the prognosis as regards life is wholly favorable. In the acute stage death may occur through the involvement of important nervous centres or of muscles of respiration. While undoubtedly undiagnosticated cases do die in early stages, the vast majority of cases of poliomyelitis recover from the immediate effects, unless the malady is complicated with pulmonary or other visceral disease. The question will arise as what element of the disorder will be left as a permanent condition. It may be stated that the faradic current will aid us in this particular. If after a week or ten days we find faradic contractility lost in a group or groups of muscles, we may safely say that they will remain for a long time, if not permanently, paralyzed. Some of them will certainly never recover. If the degenerative reaction is late in appearing the probability is that the muscles may recover in part, if not wholly. The retardation of growth in the affected member in children must not be forgotten, and the results of such condition and the effects of contractures should be described to the friends.

Treatment. In the acute stages perfect rest is requisite. The patient should lie on the side. If the fever is high and there are cerebral symptoms an ice-bag should be applied to the head, and cool ablutions should be given in bed. A little counter-irritation in the way of sinapisms to the back may be used, but if pain in the back is severe hot fomentations will afford the most relief. When there is a tendency to nervous excitement, delirium, or convulsions, chloral and bromide should be used. Calomel, a half to one grain, every two or three hours, is indicated as an intestinal derivative, and drop doses of tincture of aconite every half-hour should be administered for the febrile condition. At the end of three weeks after the acute symptoms have subsided tonics are needed, such as iron, quinine, and strychnine, and electrical treatment may be begun, though at first with moderation. We do not expect to restore nerve and muscle fibres which are destroyed, but with the galvanic current we can stimulate the partially degenerated tissues and improve the functions of such fibres as have been left uninjured by the disease. The faradic current is of course useless in the treatment of muscles which do not contract by its use. One pole may be placed in the hand or over the spine (a matter of indifference), and the other pole, in the shape of a sponge electrode well wet in warm salt water, should be stroked over the paralyzed muscles. It is preferable to use the anode for this purpose. The current should be strong enough to produce a contraction of the muscle with each stroke. In this way not only is the paralyzed muscle exercised, but tissue metabolism is influenced by the dilatation of the bloodvessels induced and also undoubtedly by the cataphoretic property of the galvanic current. The treatment should be given for ten minutes every other day, and later every day. A nurse can easily be taught the method of application. With a child it is best to begin with scarcely perceptible currents for a few days in order not to alarm it needlessly. Massage in the shape of effleurage and kneading of the muscles is a necessary part of the treatment. It should be given daily, at bedtime, for ten to fifteen minutes. It is best to rub upward. Cocoa butter or vaseline may be used in the rubbing. With the return of any degree of muscular power systematic exercise must be regularly carried out. Where there is tendency to contractures in opposing muscles, these should be treated by massage, stretching exercises, and by orthopedic devices. The sooner a child with poliomyelitic deformity, or tendency to deformity, is properly treated by orthopedic measures the better, and orthopedic apparatus must be constantly adjusted and readjusted to suit the improving condition and to adapt it to the growth of the child.

SUBACUTE AND CHRONIC POLIOMYELITIS. This subject hardly requires a separate heading, as the adjectives used imply that these forms differ chiefly from the acute disorder in the comparative slowness of onset and in the mildness of constitutional symptoms. Subacute types are such as require from a few weeks to a month for development, chronic types from a month to six months or so. The chronic variety is relatively exceedingly rare, and is often difficult to distinguish from multiple neuritis and progressive muscular atrophy. While subacute poliomyelitis may develop in childhood as well as in advanced age, the chronic form is almost wholly found in adults. The absence of pain, anæsthesia, and tenderness should serve to distinguish these conditions from mulitiple neuritis. They should be differentiated from progressive muscular atrophy by their rapidity of onset as compared with this very slowly developed disease; by the appearance of paralysis in both before the atrophy; by the want of fibrillary contractions; by the very early loss of faradic contractility; and, finally, by the failure to progress, for usually chronic poliomyelitis becomes stationary after a few months, and then improvement begins, and recovery may even ultimately ensue. There are cases of this type, however, that have a fatal issue by extension to important muscular groups.

As to *treatment*, electricity is of considerable value, and should be employed as described above. Tonics, and particularly the hypodermic use of strychnine, are indicated.

CHRONIC SPINAL MUSCULAR ATROPHY.

Progressive Muscular Atrophy; Wasting Palsy.

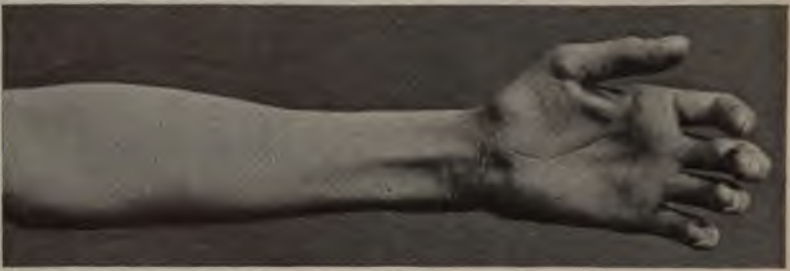
There are two classes of progressive disease, both marked especially by muscular wasting, but each having a different pathological basis. In one the disease is of spinal origin and due to the gradual disappearance one by one of the trophic ganglion cells of the anterior horns. In the other the disease is primarily muscular and produced by the gradual degeneration and disappearance of the muscular fibres. To the first category belongs the commonest form of progressive muscular atrophy—the type of Aran-Duchenne—which it is my purpose to describe here. To the second group of primary muscular dystrophies belong pseudo-hypertrophic paralysis, Erb's juvenile form and the type of Landouzy-Déjerine, described elsewhere in this volume.

Aran and Duchenne were the first to fully describe the disorder considered here (1849–50). Aran gave it the name of "progressive muscular atrophy." These men did not, however, understand its pathology, and it remained for others to discover that it is a spinal-cord disease. There are cases in which the disorder of the anterior horns is combined with sclerosis in the adjacent lateral columns, and to this the name amyotrophic lateral sclerosis has been given. Occasionally the anterior cornual disease is associated with sclerosis of the posterior columns, and we have the phenomenon of a combination of tabes and progressive muscular atrophy. But as the anterior-horn disease is often encountered singly, it seems best to treat of the Aran-Duchenne type of progressive muscular atrophy only in this article, leaving amyotrophic lateral sclerosis for a subsequent section.

Etiology. Heredity plays little or no part in the spinal form of progressive muscular atrophy, whereas it is a common factor in the primary muscular dystrophies. Three times as many males fall a prey to this disease as females. It is a disease of adult life (twenty-five to forty-five years), but cases have been reported as early as the age of twelve and as late as seventy. Mental strain, exposure, spinal concussion, syphilis, overuse of muscles, lead-poisoning, and infectious diseases have been mentioned among causes by various authors.

Symptomatology. In a typical case the disease begins by a wasting of the thenar and hypothenar muscles in one hand, especially the right. (See Fig. 200.) It may begin in both at the same time, or the shoulder muscles may be the first to exhibit atrophy. In nine-tenths of the cases the onset is in the upper extremities, but in the remaining tenth it begins in the lower limb. Some pain or paræsthesia may precede the wasting of the muscles. The disease is extremely slow and progressive, and is always a motor and trophic disorder, never accompanied with anæsthesia. The trophic disturbance is limited to the muscles and never affects the bones or skin. The wasting spreads from the parts first affected to other muscles; for instance, from the ball of the thumb to the interossei and lumbricales, then to the flexors in the forearm and extensors, and so on up to the limb; but frequently a leap is made from the intrinsic muscles of the hand to the deltoid and other shoulder muscles. Occasionally the upper arm and shoulder group are the first affected, while

FIG. 200.



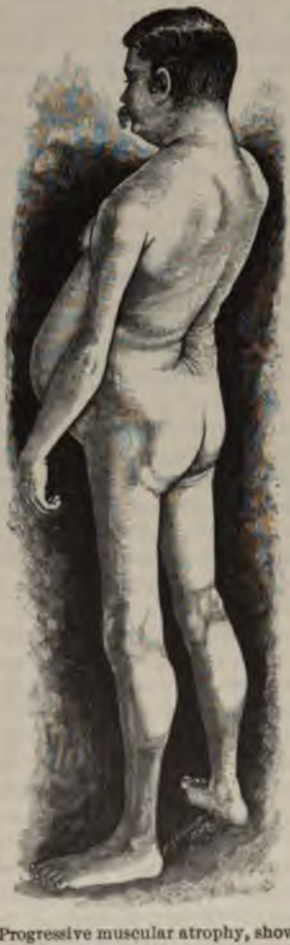
Hand and forearm in chronic spinal muscular atrophy, showing especially wasting of thenar and hypothenar eminences.

the forearm and hand escape for a long period. Muscles of the back are prone to take part in the wasting at an early stage, and lordosis is not an uncommon deformity in this disease. (Fig. 201.) The muscles supporting the head may become so affected that the head falls forward upon the chest, or is carried far backward as if balanced upon the vertebral column. Sometimes only portions of muscles atrophy, such, for instance, as the anterior half of the deltoid and the lower part of the trapezius. There is generally a peculiar escape of the upper bundles of the trapezius, so striking that Duchenne designated them as *ultimum moriens*. As the disorder progresses the muscles of respiration become involved, then of the lower part of the back, of the hips, and of the thighs. The disease gradually ascends to other groups of ganglion cells in the cervical enlargement, and even upward into the medulla, so that the diaphragm becomes paralyzed and bulbar symptoms are manifested. Usually the face escapes the widespread palsy and atrophy, but occasionally the lips, tongue, and other muscles of this region may suffer. The wasting may be so great in parts that whole muscles disappear.

The mechanical irritability of the affected muscles is increased, so that contractions of muscle bundles are caused by light blows. Continuous fibrillary tremor is observed running from one bundle to another, but it is not present in every case, though so frequent as to be considered a prominent symptom.

The deep reflexes are lost when the reflex centres are destroyed, for the groups of ganglion cells are the keystones of the reflex arcs, and when they are gone the reflex is naturally destroyed. Thus, the knee-jerk disappears

FIG. 201.



Progressive muscular atrophy, showing wasting and lordosis.

when the thigh muscles begin to be invaded; wrist-jerk and elbow-jerk when their respective groups suffer.

While rheumatoid pains may be complained of, anæsthesia never exists, nor is there loss of muscular sense.

In the early stage of wasting of a muscle the electrical reaction is apt to be nearly normal, which is explained by the fact that most of the ganglion cells, motor fibres, and muscular fibres are as yet unaffected. But after a time the whole phenomenon of reaction of degeneration will be present (loss of faradic contractility; anodal closure, greater than cathodal closure contraction with galvanic current; slow vermicular response of the muscle to the latter). The electrical examination is important in differential diagnosis, for in the primary muscular dystrophies the reaction will persist in a normal way as long as there is a sufficient number of fibres left in the muscle to contract. The adipose tissues waste as well as the muscles. There are no vasomotor changes, or discolorations of the skin, or trophic disturbances in the nails, no sympathetic derangement, no optic nerve atrophy, no loss of pupillary reflex, no disorder of the sphincters. There may be in some cases sexual impotence, and nystagmus has, in rare instances, been observed.

The disease lasts usually ten or twelve years, but termination in a year has been observed, and it has been known to endure as long as thirty years. Death is generally due to pulmonary disorders consequent upon palsy of the respiratory muscles, or to the bulbar palsy and difficulty in swallowing and deranged laryngeal apparatus; but bed-sores and septicæmia or other intercurrent maladies may end life.

Pathology and Pathological Anatomy. The anterior cornua are the chief parts affected, and usually they are changed most in the cervical enlargement, because the muscles most commonly affected (upper extremities) have their trophic centres here. The large ganglion cells have degenerated and disappeared, or may be few and shrunken into angular or globular bodies without processes. The nerve fibrillæ degenerate also, and connective-tissue elements are increased in quantity. Larger bloodvessels are found dilated and the perivascular spaces increased in size. The size and shape of the cornua may not be especially changed. They will be found wholly normal in segments representing unaffected muscle-groups. In many cases morbid changes may be traced into the antero-lateral white columns. Degenerated fibres are found in the anterior roots springing from the diseased segments of the cord, and correspond to the degenerated ganglion cells in such parts. In some cases degenerated fibres have been followed up through the pyramidal tracts, through the medulla, pons, crus, internal capsule, and to the cortex, where the cortical cells have also been found fewer in number and atrophied. Where there has been bulbar paralysis similar changes are observed in the motor nuclei of the medulla. The posterior horns, posterior columns, cerebellar tracts, and posterior roots are always normal. In the peripheral nerves degenerated motor fibres are found in greater or less quantity according to the degree of disease in the anterior horns and the degree of muscular wasting. The muscles are found small and pale, and the fibres narrowed, undergoing fatty or vitreous degeneration, granular, at times without striation, and at times presenting a longitudinal striation. The interstitial connective tissue is increased, and between the fibres collect fatty globules and masses of brownish pigment. The disease is thus essentially a slow progressive degeneration of the spino-muscular portion of the motor tract, with consequent paralysis and atrophy of the muscles. Since there is at times more or less degeneration of the cortico-spinal portion of the tract, it must be assumed that the same primary pathological cause may attack other portions of the nervous system than the ganglion cells of the anterior horn. But the actual cause of the beginning of the degenerative process is unknown.

Diagnosis. The diagnosis of this disease should not be difficult if we bear in mind the distinctive features of the malady—the beginning in the hand or shoulder, the slow advance, the peculiar distribution of the wasting (in muscles more or less remote from each other), the absence of anæsthesia, the normal condition of the sphincters, lost reflexes, the fibrillary tremor, and the character of the electrical reaction. Local and multiple neuritis are distinguished by rapid onset and sensory symptoms.

Lead-poisoning is usually marked by paralysis and atrophy of the extensors of the wrists and fingers only; but this is symmetrical, and even where the wasting extends to shoulder muscles and the lower extremities, which is rare, it begins as a characteristic "lead palsy." Besides the history of exposure to lead, the blue line on the gums, and examination of the urine will finally exclude this disorder.

In diffuse myelitis we find sensory and sphincter symptoms.

In syringomyelia, where the atrophies may have a similar distribution to that of progressive muscular atrophy, as this is generally a disease of the cervical enlargement of the cord, we find peculiar sensory and trophic disorders and exaggerated deep reflexes.

In amyotrophic lateral sclerosis also the atrophies may be in every respect like those of the disease in question, but the deep reflexes are invariably exaggerated to a very marked degree.

Chronic poliomyelitis has a more rapid course at the beginning, then reaches a point where it becomes stationary or improves. There is first paralysis, then wasting. The reaction of degeneration appears earlier.

In pachymeningitis cervicalis hypertrophica, besides the muscular wasting in the arms, there is pain and rigidity of the neck and exaggerated tendon reflexes.

A local atrophy of muscles from overuse may be difficult to differentiate from a beginning Aran-Duchenne disease. Thus, in a young woman whom I treated for a year, there was wasting of the thenar muscles and first interosseous in the right hand. She had worked ten hours a day for ten years as a jewelry polisher, and the muscles affected were those most needed in her work. She recovered entirely by taking up general housework and under electrical treatment, but for a long time the diagnosis was impossible.

The progressive muscular dystrophies are often hereditary. Usually the disorder begins in youth. Some muscles are often found to be hypertrophied. There is no fibrillary tremor. The faradic reaction is normal.

Prognosis. As regards cure, the disease is incurable. The prognosis, as far as life is concerned, is that the disease may last for an indefinite number of years without developing dangerous symptoms, such as paralysis of respiration and deglutition, or pulmonary disease.

Treatment. Properly conducted electrical treatment, massage, systematic exercises of the muscles, and hypodermic injections of strychnine in the affected muscles (nitrate of strychnine gr. $\frac{1}{60}$) may be used with a fair prospect of staying the progress of the disease for a time, and at times even occasioning temporary improvement. The galvanic current should be employed (stroking the muscles with the anode so as to produce contraction), and when there is any response at all to the faradic, that also should be made use of. In all other respects the treatment should be merely symptomatic.

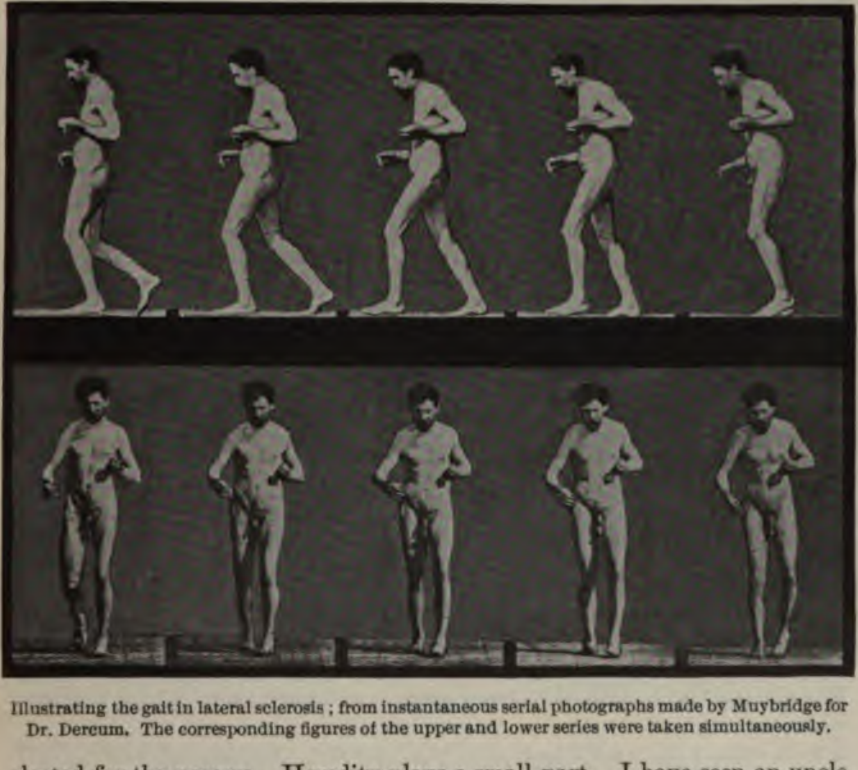
LATERAL SCLEROSIS.

Primary Lateral Sclerosis; Primary Spastic Paraplegia; Spastic Spinal Paralysis; Spasmodic Tabes Dorsalis.

Lateral sclerosis is a chronic disease characterized by rigidity and weakness of the limbs with enormously exaggerated tendon reflexes, and without atrophy or sensory or vesical disturbance. Since Erb, in 1875, and Charcot a little later, described this syndrome, its existence has been frequently verified by neurologists, and yet no pathological evidence has thus far been brought forward to establish it as a separate primary systemic disease of the spinal cord. But while such evidence is wanting, there is good reason for assuming a primary sclerosis of the pyramidal tracts to be a real pathological and clinical entity quite distinct from chronic dorsal myelitis, cerebral diplegia, or other conditions which often give rise to somewhat similar symptoms.

Etiology. As to etiology, little can be said except that probably the same causes which tend to bring about other chronic systemic degenerations in the cord are at work in this case, except that here the pyramidal tracts are elected for the process. Heredity plays a small part. I have seen an uncle and nephew affected with the same disease. Both sexes are equally affected. Three-fourths of the cases begin between the ages of twenty and forty years. Syphilis is not as frequent an antecedent disorder as in posterior sclerosis. Spinal concussion and exposure are often described as causes.

FIG. 202.



Illustrating the gait in lateral sclerosis; from instantaneous serial photographs made by Muybridge for Dr. Dercum. The corresponding figures of the upper and lower series were taken simultaneously.

Symptomatology. At first there is a sense of fatigue and weakness in the legs. This is followed by gradually increasing rigidity and great increase of the deep reflexes. The knee-jerks become exceedingly quick and strong, and often a quadriceps clonus is obtained. Ankle-clonus is marked. Although most marked in the lower extremities, the upper may also suffer, so that the wrist-jerks, periosteal, supinator, biceps, and triceps jerks become excessive. There is no wasting of muscles; on the contrary, they are large and firm. The spastic rigidity is so great that it becomes markedly manifest in the gait of the patient, who scarcely flexes the knees in walking, drags his toes, and often develops a tremor in the legs (from ankle-clonus) as he steps upon his toes in moving. (See Fig. 202.) This spastic rigidity is chiefly due to the increased deep reflexes in all of the muscles, but at times we find spasmodic movements, single contractions in the legs, due, as in some forms of myelitis, to motor irritation. Reflex action of the skin is often excessive. There are no sensory disturbances. There are no trophic changes in the skin or joints, no ocular symptoms, and rarely any sphincter trouble or loss of sexual power. A cold atmosphere increases the rigidity of the muscles. Occasionally a jaw-jerk is found in these cases.

Pathology and Pathological Anatomy. There are no recorded autopsies in cases of pure primary lateral sclerosis; but sclerosis of the lateral columns has been found frequently enough in conjunction with other diseases, as in amyotrophic lateral sclerosis, general paresis of the insane, cerebral diplegia of children, and ataxic paraplegia.

Diagnosis. From a study of the above symptoms and the exceedingly slow progress of the disease, we should be able to make an accurate diagnosis. At the same time caution is required, for what may appear for years to be a primary lateral sclerosis may turn out in the end to be a multiple sclerosis. Such an instance I saw not long ago in a young girl, who five years before had been pronounced by several eminent neurologists to be a case of primary lateral sclerosis, but who now presents all of the typical symptoms of the latter disease. Tumors of the cervical cord, peculiar forms of transverse myelitis, compression in Pott's disease, and hydromyelus may each simulate at times the clinical symptoms described above. Thus it behooves us to use care in our diagnosis of the character and locality of the lesion.

Prognosis. The disease is incurable, but its progress is exceedingly slow, generally imperceptible for years, and the symptoms are never as distressing as in many other forms of chronic spinal cord disease. There is the possibility of occasional temporary improvement in these cases.

Treatment. Hydrotherapy is of some avail in these cases, especially for the purpose of lessening the rigidity, and to this end prolonged warm baths (one to two hours at a temperature of 90° to 95° Fahr.) are well adapted. In cases with syphilitic history, iodide of potash and mercurial inunction are indicated. Massage is of some service, while electricity is absolutely valueless. Strychine, nux vomica, and other drugs which may tend to increase the spasticity are interdicted. Residence in a hot climate is advantageous, where it is feasible, and where there are no objections on other grounds against it. Arsenious acid (gr. $\frac{1}{60}$) and the bromides are sometimes useful. Solanin may be employed in some cases to overcome rigidity with moderate success.

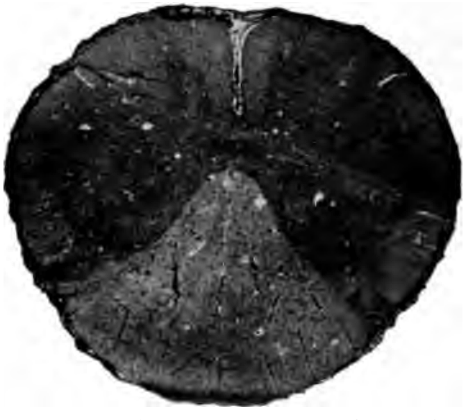
AMYOTROPHIC LATERAL SCLEROSIS.

This disease is clinically and pathologically well defined, in that it is a combination of the two diseases hereinbefore described, viz.: lateral sclerosis and chronic spinal muscular atrophy. Keeping the clinical pictures of these maladies well in mind, it would be difficult to make an error in diagnosis in this case. We are indebted to Charcot (1869) and Joffroy (1869–1874) for our first careful delineations of this disorder.

Etiology. We do not know what factors are at work in the production of amyotrophic lateral sclerosis. Doubtless when we understand better the etiology of other chronic systemic diseases or degenerations of the spinal cord, the causes in this disorder will be clear. It is essentially a disease of adult life (twenty-five to forty-five years), but younger and also older persons have been subjects of it. It is more common in males than in females.

Symptomatology. Usually the first symptoms to be noticed are those belonging to progressive muscular atrophy, and the wasting is prone to follow the typical course of that disease, beginning in some part of the upper extremity, generally the thenar and hypothenar eminences of one hand. In addition to the wasting there is a sense of fatigue and weakness in the whole arm. The disease is very slow in its onset and extension. In six months or a year the symptoms of a primary lateral sclerosis begin also to be manifested, weariness, weakness, stiffness, and uncertainty in the lower extremities, combined with more or less tremor in walking, such as we know to be due in the above-named disorder to the increase of the reflexes. As a rule, patients present themselves for examination when these symptoms are obvious, and upon examination we find atrophy and paralysis of muscles of the hands, arms, or shoulders; in fact, very much the same state of affairs as previously described in the section on Chronic Spinal Muscular Atrophy. But in addition to the amyotrophy there are symptoms directly the reverse of the disease referred to, in that the deep reflexes instead of being lost are greatly exaggerated. They are invariably increased even from an early period. The knee-jerks are quick and extensive in response to taps on the patellar tendons, top of the patella, or muscles. There is ankle-clonus. The wrist-jerks are strikingly apparent. The periosteal biceps and supinator-jerks and the triceps reflexes are very marked. This is true, even though the wasting in the extremities may be considerable. There is thus a singular combination of atrophy and spastic rigidity. The gait is stiff and spastic as in lateral sclerosis. The legs resist passive movements, but are found to be paretic. Upon electrical examination of the atrophied muscles we find

FIG. 203.



Amyotrophic lateral sclerosis. Second cervical segment. The degeneration of the crossed pyramidal tract shows in the characteristic faint deepening of the stain in this region. Carmine stain.

the same condition as in spinal progressive muscular atrophy, viz.: always the reaction of degeneration, when the wasting is considerable, sometimes response to faradaism, still, if the atrophy is in an early stage. Sensibility is never in any wise disturbed. There is no change in the cutaneous reflexes. The sphincter of the bladder is properly controlled, and that of the rectum also normal in function. As in progressive muscular atrophy, bulbar symptoms come on in the course of years. Paralysis of the palate gives a nasal intonation to the speech. Difficulty in swallowing is noted. The lips and tongue become more or less atrophied and paretic, so that labial and lingual sounds are improperly connected. Fibrillary tremor is observed in these as in many other atrophied muscles. At times we find a strong jaw-jerk present. Death finally comes from respiratory failure, inhalation pneumonia, or some intercurrent disease.

The increased reflexes are due, of course, to the degeneration in the pyramidal tracts (inhibitory fibres), and apparently this degeneration precedes to a certain extent many of the changes in the anterior horns. As long as the reflex arc is not totally destroyed the reflexes are increased, but as soon as this takes place there can no longer be an exaggerated condition of the tendon-jerks. As a rule, however, the wasting never goes to such an extent before death that the reflex arc is interrupted, and consequently all of the deep reflexes are apt to be overactive to the last.

Pathology and Pathological Anatomy. The remote causes of this disease are unknown. The morbid anatomy of the malady is clearly understood from numerous autopsies. We find in spinal cord well-marked sclerosis of the pyramidal tracts and degeneration and atrophy of the ganglion cells of the anterior horns. The sclerosis is observed in the lateral columns and also in the columns of Türck, and may be traced upward through the medulla and pons to the internal capsule, and rarely as far as the Rolandic cortical areas. The changes in the anterior horns are such as have been described in the pathology of progressive muscular atrophy. Where bulbar symptoms have existed, similar changes are found in the nuclei of the hypoglossus, vagus, spinal accessory, etc. From these parts the degeneration may be followed outward toward the periphery in the motor fibres of the nerves affected as far as the muscles. The muscles are small and pale, and often more than half replaced by connective tissue and fat. The fibres are narrowed, granular or fatty, without striation, as in progressive muscular atrophy; and often quite normal fibres are to be observed among the degenerated ones.

Diagnosis. One is not apt to confuse so clear a clinical picture with that of any other similar syndrome. If confusion were to arise it would be from too superficial an examination of a case. The coexistence of muscular atrophies and exaggerated reflexes is seldom noted in other maladies. We find it in syringomyelia and other affections (tumors) of the cervical cord, but here attention to the sensory disturbances is sure to lead to a correct idea of the case. Chronic hypertrophic cervical pachymeningitis might at times mislead somewhat, but the pain and rigidity of the neck and the absence of reflexes in the wasted upper extremities should suffice to make the diagnosis evident in this case.

Prognosis. There is no hope of a cure in amyotrophic lateral sclerosis. Death occurs in the course of a few years. The atrophies often lead to complete helplessness after a time. Only exceptionally does the disease evince any tendency to become stationary.

Treatment. Electrical treatment may be used upon the atrophied muscles, stroking with the anode so as to produce contractions with the galvanic current. Massage may also serve to stimulate nutrition. Warm baths diminish the spastic rigidity to a certain extent. Arsenious acid (grain $\frac{1}{60}$) may be used internally in conjunction with tonic or other treatment deemed advisable for coincident conditions.

LOCOMOTOR ATAXIA.

Posterior Sclerosis; Tabes Dorsalis; Tabes Dorsualis. Locomotor ataxia is a chronic progressive disease of the nervous system, with degenerative lesions chiefly marked in the posterior columns of the cords and posterior roots, and characterized clinically by inco-ordination of movement, pains, loss of knee-jerks, anæsthesia, loss of the pupillary reflex to light, and various visceral and trophic symptoms. There is considerable variation in the symptoms as regards the degree of their manifestation and the number of them present, so that complex and atypical forms are met with. Not only are there types which may be termed neuralgic and paralytic, and types with initial optic atrophy and with muscular wasting, but there are cases complicated with other forms of disease, such as lateral sclerosis and general paralysis of the insane. There are maladies which resemble locomotor ataxia, but which should not be classed with it, such as hereditary ataxia and cerebellar heredo-ataxia.

Etiology. Males suffer from the disease much more frequently than females. The proportion is something like ten to one. It is more common in middle life than at any other age, fully half of the cases developing between thirty and forty. Next in frequency is the period between forty and fifty, and next between twenty and thirty. Thus, almost all the cases occur between the ages of twenty and fifty. In very rare instances it has been met with as early as ten and as late as sixty or more. Heredity has little to do with the disease, a general neurotic taint being present in only about 10 per cent. of the cases. Direct inheritance is extremely rare, though one or two such instances have been reported.

Syphilis is by far the most important etiological factor in tabes. Fournier places the percentage of syphilis in cases of locomotor ataxia at 91–98 per cent., Erb at 88 per cent., Rumpf at 80-85 per cent., and Sachs at over 90 per cent. Gowers found only 58 per cent. of his private cases syphilitic, but assumes a percentage of 80 among the lower classes. In 83 cases of locomotor ataxia observed by me at the Vanderbilt clinic, in which this factor was carefully investigated, a specific history was verified in 71 per cent. But, while syphilis is an antecedent in three-fourths of the cases, the syphilitic cases resemble the non-syphilitic as regards the histological characters of the lesions, and specific treatment has no effect whatever upon the cases with syphilitic history. Hence the malady is not a syphilitic disease, not a direct sequel of syphilis, but the venereal disorder prepares the constitution like a cachexia for the development of the degenerative process. Inherited syphilis seems to be an etiological factor in every case of tabes occurring in children. The interval between the contraction of syphilis and the development of locomotor ataxia is commonly between six and twelve years, though cases are on record of intervals of less than a year and more than twenty years.

The disorder is more common in city than in country populations.

Concussion of the spine from falls and the like is sometimes found to be a cause. Overexertion, great fatigue, exposure to damp and cold, some acute diseases (acute rheumatism, typhoid fever, diphtheria, typhus, pneumonia), and alcoholic excess have been mentioned as causes.

What is known as "secondary tabes" is a form that sometimes succeeds myelitis, tumor, or syphilitic inflammation of the meninges or cord.

Symptomatology. In typical locomotor ataxia there are peculiar symptoms in motor, sensory, and reflex spheres. There is no paralysis or wasting (except in certain rare cases to be mentioned later on), but the motor symptom is an inco-ordination of the muscles, an ataxia chiefly observed in the lower extremities, sometimes in the upper. The sensory disturbances consist of "lightning pains," anæsthesias in the limbs, and the "girdle" sensation. The reflex symptoms are the loss of the knee-jerks and the loss of the pupillary reflex to light. These are the chief manifestations, but there are many others that present themselves occasionally, such as optic atrophy, diplopia, visceral "crises," difficult micturition, impotence, muscular atrophies, and trophic changes in the skin, joints, and bones.

These various symptoms appear at various periods in the progress of the disease, and it is usual to consider three stages in its course. They are not very distinct, it is true, but it is convenient to have an artificial division of the kind, and the following table will serve to indicate in a general way the relations of the symptoms to each stage:

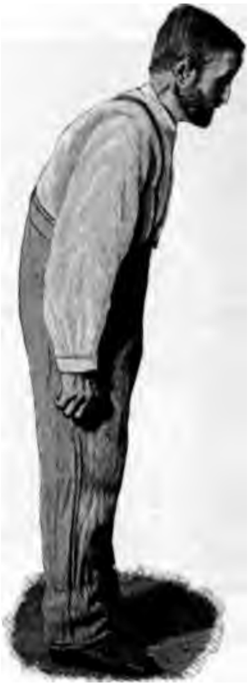
Initial Period.	Second Stage.	Final stage.
Inco-ordination, but no change of gait.	Greater inco-ordination, and marked ataxic gait.	Cannot walk because of ataxia.
Numbness of the feet.	More marked anæsthesias.	Extensive anæsthesias.
Shooting pains in the legs.	Pains worse.	Pains less.
Diminished or lost knee-jerks, one or both.	Lost knee-jerks.	Lost knee-jerks.
Sluggish or lost pupillary reflex to light.	Lost pupillary reflex to light and myosis.	Lost reflex to light, myosis, paralysis of accommodation.
Weakness of sexual function.	Impotence.	Impotence.
Transient diplopia; transient ptosis.	Ocular palsies rare, or marked ophthalmoplegia.	Ophthalmoplegia.
Sluggish micturition.	Increased vesical weakness.	Catheterization needed.
Optic atrophy.	Optic atrophy rarely develops.	Blindness.
Trophic changes in the joints.	Trophic changes not so common.	More marked if they began in early stage.
Hemiatrophy of tongue.	Deafness.	Increased.
	Laryngeal and visceral crises.	Not so common.
	Girdle sensation.	Unnoticed.

There are very rarely any mental symptoms, the mind usually remaining clear to the last, but sometimes we have a complication with general paralysis of the insane (though the presence of transient tabetic symptoms in paresis is not to be considered as evidence of there being a combination of locomotor ataxia proper with this malady, see also Chapter XXIII.); and I have seen a number of cases in which insanity, melancholia, or subacute mania has developed during the course of the affection.

We will now examine the various symptoms somewhat more in detail.

MOTOR SYMPTOMS. The ataxia is a symptom of gradual development, first showing itself by slight unsteadiness in walking or in standing, especially when the eyes are closed, and when the feet are placed closely together ("Romberg symptom" or "tabetic swaying"). A slight amount of swaying under these circumstances is observed in a healthy person, and familiarity with the test is required for the determination of what amount of unsteadiness is a morbid symptom. The patient often discovers this difficulty with equilibrium first himself when getting out of bed at night, walking on dimly-lighted stairs, or when out for an evening stroll. As time goes on the inco-ordination increases, until walking and standing are noticeably unsteady. The feet are kept apart in standing, in order the better to preserve equilibrium. When in bare feet the inco-ordination is more marked, and also more readily discovered in its early manifestation. The shoes give a stiff support to the feet, and their removal throws more effort upon the unsteady muscles. After a while the gait assumes its peculiar characters (see Figs. 205 and 206), which can readily be imitated by walking with the legs a little farther apart than normally, throwing the feet a little high in stepping forward, and bringing the whole sole down upon the floor at each step. Soon a cane, or two sticks, or the assistance of another person becomes necessary, and at last locomotion is altogether impossible, owing to the complete ataxia of the legs, which move in every direction but the one desired.

FIG. 204.



Attitude in locomotor ataxia, advanced stage. (Philadelphia Hospital.)

The arms are also at times included in the ataxic condition, occasionally before the inco-ordination develops in the legs. It may be noted in writing, in bringing the index fingers together with the eyes shut, touching the nose with the forefinger, and so on. Later it may be impossible for the patient to use his fingers in picking up articles, buttoning his clothes, and so forth. Sometimes the trunk muscles are also ataxic, but the inco-ordination does not affect the movements of the face, tongue, eyes, or head.

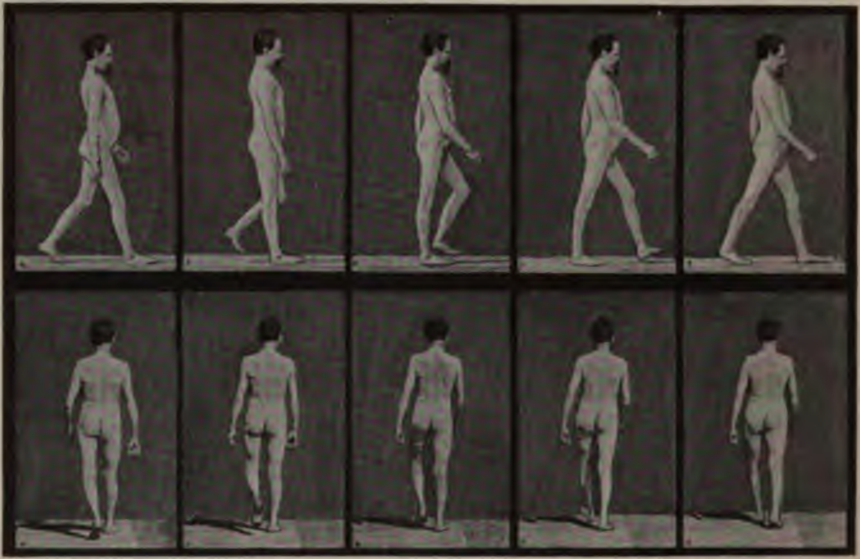
In spite of all this loss of control over the muscles, it is unusual to observe any loss of power in them. There is no paralysis or paresis, even in the last stages, unless, indeed, the disorder is conjoined with lateral sclerosis, peripheral neuritis, or progressive spinal muscular atrophy.

SENSORY SYMPTOMS. Nearly all cases of tabes suffer from sensory symptoms of one kind or another, either neuralgic pains or anæsthesias and paræsthesias, or all of them together. The pains are peculiar in their shooting or lightning-like character, and in their tendency to come on paroxysmally, especially at night, and usually in the legs, through the arms, trunk, viscera, or head may be their seat. The pains may be localized at some particular cutaneous area or shoot through the long, deeply-seated nerves. Often they have no relation to the nerve-trunks, and, as they depend upon irritation of sensory roots at certain levels, they vary much in their peripheral manifestations. The pains may last a few hours or several days, may return in the same parts, or change from one locality to another. They are not always shooting pains; occasionally they are continuous, dull, burning, or gnawing. As in rheumatic and other affections, the pains are often influenced by the weather. They are among the earliest symptoms of the disease. Rarely they are altogether absent.

Another sensory symptom is the "girdle sensation," or sense of constriction about the trunk at different levels, sometimes as low down as the groins or thighs.

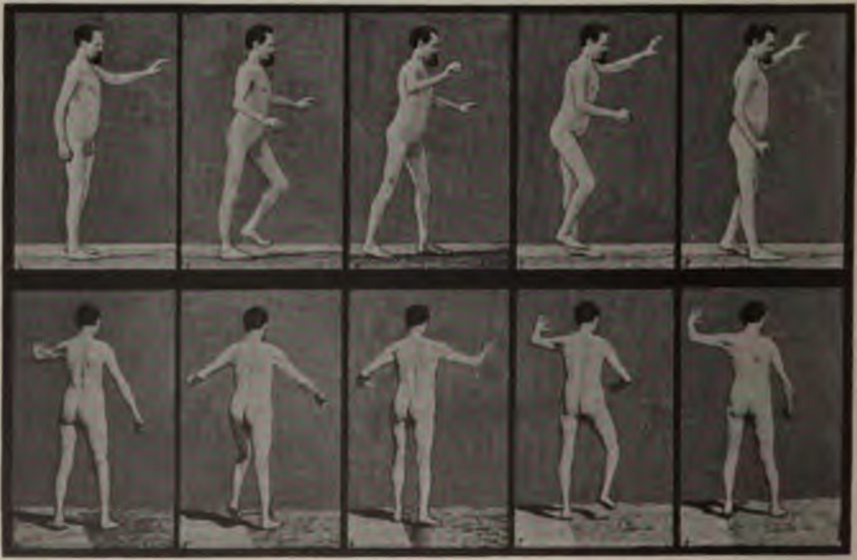
Paræsthesiæ in the form of burning, prickling, tingling, numbness, cold, heat, or the like, are noted usually in the feet and legs, and often also in the hands. Sometimes there is hyperæsthesia, hyperalgesia, hypercryæsthesia,

FIG. 205.



Gait in a case of locomotor ataxia. From instantaneous serial photographs of a patient of Dr. Dercum, made simultaneously from two different point of view by Muybridge.

FIG. 206.



Showing the same patient (as in Fig. 205) on attempting to walk with the eyes closed. From photographs by Muybridge, as before.

and hyperthermæsthesia. More commonly there is loss of sensibility in parts, especially in the feet and legs, to touch, pain, cold, and heat, but there is not the dissociation of sensibilities as in some other diseases, like syringomyelia, for instance. Delay in the perception of sensations, especially of pin-pricks, is rather common, the delay often amounting from five to fifteen seconds. A single sensation may be perceived as many (polyæsthesia), or referred to the opposite extremity (allochiria). The sense of posture is usually affected.

As regards the special senses, Gowers has recorded one case with a double temporal hemianopia, and not long ago I presented to the New York Neurological Society a case of tabes with homonymous hemianopia (or rather three quadrants lost symmetrically in each field). Optic atrophy will be mentioned later. Deafness is occasionally met with, as are also anosmia and anæsthesia in the distribution of the fifth nerve.

REFLEX SYMPTOMS. The loss of the knee-jerk is the most important symptom of this class. It is usually early. In the preataxic stage it is sometimes unequal on the two sides. The plantar reflex is abolished next in frequency. As the disease progresses the cremasteric, gluteal, and abdominal reflexes are apt to disappear. Loss of the sexual reflex is usual and generally one of the earliest symptoms.

The bladder reflex is commonly much impaired; as a rule, there is slowness of micturition and only partial evacuation of the bladder. Sometimes there is incontinence, though rarely paralytic. Constipation is frequent, but paralysis of the sphincter ani is not common.

OCULAR SYMPTOMS. It is extremely seldom that the reflex of the iris to light is not lost. In nearly every case there is the Argyll-Robertson pupil (reflex to light lost, movement in accommodation still present). I have found in all my cases that the cilio-spinal reflex is also lost. Occasionally the accommodative movement is absent. It is common for the pupils to be small (spinal myosis), sometimes irregular, and rarely unequal.

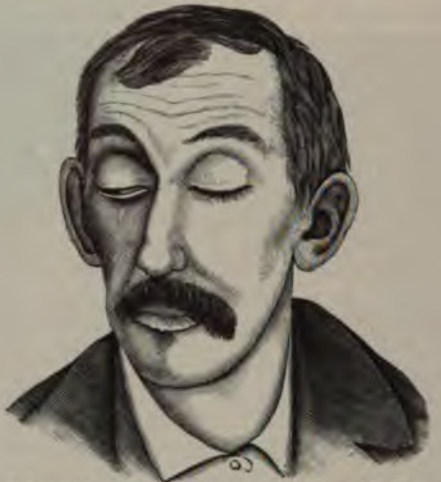
The external muscles of the eye are also frequently involved, so that one may have transient palsies of one or more muscles, lasting for a few days to a few weeks or remaining permanently. Temporary palsies are met with in the early stage, the permanent paralyses later. One of the external recti or levators is usually selected. Sometimes the whole of one or both third nerves is affected, and occasionally all of the muscles of one or both eyes, external and internal. (Fig. 207.)

In perhaps one case in ten or twelve optic atrophy occurs, and usually as an early symptom. It is a gray atrophy. When optic atrophy develops the spinal symptoms are often retarded or arrested. Blindness may be gradual, sometimes speedy, total, or only partial.

VASOMOTOR AND TROPHIC SYMPTOMS. Local sweating, ecchymoses, temporary loss of hair in spots or change in its growth, and herpes are observed as symptoms of locomotor ataxia. Thickening of the skin of the sole with the formation of blisters and indolent ulcers is not uncommon. These ulcers are peculiar to tabes, and are known as "perforating ulcers" of the feet. They burrow deeply and heal with difficulty. Similar ulcerations about the toe-nails are met with. Sometimes there are trophic changes in the nails of both hands and feet, but especially of the latter. The nails become thickened, brittle, rugose, and may fall off, growing again in the course of time. Occasionally the teeth are loosened, fall out, or decay rapidly.

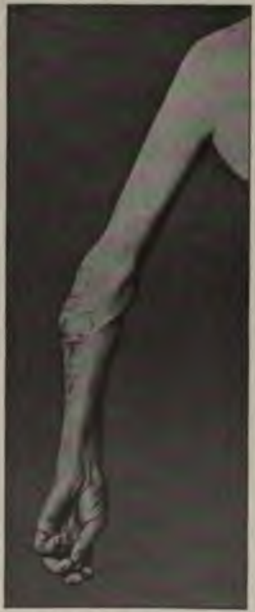
Muscular atrophy, sometimes local, sometimes widespread, is not infrequent, occasionally no doubt due to disease of the ganglion cells of the anterior horns, but probably more often due to neuritis. In the case here photographed (Fig. 207), to show double ophthalmoplegia, external and internal, there were also paresis and wasting of the masseters, temporals, and pterygoids on both sides, hemiatrophy of the tongue, wasting of right trapezius and a number of muscles in the forearms, hands and thighs, all of which showed degenerative reaction. Déjerine reported eleven out of 106 cases of loco-

FIG. 207.



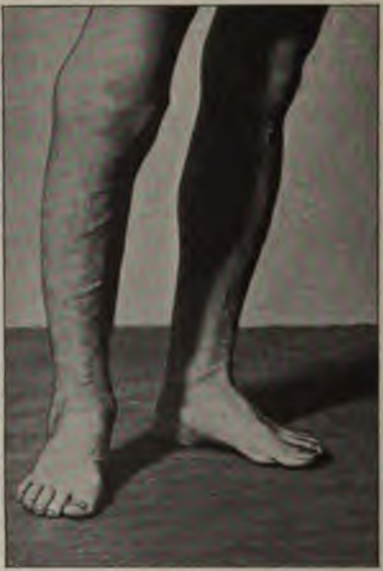
Case of Dr. Peterson. Tabes with palsies of the ocular muscles of both eyes and wasting of the masseter and pterygoid muscles.

FIG. 208.



Extensive dystrophy of elbow with fracture of ulna. (Philadelphia Hospital.)

FIG. 209.

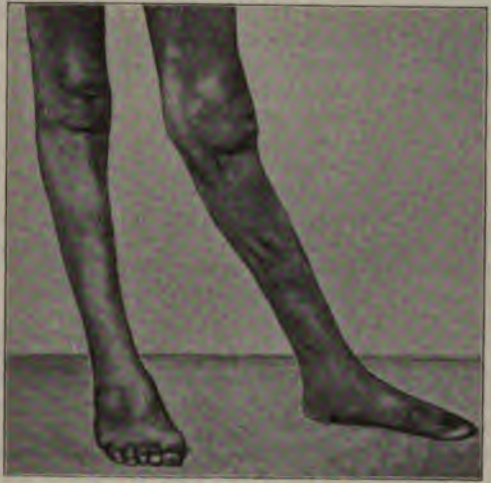


Excessive dystrophy of right knee. (Philadelphia Hospital.)

motor ataxia presenting muscular atrophies. Hemiatrophy of the tongue is occasionally an early symptom.

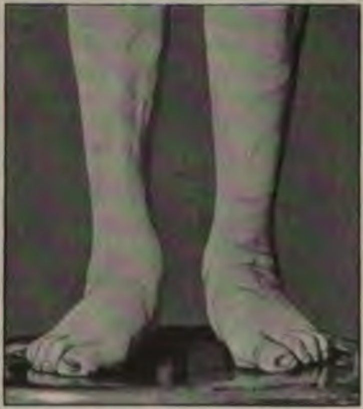
Among the more remarkable trophic phenomena are the so-called "Charcot joints." The knee is the joint most frequently affected (one-half the cases), and next in order follow the hip-joint (one-fifth of the cases), shoulder, tarsus, elbow, and ankle. The trophic change may be atrophic or hypertrophic. In the former case the cartilages and heads of the bones are eroded, and dislocation or abnormal extent of movement may be presented. In the hypertrophic form the joint becomes greatly enlarged by the formation of hyperostoses of irregular shape on the heads or shafts of the bone, and the ligaments may ossify. Usually the onset of the arthropathy is sudden and

FIG. 210.



Dystrophy of both knees, with marked retro-flexion.

FIG. 211.



Dystrophy of ankles and feet. Loss of plantar arch; marked flat-foot. (Philadelphia Hospital.)

characterized by great swelling, generally painless, sometimes preceded by rheumatoid pains.

A marked fragility of the bones with spontaneous fractures is noticed in some cases, usually in such as show also a tendency to the development of arthropathies. The fractures are commonest in the femur, tibia, and fibula, radius, and ulna, humerus, and clavicle, though other bones may also suffer.

The formation of bony masses in muscles, a myositis ossificans, has lately been described by two or three authors as sometimes occurring in tabes.

VISCERAL SYMPTOMS. Certain peculiar paroxysmal visceral disorders occur in tabes. They are termed *crises*. Gastric and laryngeal crises are the most common forms; but rectal, intestinal, cardiac, bronchial, renal, vesical, and urethral crises are met with at times. In the gastric crises there is severe epigastric pain, sometimes diffused through to the back and over the whole abdomen, and vomiting, usually incessant, with or without nausea. The attack lasts from a few hours to two or three days, then ceases, to return in some weeks. While such an attack is a typical one, there are apt to be great variations in the degree of pain and other symptoms, but the paroxysmal character of the seizure is always significant.

In the laryngeal crises there is a similar paroxysmal disturbance of function, with a strong resemblance at times to pertussis and laryngismus stridulus. There is spasm of the adductors or paralysis of the abductors of the larynx, noisy respiration, and dyspnœa. The onset is sudden, and the seizure may last for a few minutes or for hours. Just as paroxysms of pains in the legs, arms, or hand are related to processes at various levels in the central nervous system, so these crises represent changes going on in various visceral centres of the spinal cord and bulb.

In rare instances we observe transient epileptiform and apoplectiform attacks in tabes, resembling very much those of general paresis. (See Chapter XXIII.)

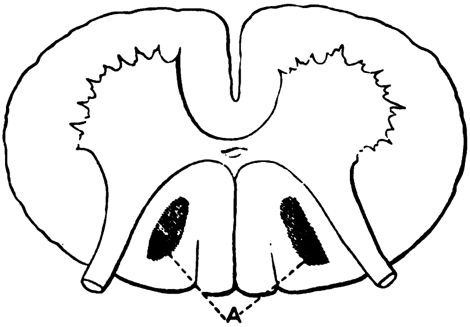
Course and Termination. While usually progressive and at a more or less rapid rate, there is great variation in the duration of tabes in limits from three to thirty years. Sometimes the first stage may last twenty years, and the second stage has in some instances been protracted to fifteen years. Acute cases lasting less than a year have been recorded. Death is usually produced by intercurrent diseases, for there is nothing in the nature of tabes itself to produce death. Occasionally a laryngeal crisis has proved fatal, but the other crises never. Patients become bed-ridden, remaining so for years. Bladder disease, renal disorders, sometimes bed-sores, and septicæmia, carry them off.

Complications. Acute or subacute myelitis, sclerosis of the lateral columns (ataxic paraplegia), progressive muscular atrophy, disseminated sclerosis, general paresis, valvular heart disease, and syphilitic disease of the bloodvessels or gummata are among the complications met with in locomotor ataxia.

Pathology and Pathological Anatomy. Of late considerable advance has been made in our knowledge of the pathological conditions underlying tabes. It is not so long ago that the degeneration of the posterior columns was looked upon as the chief, if not the only, morbid process. But the disorder is now known to be a general disease of the nervous system, affecting both central and peripheral portions, though mainly limited to sensory or afferent structures. While it is true that in the posterior nerve-roots and posterior columns we have sensory fibres which are affected in tabes, there are in other parts of the central nervous system motor ganglia and nerves that suffer in this disease (ocular palsies, progressive muscular atrophy, etc.). The ganglia of the posterior roots are probably first acted upon by the morbid process,

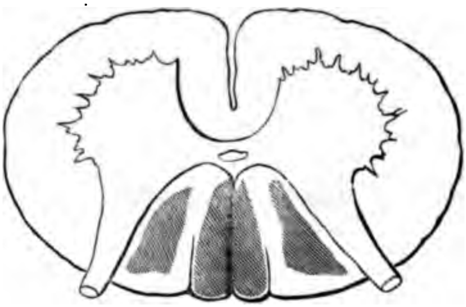
and following upon this we have degeneration of the ascending fibres in the posterior columns. The first segments to be affected are those of the lower dorsal and upper lumbar region, as a rule; but the point of origin varies: occasionally it is in the sacral segments or in the cervical enlargement. The

FIG. 212.



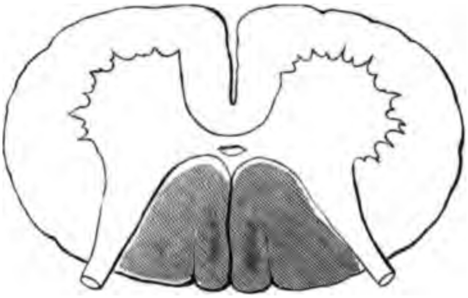
Early stage of tabes. A. Site of first degeneration in the posterior columns.

FIG. 213.



Second stage of tabes. Progress of degenerative process in other parts of the posterior columns.

FIG. 214.

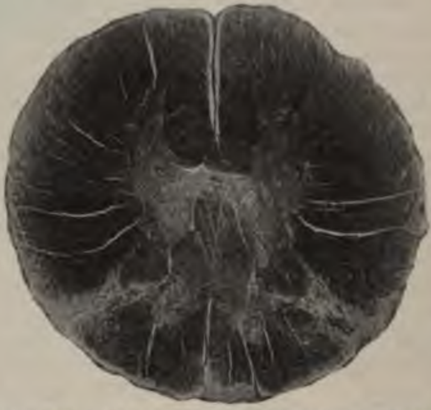


Final stage of tabes. Degeneration of all parts of the posterior columns.

symptoms vary with the point of origin. Vesical and genital symptoms **at** the inception of tabes point to origin in the sacral **segments; ataxia and** lightning pains in the legs, with lost **knee-jerk,** signify a higher level; **pains,** numbness, and ataxia affecting the arms indicate a beginning in the cervical enlargement.

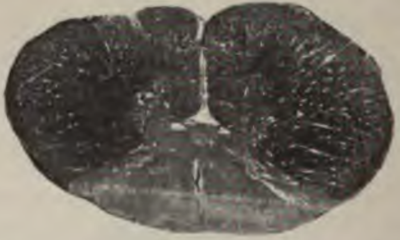
The first part to be affected after the spinal ganglia and posterior roots become involved is the portion of the posterior column lying in the middle-root zone between the columns of Goll and Burdach, but it gradually extends until the whole of the posterior column is ultimately converted into sclerotic tissue. (Figs. 212, 213, 214). The atrophy and grayish color of these columns in a case of tabes is easily noted at autopsy with the naked eye. When stained the sclerotic columns become conspicuous for their deep color. Lissauer's tract is generally affected, and sometimes the antero-lateral and direct cerebellar tracts are more or less diseased. In some very rare cases there is degeneration in the pyramidal tracts; occasionally the gray matter of the posterior cornua suffers, and degeneration has been noted in the column of Clarke. In some. rare instances the anterior horns have been invaded, with resulting muscular atrophies, though these may depend in some cases upon degeneration in the peripheral nerves, which is not uncommon in advanced tabes. This degenerative atrophy of the nerves begins in the distal ends, travels slowly upward, but does not often reach the larger nerve-trunks. Usually it is the nerves of the leg which are the ones to suffer.

FIG. 215.



Tabes dorsalis. An early stage, which involves one root-zone.

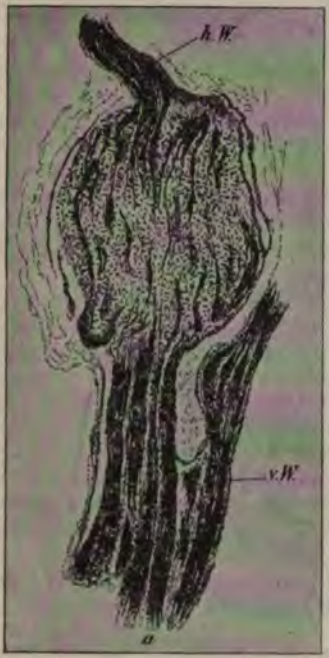
FIG 216.



Tabes dorsalis. A much later stage of the posterior sclerosis. All of the posterior columns are quite uniformly involved.

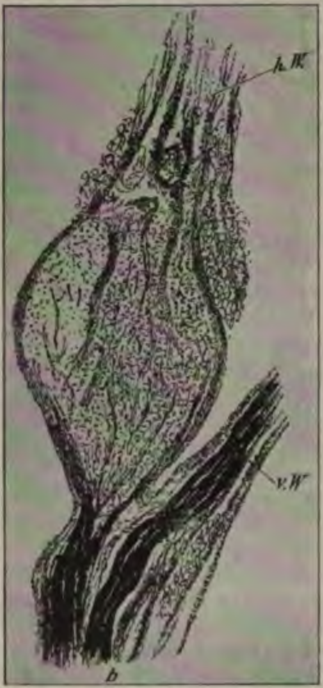
Under the microscope we find, as a rule, some variety in the histological characteristics of the sclerosis. There is more or less thickening of the trabeculæ, with increase of nuclei; the nerve-fibres are narrowed or absent, the vascular walls thickened, and the pia and its septa increased in thickness, either over the posterior columns or even over the whole cord at times. Diffuse myelitic changes and disseminated patches of sclerosis have been occasionally observed. The posterior nerve-roots are found altered, sometimes only slightly, often considerably, as far as the ganglia. The ganglia are usually but slightly effected, and the nerve beyond them, as a rule, not implicated in the process (Figs. 217 and 218). But it must be remembered that the function of ganglion cells may be seriously impaired without noticeable structural changes, and the latest view of the pathology of tabes may be stated to be that some irritative agent acts upon the spinal ganglia and their homologues, producing a lesion which, while not structurally visible, suffices to bring about degeneration in the sensory fibres of the cord and medulla and in the peripheral nerve-ends. Another view put forward of late also is that the cause of tabes is a meningitis posterior.

FIG. 217.



Normal posterior nerve-root and ganglia.

FIG. 218.



Posterior nerve-root and ganglia in tabes.

Diagnosis. Locomotor ataxia is recognized by the combination of its characteristic symptoms. Loss of the knee-jerk, the Argyll-Robertson pupil, unsteadiness of gait without loss of power in the limbs, lightning pains, a slow onset, and a specific history are the chief points to be relied upon. The lost knee-jerk is the most important of these symptoms, and, as a rule, it is lost in the earliest stages. A diagnosis of tabes should not be made unless this feature is noted, even though most of the other signs are present. Sometimes in the very earliest period one knee-jerk will be present and the other lost, which in itself is very significant. We do not always find the Argyll-Robertson pupil in tabes, though it is one of the cardinal symptoms. Lost knee-jerks and lost iris-reflex exclude all other disorders except general paresis, which has its own distinguishing symptoms.

Tabes and alcoholic neuritis are apt to be confounded, but hardly unless the latter takes the form of pseudo-tabes. In pseudo-tabes the alcoholic history, normal pupils, slight weakness of the limbs, muscular tenderness, and occasionally altered electrical reactions should guide one to a correct interpretation of the case.

In ataxic paraplegia and in other forms of paraplegia there is exaggeration of the knee-jerks and ankle-clonus.

In post-diphtheritic paralysis the differential criteria are the preceding paralysis of the palate and of accommodation, together with loss of muscular power and the absence of pains.

While the ataxia of cerebellar neoplasms and that of tabes sometimes resemble each other, the differential diagnosis is generally made clear by pains and anæsthesia in the former and headache in the latter. Their resemblance consists in the ataxia, occasionally absent knee-jerk in cerebellar tumor, and optic nerve atrophy.

It is not unusual for the pains of locomotor ataxia to be treated for rheumatism, sciatica, and the like, and the various crises for essential diseases of the affected organs, and the physician should be on the lookout for other symptoms, such as lost knee-jerk, in such cases.

Prognosis. Arrest and improvement of the disease is possible in the earliest stages, but a cure is not to be looked for after the second stage is reached, though even then amelioration is often brought about by careful treatment. The rule is, however, that the disease in the majority of cases is progressive, but death results from some intercurrent disorder, almost never from tabes itself. The occurrence of optic nerve atrophy is generally an indication of the arrest of progress of the malady in the spinal cord.

Treatment. The general treatment consists in a careful regulation of the daily life of the patient, the avoidance of mental or physical fatigue, anxiety, or exposure. When possible the patient should give up any regular occupation that may sap his energies. Often prolonged rest in bed is useful. Residence in a warm, dry climate, with low altitude, where a quiet life, out of doors, may be enjoyed, and long sea-voyages are often to be recommended, but with due regard to all the conditions.

Light and easily digested food should be ordered, and the patient should be absolutely abstinent as regards alcohol, and moderately so as regards tobacco. Sexual excess is injurious.

Many cases have been improved much by hydrotherapy alone. The best application of water is the Charcot douche, three or four times weekly.

It is difficult to determine whether any actual benefit is to be derived from electricity. The pains sometimes yield to the faradic brush or to the stabile galvanic anode placed over the seat of pain. A weak solution of aconitia, in combination with a 10 per cent. solution of cocaine, placed upon the anode, will sometimes relieve by electric cataphoresis these pains, particularly if the pains be superficial. Sometimes faradization of the muscles improves the gait of a patient for a time, and in vesical weakness faradization has been of service. Much benefit is to be expected from drugs, though it is true that there is no panacea for all the symptoms of locomotor ataxia, nor is there any particular remedy that can be looked upon as in any respect a specific.

So large a percentage of cases being due to syphilis, it is advisable, when there is such a history, to give a thorough course of treatment with mercurial inunctions and iodide of potash, especially in the early stages, though it must be confessed that anti-syphilitic treatment rarely, if ever, modifies the symptoms in any respect. In the later stages of tabes it is well to be moderate in the use of mercury and the iodide of potash.

One of the oldest and most efficient remedies is nitrate of silver in doses of ¼ to ⅜ of a grain three times daily. After long use of silver salts a certain degree of argyria may be produced, and this should be explained to the patient when he is put upon the drug. The discoloration of the skin from protracted exhibition of nitrate of silver is, however, more than counterbalanced by the good results generally obtained. Next to silver, arsenic is distinctly of service. It may be given in the form of Fowler's solution, 3 to 5 drops three times daily in water after meals, or in the shape of a $\frac{1}{12}$ grain pill of arseniate of soda, or $\frac{1}{60}$ to $\frac{1}{40}$ grain pill of arsenious acid. An excellent method of treatment is to alternate the silver and arsenic for two or three months at a time. The chloride of aluminum in 2 to 4 grain doses has been highly commended by some authorities. Among other remedies that may be looked upon as useful adjuncts at times are strychnine, nux vomica, iron, quinine, Calabar bean, ergot, phosphorus, chloride of gold and sodium, and belladonna. Strychnine is said to be especially useful when combined with nitroglycerin (gr. $\frac{1}{200}$).

The relief of the pains is most often called for by the patient, and ease from them is commonly obtained with difficulty. The use of morphine is most reprehensible, though its efficacy is undoubted. Antipyrin, antifebrin, and phenacetin have taken the highest stand as anodynes in this disorder, and their efficiency is sometimes markedly increased by being combined with codeine. Extract of cannabis indica (gr. ¼ to ½) is frequently of service. Massage, ice-bags, or hot applications are at times beneficial. Frequently much good is experienced from the use of sinapisms or even the actual cautery. The value of electric cataphoresis has already been alluded to.

Atropine or belladonna is useful for urinary incontinence, and strychnine for vesical weakness. Vesical and rectal neuralgias yield to suppositories containing opium or codeine, combined with belladonna. In severe visceral crises it sometimes becomes necessary to employ morphine hypodermically, which affords immediate relief. Nitrite of amyl or nitroglycerin act well in laryngeal crises.

When the bladder is not perfectly emptied, daily catheterization will become necessary.

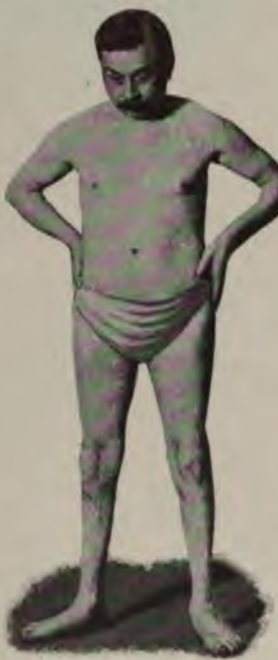
Suspension by means of the suspensory spinal apparatus, employed originally for putting on plaster-jackets, was recommended two or three years ago for locomotor ataxia, and at one time had quite a vogue. Undoubtedly the slight stretching of the spinal cord produced in this way altered at times the chronic degenerative process going on in the roots and columns, so that symptoms were in many cases considerably improved by the method. Bonnuzzi ascertained by careful experiment that this cord-stretching could be carried out much more efficiently and with greater safety by a method of his own than by suspension, The method is briefly this: The patient lies upon his back upon a couch. The operator then lifts up his two legs, the knees being kept extended, and flexes the thighs as far as possible over upon the abdomen. This is done at first gently, as it is painful, and but for a few seconds, several times each week. Gradually the flexion is increased and the duration of the process prolonged.

FRIEDREICH'S ATAXIA.

Friedreich's Disease; Family Ataxia; Hereditary Ataxia; Generic Ataxia; Friedreich's Form of Locomotor Ataxia; Hereditary Ataxic Paraplegia.

Hereditary ataxia would seem to be the best term for this disease were it not for the recent description of another form of hereditary ataxia, which, however, the designation "cerebellar heredo-ataxia," suggested by Marie, will probably serve to distinguish. The pathology of the two forms of hereditary ataxia would seem to be quite distinct, for in Friedreich's disease the lesions are spinal, in the other variety cerebellar. Friedreich first described the form to which his name is attached in 1861. It is a species of combined sclerosis, differing from other and similar systemic degenerations of the cord in the early age at which it begins to be manifested, in its tendency to occur in families, and in certain additional characteristics which will be described under symptomatology.[1]

FIG. 219.



Case of Friedreich's ataxia showing attitude and atrophy of muscles of legs. (Philadelphia Hospital.)

Etiology. The family character of the disease is one of its characteristics. Direct inheritance is rare. Cases are apt to occur in brothers and sisters in one family, sometimes in paternal and maternal uncles or aunts also. As many as ten cases in three generations of a single family have been reported. Sometimes the parents are perfectly healthy; often some degenerative factor is found in ancestors or collaterals, such as alcoholism, chorea, syphilis, epilepsy, or other disorders. Isolated cases are discovered at times. Males and females are about equally affected, taking all recorded cases together, but occasionally in some particular family only the males suffer, all the females escaping, or *vice versa*.

As regards age, most cases are apt to develop symptoms of the disease between the ages of six and fifteen, probably most frequently between six and eight. The extremes in recorded cases have been two years and twenty-four years of age. One solitary instance is given as beginning at the age of sixty-six years.

Acute infectious diseases are reported among antecedent exciting factors.

Symptomatology. The earliest and the cardinal symptom is ataxia, a gradually developing inco-ordination at first most noticeable in the lower, later in the upper extremities. It affects the gait, and in standing the feet must be kept wide apart. As in locomotor ataxia, closing the eyes increases the swaying, though in exceptional instances this is not the case. Frequently a tendency to fall easily and to stumble in walking is first observed. Later on a jerky inco-ordination in the arms is noticed. There are usually no pains in the extremities or other sensory disturbances, but sometimes rheumatoid pains are complained of. The knee-jerks are always absent in typical cases very early in the disease. As time goes on the neck muscles participate to a greater or less degree in the ataxic movements, and a sort of jerky tremor is manifested. The tongue may also be included, and the speech takes on a peculiar character in the course of a few years—a tendency to elision and hesitation in the enunciation of sentences. It is not unlike the scanning or staccato utterance of multiple sclerosis. Nystagmus, either vertical or lateral, and developed, as a rule, only upon movement of the eyes upward or laterally, is present in most cases. It is seldom noticed when the eyes are at rest. As a rule, the pupils react normally to light. There is never optic-nerve atrophy. There are never trophic changes in the skin, or joints, never trouble with the vesical and rectal sphincters. There is often vertigo. In the later stages there may be paraplegia, contractures (talipes equinus or equino-varus), lateral spinal curvature, and some muscular wasting. The progress of the malady is exceedingly slow and gradual, as a rule, and may remain at a standstill for years. Death may occur at the end of ten or twelve years or not for thirty years. The usual cause of death is some intercurrent disease not especially related to the chronic disorder.

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[1] See Brain, 1890, article by Ladame, for full bibliography.

In isolated cases atypical symptoms have been at times observed, such as diplopia, lost pupillary reflexes, frequent pulse-rate, vasomotor disturbances, headache, anæsthesia, difficult micturition, diminished electrical contractility, impotence, glycosuria, and imbecility.

There are never visceral crises or mental disturbances.

The chief peculiarities in typical cases may then be summarized as follows:

1. Ataxic gait and jerky inco-ordination of the upper extremities.
2. Slow and jerky articulation.
3. Absent knee-jerks.
4. Nystagmus.
5. Scoliosis and talipes equino-varus.
6. Occurrence in several members of the same family.
7. Inception before the age of puberty.
8. No sensory disturbances, no pupillary disorders, no mental disorder.

Pathology and Pathological Anatomy. The essential lesions are in the spinal cord—sclerosis of the posterior columns and of the pyramidal tracts—in fact, a combined sclerosis, such as is also noted in ataxic paraplegia. The posterior columns are usually more affected than the lateral and anterior. As in tabes, the posterior nerve-roots are apt to be involved in the degenerative process. Sometimes the adjacent direct cerebellar tract is included in the lateral sclerosis. There have been some variations in the pathological findings in the many autopsies that have been made, but those just given are the chief features in all. The process may not be equally intense in all parts of the cord. Atrophy of the posterior vesicular columns has been observed. Occasionally slight changes in the ganglion cells of the anterior horns have been seen. The pia over the posterior columns is at times thickened. Thickening and induration of the pons and medulla, with atrophic changes in the cells of the post-pyramidal nucleus and in the restiform bodies, have been reported. It has been also stated that the cord is at times congenitally imperfect. Two central canals have been noted. There are no changes in the brain and rarely in the peripheral nerves.

It has been suggested, for various reasons, that the disorder is a manifestation of a sort of arrest of development, so that the nerve tissues involved have a lower vitality than other structures, and thus tend to degeneration early in life. It has also been supposed that the true pathology is a tendency to a proliferation of the interstitial neuroglia by which the nervous elements are forced to succumb.

Diagnosis. In the first place it is necessary to distinguish cerebellar heredo-ataxia from Friedreich's form of hereditary ataxia. The former, due to atrophy of the cerebellum, is a peculiar and rare syndrome whose characteristics may be drawn from a study of the cases recorded by Fraser, Nonne, Sanger Brown and Klippel, and Durante. I give the contrasting and corresponding symptoms of the two disorders in the following parallel;

Friedreich's Disease.	*Cerebellar Heredo-ataxia.*
1. Ataxia.	1. Ataxia.
2. Disordered speech.	2. Disordered speech.
3. Absent knee-jerks.	3. Normal or exaggerated knee-jerks.
4. Nystagmus.	4. Nystagmus.
5. Normal pupils.	5. Argyll-Robertson pupils.
6. No ocular symptoms.	6. Limitation of the field of vision, dyschromatopia, optic-nerve atrophy often.
7. Scoliosis and club-foot.	7. No scoliosis or club-foot.
8. Heredity.	8. Heredity.
9. Inception before the age of puberty.	9 Inception after the age of twenty.

From locomotor ataxia, the early age of onset, nystagmus, defect in articulation, and absence of pupillary symptoms ought to render the differentiation of Friedreich's disease easy.

The exaggerated knee-jerks, age of onset, and the like sufficiently distinguishes ataxic paraplegia, although the pathological changes in the two diseases are very similar.

In disseminated sclerosis we have the similar symptoms, nystagmus, disturbance of speech, and inco-ordination of muscles, but the reflexes are exaggerated, the progress of the disease is more rapid, and it is isolated, not hereditary. Besides, the utterance is quite different in the two maladies.

In cerebellar tumor there is optic neuritis, headache, vertigo, vomiting, though in other respects there may at times be considerable similarity of symptoms.

Prognosis. The disease is progressive and incurable, but continues often for many years without very distressing symptoms. Death usually occurs from intercurrent disorders.

Treatment. Little or nothing can be done in so unfavorable a complaint. Arsenic and nitrate of silver may be tried. Electricity may be helpful at times, and injections of phosphate of soda may ameliorate occasionally some of the symptoms.

COMBINED SCLEROSIS.

Ataxic Paraplegia; Progressive Spastic Ataxia; Combined Lateral and Posterior Sclerosis.

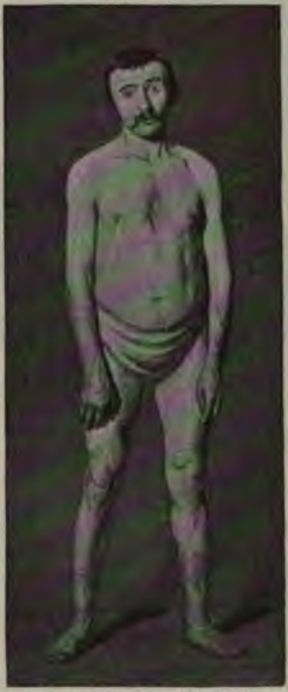
In this disease we have a combination of lateral sclerosis (spastic paraplegia) and posterior sclerosis (ataxia), to which the name ataxic paraplegia is therefore properly applied. The symptoms accordingly partake more or less of the nature of locomotor ataxia and of primary lateral sclerosis, sometimes more akin to one than the other, but always affording a sufficiently clear clinical picture to distinguish this syndrome from either of the others. There are occasional irregular forms such as one described by Putnam and Dana as a combined sclerosis with terminal softening.

Etiology. In one-tenth of the cases there is a neuropathic hereditary taint. Syphilis is very rare as a causative factor. It is more common in males than in females. It is a disease of adult life; overexertion, exposure, traumatism to the spine, and sexual excess have been described as antecedents in the history of this disorder.

Symptomatology. The malady is slow in onset, though occasionally a few months only instead of a few years are required for the manifestation of the typical symptoms. Either ataxia or spastic rigidity of the lower extremities may appear first. A certain amount of paresis will be found on examination of the legs. There is a sense of fatigue after even a short walk, and the unsteadiness becomes more or less marked on walking with the eyes closed or in the dark. The Romberg symptom, or "tabetic swaying," is present. There is stiffness of the legs, so that the gait seldom resembles the high, stepping movement of tabes. There may be dull pain or numbness in the lower extremities, but almost never the lightning pains of locomotor ataxia. On the other hand, dull pain in the back or sacrum is common, and is frequently an early symptom. The girdle sensation is very exceptional. There is no anæsthesia of the extremities or trunk. The chief difference from tabes lies in the exaggeration of the knee-jerks and the usual presence of ankle-clonus. The knee-jerk is generally so great as to be obtained by striking the top of the patella or the belly of the quadriceps extensor. Often the arms are involved, and in such cases weakness, inco-ordination, and exaggerated wrist and elbow-jerks are elicited. There is no atrophy of muscles anywhere.

The sphincters of the bladder and rectum are not always affected, but frequently there is difficult micturition. Sexual power is often impaired at an early period.

FIG. 220.



Attitude in a case of combined sclerosis. (Philadelphia Hospital.)

We do not usually find the Argyll-Robertson pupil in this malady, but occasionally the iris reflex is lost as in tabes. Atrophy of the optic nerve is extremely rare, and the ocular muscles do not suffer. The mental state is normal.

With the progress of the disorder, the muscular paresis and rigidity become more marked, and the inco-ordination rather less noticeable, so that the condition is very similar to that of spastic paraplegia. The patient may become bedridden after a time, but the progress of the disease extends over a period of years usually. The above-described symptoms are those of a typical case, but slight variations, giving a nearer resemblance to tabes, sometimes occur. Visceral crises are never observed.

Pathology and Pathological Anatomy. The actual cause of the setting up of the degenerative process is unknown. The fact that the posterior and lateral columns both are involved explains the peculiar combination of symptoms, some belonging to tabes and some to primary lateral sclerosis.

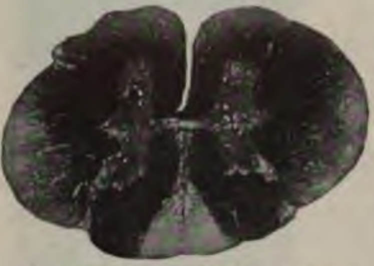
The condition of the posterior columns differs slightly from that in tabes, in that the lumbar and dorsal portions of the cord may suffer equally, or the dorsal even more than the lumbar; and the intensity of the sclerosis is not apt to be so great in the root-zone of the postero-external column in ataxic paraplegia.

As to the condition of the lateral columns, while the pyramidal tracts are the parts chiefly affected, the sclerotic process is often diffused into the mixed zones of the lateral columns, the lateral limiting layers, and even the direct cerebellar tracts. The direct anterior tracts are more or less diseased in nearly all cases.

There is no morbid change in the gray matter or membranes of the cord.

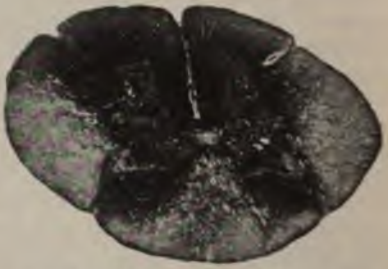
Diagnosis. There should be no difficulty in recognizing the nature of the malady when fully developed. We must remember that at first there is ataxia, and that the spastic condition develops later on in its course. Hence in the incipient stage a possibility of its being tabes is to be borne in mind. The exaggeration instead of abolition of the knee-jerk will exclude this. The inco-ordination present at some period of the disorder should eliminate primary lateral sclerosis.

FIG. 221.



Combined sclerosis. Mid-dorsal region.

FIG. 222.



Combined sclerosis. Same case as preceding and from a lower level in the cord (about D 11 o 12), where the degeneration is more extensive in both sets of columns.

If we recall some of the chief symptoms of Friedreich's ataxia, such as hereditary or family character, onset in early youth, loss of knee-jerk, nystagmus, disordered speech, scoliosis, and club-foot, we can scarcely confuse this disorder with the one under consideration.

In cerebellar heredo-ataxia the distinctive symptoms are nystagmus, Argyll-Robertson pupils, limitation of the field of vision, occasional optic atrophy, dyschromatopsia, heredity, and disordered speech.

In tumor of the middle lobe of the cerebellum there is sometimes a considerable gross resemblance to ataxic paraplegia, but headaches, vertigo, vomiting, and optic neuritis will serve us in the differential diagnosis here.

Some cases of myelitis, diffuse or local, and chronic, may simulate closely ataxic paraplegia and render the diagnosis very difficult. In myelitis the course of the disease is more rapid as a rule, and there is a regressive instead of a progressive tendency.

Prognosis. The chronic course, running over a period of years, with occasionally arrest of the disorder for a time, is to be remembered. Recovery never takes place. Death, as a result of intercurrent affections, or from septicæmia from bed-sores, or from renal disease due to overdistention of the bladder, is the usual termination.

Treatment. Warm baths and residence in a warm climate are indicated for the relief of the spastic symptoms. Where there is a specific history the iodides and mercurial inunctions should be used. Electricity is of no service. Strychnine or nux vomica should not be employed. The bromides are occasionally of service to alleviate trembling and twitching, especially at night. Arsenious acid may be given as in spastic paraplegia.

LESIONS OF THE CONUS MEDULLARIS.

The conus medullaris, or, as it is sometimes called, the conus terminalis, is the lowest portion of the spinal cord, lying below the lumbar enlargement. It is club-shaped. From it passes forth the long narrow prolongation known as the filum terminale.

The conus medullaris is probably the true spinal centre for the vesical and anal sphincters. At least, that seems to be the fact from the results of isolated lesion of this particular part of the cord. Such lesions are extremely rare, for, as a rule, the cauda or a part of the lumbar enlargement is apt to be implicated in any lesion affecting the conus. Disease of the conus alone would therefore involve only the functions of the bladder and rectum. There would be no disturbance of sensation and no motor disability.

Some of the cases described as pure conus lesions have not been such altogether. Thus, a case of Lachmann's, frequently referred to in literature as a conus lesion, is described in the *Arch. für Psych.*, vol. xiii. p. 50, under the title of "Glioma in the Upper Part of Filum Terminale with Isolated Compression of the Nerves of the Bladder" (*Gliom im obersten theil des Filum terminale mit isolirte Compression der Blasennerven*). The case was one aged forty-six years, with difficult urination and constipation of two years' duration. Upon examination there were found retention of urine, constipation, exaggerated knee-jerks, occasional fibrillary twitchings in the calf muscles, and lumbar pain. At the autopsy the dura around the cauda was distended. The conus was merged into a tumor 6.5 c.cm. long. The tumor did not include the nerves of the cauda and did not extend to the extreme end of the filum terminale. There were no changes in the lumbar or dorsal portions of the spinal cord.

A case reported by Oppenheim (*Arch. für Psych.*, vol. xx. p. 298), under the title, "On a Traumatic Lesion Limited to the Conus Terminalis of the Spinal Cord" (*Ueber eine sich auf den Conus Terminalis des Rückenmarks beschränkende traumatische Erkrankung*), is interesting. It was as follows: A man, aged twenty-four years, fell from a height, striking upon the sacrum. He was unconscious for a short time. For a brief space there was a dead feeling and weakness in the limbs and retention of urine; then incontinence of urine and feces, which persisted. There was lack of power of erection and some loss of sensation about the anus. Upon examination there were found tenderness and gibbus over the first and second lumbar vertebræ. There was no paralysis and no atrophy. The knee-jerks were active. There was ankle-clonus. Paralysis of the bladder and rectum was complete. There was anæsthesia about the anus, over the nates slightly, in the perineo-scrotal region, on the penis, and a narrow band on the posterior part of the inner surface of the thigh. The anæsthesia was to all kinds of sensation, especially for pain. It was limited above by a line drawn across the middle of the sacrum, and externally by the depression between the tuber ischii and trochanter major. The patient died three and one-half months later from cystitis and pyelonephritis. The conus medullaris was put in celloidin and cut from below upward. In the lowest part the whole of the posterior portion was destroyed. The anterior horns and anterior horn cells and pyramidal tracts were only partially injured. These changes became less marked in the sacral portion of the cord, and in the lowest lumbar portion there was only an ascending degeneration.

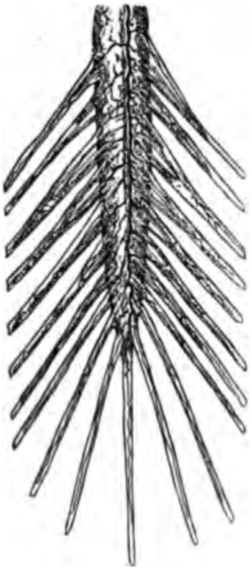
It is seen then that this case of Oppenheim's is also not a pure conus lesion, although his title would indicate it. His pathological description, however, shows the involvement of other parts than the conus, and the clinical syndrome presented evidence of disturbance in the cauda or in the sacral cord.

While the evidence is not at all conclusive, we may safely assume for the present the truth of the statements made above, that in the conus medullaris lie the centres for the bladder and rectum, and that isolated lesions here will cause no disturbance of sensation or motion.

LESIONS OF THE CAUDA EQUINA.

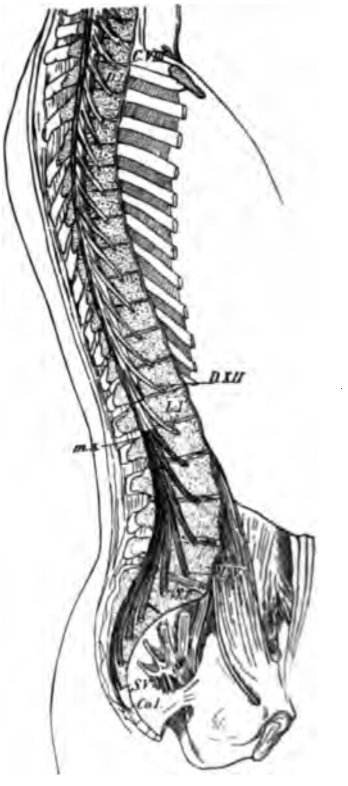
The spinal cord terminates in the spinal canal at the upper level of the second lumbar vertebra. The lumbar enlargement of the cord is situated above this point. From this lumbar enlargement spring the lumbar, sacral, and coccygeal nerves and filum terminale which enter into the formation of the cauda equina, as shown in Fig. 223. The spinal canal extends some nine or ten inches beyond the lower end of the cord, and this space is filled by the long nerve-roots which constitute the cauda equina. Fig. 224 gives an adequate idea of the termination of the cord at the inferior level of the first

FIG. 223.



Lumbar enlargement and conus with lumbar and sacral nerves which form the cauda equina.

FIG. 224.



Nerves forming cauda equina shown heavily shaded. Point of termination of cord at second lumbar vertebra shown.

lumbar vertebra and of the occupation of the rest of the space in the spinal canal by the nerves springing from the lumbar intumescence of the cord.

The cauda equina thus contains most of the nerves which have to do with the motor, sensory, trophic, and reflex functions of the lower extremities, bladder, rectum, and genital organs. These nerves represent the functions of the segments from which they spring, and any lesion of them affects the above-named functions in the same manner as similar lesions in the spinal centres of the corresponding nerves.

It is, therefore, not always easy to differentiate cauda lesions from spinal cord lesions. A study of the anæsthetic areas helps us very little in this respect, since, for instance, lesions affecting the sacral nerve roots produce the same areas as do lesions of the sacral part of the cord, together with paralysis of the sphincters of the bladder and rectum. A lack of symmetry on the two sides in the distribution of the paralyses and of the anæsthesias would be strongly presumptive of a cauda rather than of a cord lesion. An exceedingly slow and irregular development of the symptoms would also be suggestive of a cauda lesion. The fact is that the cauda and the lower part of the cord are often involved together in the pathological process. We owe a great deal to Thorburn, Starr, and others who have helped to elucidate the clinical, diagnostic, and pathological features of such lesions.

It has been demonstrated that pressure on the cauda equina affects to a greater degree the nerves in the middle part of the cauda than those near the surface (Thorburn). The lower nerve-roots of the cauda are nearer the middle line than the nerves which pass out above them; and when pressure is brought to bear upon the cauda the nerves which pass out at the lower levels are apt to suffer more seriously than those emerging above them. So, too, in lesions, such as hemorrhage or compression from fracture-dislocation, when improvement may be expected to take place, the upper roots tend to show signs of betterment soonest, and may indeed recover completely, while the lower ones remain as before. Pressure upon the nerves of the cauda is often sufficient to produce widespread paralysis when sensation is but slightly affected (Starr). This is often of value in the differential diagnosis, since lesions of the same magnitude in the lumbar enlargement of the cord would produce only a moderate amount of paralysis depending upon the segment or segments involved. (See section upon the Structure and Functions of the Spinal Cord.)

In traumatic conditions the external evidence of fracture or dislocation below the first lumbar vertebra, affecting the lumbar or sacral portions of the spinal column, is an important indication of the involvement of the cauda equina.

Pain and tenderness in the same regions, especially the sacral, have often been noted in cases of cauda lesion. This is particularly true of neoplasms.

In cauda lesions the sciatic and pudic nerves, and sometimes the anterior crural and obturator, are prone to suffer with especial frequency. Whether the degenerative reaction in the muscles supplied by the affected nerves will be present depends naturally upon the amount of damage done them, which is extremely variable.

As to the reflexes, the knee-jerks will be absent if the nerves to the anterior thigh muscles are affected. In no case of purely cauda lesion will they be exaggerated. The plantar reflexes are usually absent. The extent of the lesion in the cauda equina will determine to what degree the sphincters are disturbed. Disorder of the mechanism governing the sphincters of the bladder and rectum will depend upon whether their particular nerves are involved and to what degree. Urinary retention with overflow incontinence is not very frequent, and when it does exist may be recovered from as pressure diminishes. The presence of complete paralysis of the bladder and rectum is rather suggestive of coincident lesion of the conus medullaris.

Bed-sores and sometimes perforating ulcers have been noted in a considerable proportion of cases.

In addition to the necessity of excluding cord lesions, it is necessary to avoid confusion with peripheral nerve lesions, such as multiple neuritis and disorders of the lumbar and sacral plexuses.

Ordinary multiple neuritis should present no difficulty in the way of differentiation, but a bilateral lumbo-sacral neuritis may offer obstacles to diagnosis for some time, at least until absolutely characteristic symptoms have appeared.

In other lumbo-sacral plexus lesions, which are almost always entirely unilateral, a bilateral manifestation of symptoms should favor a diagnosis of cauda disorder, though in some very rare instances an involvement of the cauda upon one side might for a time give rise to confusion.

Occasionally a lesion of the cauda (tumor) may for a time simulate locomotor ataxia by presenting some of the symptoms common to both, viz.: loss of knee-jerks, disorder of the bladder sphincter, sharp pains radiating down the legs, and peculiarity of gait. But there will be no ataxia, and pupillary symptoms will be wanting, while the supervention of atrophies and anæsthetic areas will in the course of time demonstrate the presence of a cauda lesion.

The most common of all pathological processes affecting the cauda equina is fracture-dislocation of the lumbar spine, which either compresses or crushes the nerve-roots, or both; moderate dislocation of the vertebræ may cause little damage because of the large size of this portion of the spinal canal and the relatively small part of it occupied by the bundle of nerve-roots. The symptoms will vary with the extent and degree of the injury.

Hemorrhage is another, though infrequent, lesion of the cauda. Caries of the lumbar vertebræ may induce compression of the cauda.

Rather more common than these are neoplasms. Among the tumors which have been described as developing in this part of the vertebral canal and affecting the cauda equina are sarcoma, fibro-sarcoma, meningocele in spina bifida, gumma, cavernous angioma, and multiple neuromata.

The chief characteristics of tumor compression of the cauda equina are slow and progressive development of the atrophic paralyses, reflex disorders, and anæsthesias peculiar to the region; intense and increasing sacral pain of a radiating character, and tenderness often very marked in degree on the sacrum. Sometimes the growth of the neoplasm may be temporarily arrested, and more rarely undergo retrogression.

From a study of the above facts in connection with the section on Structure and Functions of the Spinal Cord in another part of this chapter the chief points in the diagnosis of cauda lesions should be clear. A glance at the following tabulation of a few of the noteworthy cases in literature may also prove useful:

Author.	Reference.	Nature of lesion.	Motor symptoms.	Area of anæsthesia.	Sphincter symptoms.	Reflexes.
Thorburn,	Surgery of Spinal Cord	Dislocation 1st lumbar vertebra.	Paralysis of all muscles below knee; weakness knee flexors and adductors, abductors, and extensors of thigh; reaction degeneration.	Complete anæsthesia of back of thighs and lower part of buttocks, of outer sides of legs and of the feet; also of perineum, penis, and scrotum.	Retention of urine and incontinence of feces.	Knee-jerks and plantar reflexes absent.
Thorburn,	Surgery of Spinal Cord	Spina bifida.	Atrophy and paresis of legs below knee; pseudo-tabic gait; occasional fibrillary twitchings and choreiform movements.	Loss sense of position in right leg, anæsthesia over buttocks, backs of thighs, backs of legs, and whole of feet, also inner side thighs and legs; and perineum, scrotum, and penis.	Difficult urination, sometimes incontinence of feces.	Knee-jerks and plantar reflex absent; cremasteric, abdominal, and epigastric present.
Thorburn,	Surgery of Spinal Cord	Fibro-sarcoma of cauda.	Wasting and paresis of lower limbs.	Anæsthesia same distribution as last, save that inner sides of feet escaped. Sacral pain.	…………	Knee-jerks active. plantar and cremasteric exaggerated.
Thorburn,	Surgery of Spinal Cord	Dislocation 2d lumbar vertebra.	Paresis lower extremities; paralysis at ankle and foot; atrophy and degenerative reaction.	Anæsthesia over lower gluteal regions, down backs of thighs to soles of feet and over front of legs and dorsum of feet: genitals included.	Difficult micturition; constipation.	Knee-jerks and plantar reflexes absent; cremasteric and gluteus exaggerated.
Westphal,	Char. Ann. l. 421.	Gumma with hemorrhagic exudate.	None.	Anæsthesia of genitals, perineum, anus, and buttocks.	Paralysis of sphincters.	
Starr and Lloyd,	Am. Journ. Med. Sci., civ. p. 15.	Fracture lumbar vertebræ.	Atrophic paralysis of peronei, ant. and post. tibial group, and glutei of right leg.	Saddle-shaped anæsthetic area, but extending down right leg behind in a narrow strip to sole of foot.	Paralysis of sphincters.	
Starr and McBurney,	Am. Journ. Med. Sci, civ. p. 15.	Fracture lumbar vertebræ.	Atrophic paralysis of all muscles of both lower extremities.	Almost complete loss of sensation over whole of both legs.	Paralysis of sphincters.	Lost knee-jerks.
Herter,	New York Med. Journ. Aug. 22, 1891.	Old hemorrhage and areas of inflammatory material in cauda and involving ¾ inch of lower tip of cord.	Atrophic paralysis of both legs below knees; thighs could be moved.	Anæsthesia over buttocks, perineum, genitals, and extending slightly down backs of thighs; anæsthesia also over backs of both legs from half-way below knees down over heels and soles.	Paralysis of sphincters.	
Shaw and Bush,	Brit. Med. Journ., vol. xi. No. 41.	Old hemorrhage opposite 4th and 5th lumbar spines.	Atrophic paralysis of hamstring, peroneal, calf, and tibial muscles.	Anæsthesia of anus, perineum, buttocks, back, and inner sides of thighs, popliteal spaces, calves, lower parts, front of legs, outer side left leg, entire feet except inner surfaces, also of urethra and rectum.	Sphincters paralyzed.	Right knee-jerk normal left active; plantar absent; cremasteric present.

Illustrated cases might be multiplied, but the above selected at random will serve to emphasize some of the features of cauda lesions considered.

Treatment. Almost all of these cases fall as regards treatment within the province of the surgeon. There are few cases in which operations to relieve pressure from fracture or dislocation, or both, or to remove neoplasms, are not justifiable, and from which improvement, or rarely recovery, by surgical procedure may not be hoped for. There have already been a sufficient number of operations undertaken for this purpose to demonstrate the truth of this statement.

The cause having been removed, the general treatment should be conducted on the same principles as guide us in the management of sensory disturbances, atrophic paralysis, and sphincter disorders from lesions elsewhere. The use of the faradic brush for anæsthesias, and of both faradism and galvanism for the wasted and paralyzed muscles, is indicated; massage, active and passive movements, and hydriatric applications are of service. General tonics, such as arsenic, iron, and strychnine, should be employed.

CHAPTER XXI.

BULBAR PALSY.

By FREDERICK PETERSON, M.D.

Bulbar Palsy is an associated paralysis and wasting of muscles of the lips, tongue, fauces, and pharynx, due to disease in the "bulb," or medulla oblongata, of the nuclei of nerves supplying these parts.

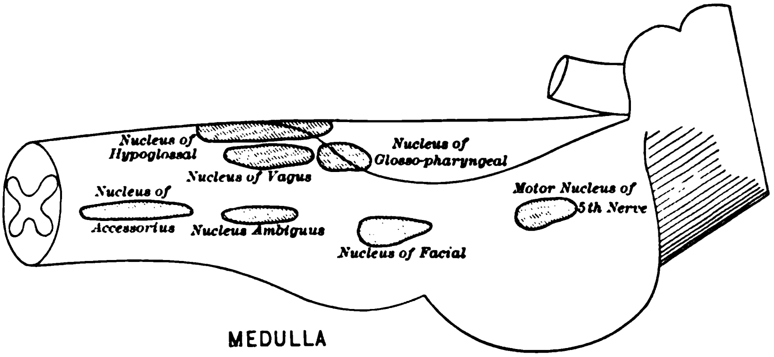
Usually the process is a chronic degenerative one, corresponding to that in the ganglion cells of the anterior cornua of the spinal cord. There are cases, however, in which the onset is acute, either apoplectiform in character (softening) or inflammatory. The symptoms are closely simulated at times by bilateral cerebral softening, and cases very like bulbar palsy have been reported where the lesion existed in but one of the hemispheres (pseudobulbar paralysis).

The nerves affected by the disease are some of the facial fibres (orbicularis oris), the hypoglossal (tongue), spinal accessory (larynx and palate), glossopharyngeal, and vagus (pharynx).

CHRONIC BULBAR PALSY.

Etiology. The disease in most cases manifests itself after the age of forty years, though cases have been reported at a much earlier period of life, even in childhood. It is more frequent in men than in women. Some direct or indirect neurotic heredity is sometimes to be traced, and a family predisposition has been noted in cases occurring in early life. Mental strain,

FIG. 225.



Schematic representation of bulbar nuclei involved in bulbar palsy.

exposure, overuse of muscles (observed by Stein in a clarionet player, and by the writer in a cornet player), lead, perhaps syphilis, are among probable causes. Elsewhere is mentioned its development as a part of a progressive muscular atrophy. Gowers relates a case developing as a sequel to diphtheria.

Symptomatology. The disease is very slow in its onset, and usually the first indication of any trouble is a weakness of the tongue, rendering the pronunciation of lingual and linguo-palatine consonants, *d, l, r, n, t,* difficult. The articulation of these letters at first becomes indistinct only after fatigue of the muscles, but later on is noticeable at all times. Occasionally some pain in the back of the head has been complained of antecedent to the change in speech. Following closely upon the weakness of the tongue, the lips begin to lose power, and the letters *o, u, p, b,* and *m* become difficult to enunciate distinctly. The patient cannot whistle. Coincident with this, or more often following this, there is some trouble in swallowing. The patient begins to drool. The palate grows weak, increasing the difficulty of speech, giving the voice a nasal sound, and allowing liquids to regurgitate through the nares in attempting to swallow.

The tongue can no longer be thrust forward, and becomes more or less atrophied and rugose. The mouth remains open. Other muscles about the mouth besides the orbicularis oris may suffer. As a rule, the zygomatici escape, and, by contracture, deepen the naso-labial folds. It becomes impossible to understand any words the patient attempts to utter. The muscles of the vocal cords are the next to suffer. Swallowing becomes more and more difficult. Solids cannot be taken at all, and are apt to get into the larynx. Liquids run out of the mouth and nose. Hence, semi-solids are best taken. The face of the patient takes on a look of great distress that is very characteristic. Sensibility is nowhere lost, though the pharyngeal reflex disappears. As in the tongue, atrophy may be noted in the lips, and an electrical reaction of degeneration may be at times found. Sometimes there is a marked gross tremor (not fine as in general paresis) in attempting to speak. Occasionally a markedly increased jaw-jerk, or even jaw-clonus, is observed. The mind is not affected, but the patient may be very emotional. Frequency of the pulse has been observed in some cases, especially in the later stages.

The disease is found associated with progressive muscular atrophy and amyotrophic lateral sclerosis, and even lateral sclerosis, in their later stages, and occasionally it is noted in combination with ophthalmoplegia.

Course of the Disease. The malady is gradually progressive, though an arrest of its progress for weeks or months may take place. It usually lasts for from two to four years. Patients are apt to emaciate from lack of sufficient nourishment, and inanition is a common cause of death. Pneumonia is often set up by particles of food slipping into the air-passages (*Schluck pneumonie*).

Pathology and Morbid Anatomy. As in other degenerations of the nervous system, the actual cause of this particular manifestation of disease is unknown. It is probable that some toxic condition of the blood has an elective affinity for these particular groups of cells, which are functionally associated, and which have, therefore, a similar constitution and vulnerability. The morbid process is essentially the same as that affecting homologous structures elsewhere (as in progressive muscular atrophy).

To the naked eye the medulla appears to be normal, but upon microscopical examination characteristic changes are found in the nuclei of the nerves mentioned and in the fibres passing off from them within the medulla. The nerve-cells are shrunken and lose their processes, and granule-corpuscles, together with other degenerative elements, are found in the interstitial tissue.

Sometimes the vascular walls are thickened and the connective tissue increased. While such nuclear changes are observed in a typical case, there are many variations from this typical picture in the degree of degeneration and character of changes. It has been suggested, indeed, that there are parenchymatous and interstitial forms of bulbar palsy. The most marked alterations are noted in the nuclei of the hypoglossal, glosso-pharyngeal, spinal accessory, and vagus nerves. Besides these nuclei, some of the cells of the facial nerve and those of the nucleus ambiguus may be affected, rarely the nucleus of the fifth nerve. Degeneration is often to be seen in the anterior pyramids. When bulbar paralysis is associated with any of the spinal-cord diseases already mentioned, or with ophthalmoplegia, we discover in addition atrophic processes in the cells of the cord and in the oculomotor nuclei. Degeneration of fibres in the raphé in the loop of the facial nerve, and in the posterior longitudinal fibres, has been noted. The involvement of the orbicularis oris is not yet clearly understood, though it is believed that orbicularis fibres may descend to or even originate in the hypoglossal nucleus. The close functional relation of the tongue and lip movements would suppose some close central anatomical relation. Degeneration of the anterior pyramids in the pons and crura has also been observed. The motor nerve-trunks degenerate and are gray and softened. The nerve-endings in the muscles are degenerated. As far as the muscles are concerned, there is found in a typical atrophic case the same pathological changes as in muscles in a progressive muscular atrophy of the Aran-Duchenne type. The fibres become narrowed and granular or fatty, and ultimately disappear from the sarcolemma sheaths. The interstitial tissue increases, the nuclei of the sheaths are multiplied, and there may be fatty infiltration and red-pigment deposits between them.

Duchenne described two forms of bulbar palsy, a paralytic and an atrophic. In the former class the wasting in the lips and tongue is not noticeable and the jaw-jerk is increased, so that sometimes even a jaw-clonus may be obtained, and in such cases there is slight or no degeneration in the nuclei, but marked changes in the supra-nuclear tracts leading to the cortex. In the atrophic variety we have the marked degeneration in the nuclei and infra-nuclear fibres leading to the muscles.

Diagnosis. Chronic bulbar palsy is to be distinguished from the acute form (polioencephalitis inferior, hemorrhage, softening) and from bulbar tumors, multiple sclerosis, and pseudo-bulbar paralysis. From the acute variety the slow and gradual onset is sufficient to differentiate this form. It is different, however, with bulbar tumors, which also run a chronic course, but these almost always present unilateral symptoms, whereas in bulbar palsy the manifestations are always bilateral. Then, too, with tumor we have headache and sometimes convulsion. Multiple sclerosis, while it may simulate by involvement of the medulla some of the symptoms of bulbar paralysis, is also apt to be unsymmetrical in its distribution and to present other signs sufficiently marked to establish the correct diagnosis.

Pseudo-bulbar paralysis has been caused by sclerosis and by acute lesions (hemorrhage, thrombosis) affecting the two hemispheres. The lesion in such cases tends to be first on one side and then on the other, and here, too, other symptoms (hemiplegia, diplegia, and the like) aid in the differentiation.

Prognosis. The disease is serious in the extreme, and almost invariably leads to death. It is possible that in some excessively rare cases the progress of the malady may be arrested wholly or for a long period.

Treatment. Although little can be anticipated from any means of treatment at present known to us, efforts should be made to alleviate some of the symptoms, and some hope may be entertained in any case of a possible arrest of the progress of the disorder. As to general measures, the patient should avoid overexertion of any kind, be well-fed, and receive massage and electricity. The last does not offer much encouragement; but cases do occasionally reveal improvement in the use of some of the muscles after its employment. As the muscles respond to both galvanism and faradism, it is well to make use of both currents in moderation two or three times weekly, fifteen-minute séances, the currents being applied with a proper electrode to the lips, tongue, and pharynx. There is a reflex centre for swallowing immediately over the pomum Adami, which is easily excited by the cathode employed with an interrupting handle, the anode being placed at any indifferent spot, preferably the back of the neck. This reflex deglutition should not be repeated so often as to cause great fatigue.

As to drugs, various nerve tonics may be exhibited. Hypodermic injections of strychnine (gr. $\frac{1}{60}$) are useful. Arsenic, phosphorus, quinine, and nitrate of silver may be given with some little benefit. For the drooling of saliva atropine (gr. $\frac{1}{120}$ to $\frac{1}{60}$) may be administered, either by mouth or hypodermically, twice daily. Stimulant doses of morphine (gr. $\frac{1}{36}$ to $\frac{1}{24}$) subcutaneously have occasionally good effect.

The care of the nutrition of the patient, which is so apt to suffer, is important. The food must be given in a pulpy or semi-solid state, and when swallowing becomes so difficult as to become dangerous, the soft-rubber nasal or stomach-tube with funnel must be made use of and the patient given twice daily a liquid mixture of milk, raw eggs, whiskey or brandy if indicated, and meat juice. Peptonized foods per rectum may be employed instead of the stomach feeding, but is not as efficient.

ACUTE BULBAR PALSY.

It would perhaps be well, as has been suggested, to use the term "acute bulbar palsy" only for such cases as are due to an acute inflammatory change in the bulbar nuclei—polioencephalitis inferior acuta in contradistinction to polioencephalitis inferior chronica—but clinically this active inflammation is generally difficult to separate from other forms of bulbar paralysis characterized by sudden onset (the apoplectiform variety). The chief distinction is in the symmetrical bilateral character of the symptoms; in the apoplectiform cases it is the rule to discover irregularity of distribution of the palsies, as well as a wider range of symptoms, such as weakness and paræsthesiæ of the extremities. Thrombosis of a vertebral artery, rarely embolism, is the most frequent cause. The chief difficulty in diagnosis of the apoplectiform cases lies in the close resemblance to pseudo-bulbar paralysis; but a history of two distinct cerebral attacks, one on each side, should be looked for.

These cases are gravest at their onset, when a fatal result commonly occurs within a short time. The acute stage safely passed, there is amelioration of some of the symptoms, but degenerations take place to a certain degree at times, and the case comes to resemble the chronic form. Occasionally great improvement, and even recovery, takes place in apoplectiform cases. The prognosis is thus much better than in the chronic form.

As regards treatment, the apoplectiform variety should be cared for on the general principles laid down for acute softening in other parts of the brain; and the inflammatory group should be treated as are other cerebral inflammations. In the chronic conditions we employ much the same methods as were discussed above in the treatment of the chronic degenerative type of bulbar paralysis.

CHAPTER XXII.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

By FREDERICK PETERSON, M.D.

MULTIPLE sclerosis is one of the chronic degenerative nervous diseases in which the sclerotic process, instead of being limited to certain systems in the central nervous structures, is disseminated irregularly, in small patches or plaques, throughout the brain or spinal cord, and which is characterized by paralysis, spastic rigidity, tremor, peculiar disturbance of speech, nystagmus, and other symptoms, varying considerably according to the location of the sclerotic patches. The disease is more common than lateral or amyotrophic sclerosis. The pathological anatomy of the disease is thoroughly understood, although the actual cause of the sclerotic changes is as mysterious as in the other chronic degenerative disorders of the central nervous system. Three different groups, owing to the variability of the symptoms, have been made, and these are cerebral, spinal, and cerebro-spinal. The perfect type of multiple sclerosis is that in which the islets of the sclerosis are found both in the brain and spinal cord. Sometimes these types merge into one another; thus, a case of my own, which for three or four years was a perfectly typical one of lateral sclerosis, gradually became a typical cerebro-spinal disseminated sclerosis. Owing to the protean nature of the symptoms, depending, as they do, upon the localization of the patches in various parts of the central nervous system, mistakes in diagnosis are quite frequent, and other disorders are sometimes mistaken for multiple sclerosis.

Etiology. Both sexes are equally liable to this disease. As regards age, most cases are found in the early half of life, but the disorder may be observed at any age from childhood to senility. Hereditary influences play but a small part, though we may frequently trace an inherited neuropathic constitution.

The exciting causes are often difficult to discover, and perhaps, in the majority of cases, no exciting cause will be found. The disease has been ascribed to exposure, overwork, mental strain, traumatic lesion of the central nervous system, and to acute, infectious diseases, such as typhoid fever, diphtheria, smallpox, measles, and erysipelas. In some cases it has been believed to have developed after some acute or subacute inflammation in the central nervous system, such as myelitis. Syphilis seems to have little or no part in its etiology.

Symptomatology and Course. Owing to the wide diffusion of the patches of sclerosis throughout the central nervous system, we are certain to have islets somewhere in the motor tract in almost every case, and consequently we have some loss of power in one or more limbs, sometimes in all of the extremities. This amounts, usually, to a paresis, and not a paralysis.

Another very characteristic motor symptom is tremor of wide excursion, characterized by its development on intended movements only, and therefore called "intention tremor." It is not present when the parts are at rest, although occasionally, when it exists in the neck muscles, the head may show a marked tremor whenever the patient is erect or sitting up. In the legs there is seldom any particular tremor, although ataxia may be present, but usually we have a spastic paraplegia, so that the patient's gait is spastic and paretic.

Nystagmus is so common as to constitute one of the cardinal symptoms, and should be looked for in all cases. Usually this nystagmus is developed only on movement of the eyes. Nystagmus when the eyes are at rest is rather uncommon. It is generally a vertical nystagmus, or lateral, more seldom rotatory. Occasionally there are paralyses of cranial nerves. There is a peculiar disturbance of speech in most cases, the syllables of words being articulated separately, and sometimes in a peculiar accentuated manner, which has given rise to the term *staccato utterance.* The words "syllabic," or "scanning," are also used to qualify the articulation. At times there are attacks of vertigo or epileptiform or apoplectiform seizures. Sensory symptoms are occasionally present. It is not uncommon to note some slight dulness of the mind as the result of the disease, usually accompanied by a morbid placidity or complacency, quite out of proportion to the seriousness of the malady.

These are the chief features, and most of them are present in a typical case of multiple sclerosis. It may be well to enumerate again briefly these cardinal symptoms:

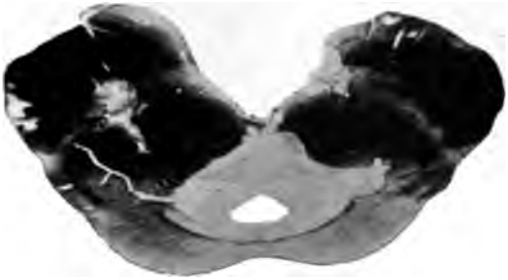
1. A muscular weakness, with rigidity, taking a hemiplegic, paraplegic, or monoplegic form, sometimes with cranial nerve paralysis.
2. "Intention tremor."
3. Exaggerated knee-jerks, wrist-jerks, with ankle-clonus, possibly jaw-jerk.
4. Nystagmus.
5. Various symptoms of nervous disturbances, like vertigo, headache, epileptiform and apoplectiform attacks, and mental dulness.
6. Disorder of speech.

These symptoms may gradually appear coincidently, or any one of them may precede the others, depending altogether upon the great variability in the mode of onset, and in the localization of the first patches of sclerosis. As a rule, the weakness and rigidity in the limbs appear before other symptoms. The jerky "intention tremor" is supposed to be due to resistance to conduction in nerve-fibres traversing the sclerosed patch. Occasionally this same sort of tremor is found in some cases of tumor of the brain, interfering with conduction in the motor path. Some authorities, however, believe, that it is necessary that the islets of disease should exist in some particular position, as in the pons, for instance, in order to affect the co-ordination of the muscles. We find cases, at times, which simulate very closely tabes, owing to the existence of a patch of sclerosis in the posterior columns. Sometimes the spastic condition of the legs is such that flexor contracture may take place, and the legs be drawn up permanently, but it is more common to have an extensor spasm. Muscular wasting is very uncommon, but it may occur from the sclerotic process affecting the gray matter of the anterior horns in some particular segment. The atrophy would then be local and irregular, depending upon the particular segment in which the lesion occurred. Sensibility varies to a great extent, according to the localization of the lesion. There may be hemianæsthesia from invasion of the sensory path in the brain by a patch of sclerosis, or the anæsthesia may be a small patch on one limb. When the disease has invaded the cord very generally there is apt to be considerable loss of sensibility in the legs. Paræsthesias are more common; feelings of numbness or prickling and tingling in the hands and feet; dull aching pains in the limbs, and in the back sharp shooting pains;

the girdle pain may occasionally be present also. Among the eye symptoms, in addition to nystagmus, we have other disturbances, such as impairment of sight in one or both eyes, or contraction of the field of vision, due to sclerotic processes occurring in the optic nerve of the chiasm. Sometimes complete blindness occurs in one eye; after a time atrophy of the optic nerves takes place, and can be observed with an ophthalmoscope. The Argyll-Robertson pupil is very rarely found in multiple sclerosis. External palsies of the eye are not uncommon, the lesions affecting the external recti, the converging muscles, and the superior rectus sometimes, but very rarely a single whole nerve, like the third; occasionally transient diplopia is met with, as in tabes. Among the other cranial nerves which may suffer from the disease are the seventh and fifth and hypoglossal. In some very rare cases there have been trophic disturbances, such as local œdema, changes in the growth of the hair and nails, arthritic troubles, and herpes.

ATYPICAL FORMS OF MULTIPLE SCLEROSIS. Sometimes the sclerotic patches are so few and so uniquely placed that they give rise to very unusual forms of multiple sclerosis. Occasionally, for instance, the lateral columns of the cord may be first affected for a year or two, making a condition simulating that of a primary lateral sclerosis. Sometimes a single island of sclerosis in the brain may give rise to a mono- or hemiplegia; for example in a case under my observation there is a monoplegia of the right arm, with typical rigidity and tremor, exaggerated reflexes, and so on, which have existed for several years, yet other symptoms have not developed to the present time. Distinctively spinal and cerebral forms of multiple sclerosis have been described. The condition known as "diffuse sclerosis" is one in which the connective-tissue elements are increased in large portions of the brain or cord, and not in small patches, as in the disseminated form which we have been considering. The diffuse sclerosis may involve any part of the cerebrum or cerebellum; may involve any portion of the white substance or the cortex, or a part or whole of the hemisphere, or both hemispheres. Sometimes this is associated with a diminution in the size of the parts affected, so that an atrophic sclerosis is spoken of. This sclerotic process is quite common in the brains of cases which have suffered from infantile cerebral palsy, and, undoubtedly, in most of these cases, the condition is due to antecedent meningeal hemorrhage, but embolism, thrombosis, possibly acute encephalitis, may give rise to the same condition. It is sometimes found as a result of congenital syphilis, and a diffuse sclerosis has been observed after long indulgence in alcohol. In many cases of idiocy

FIG. 226.

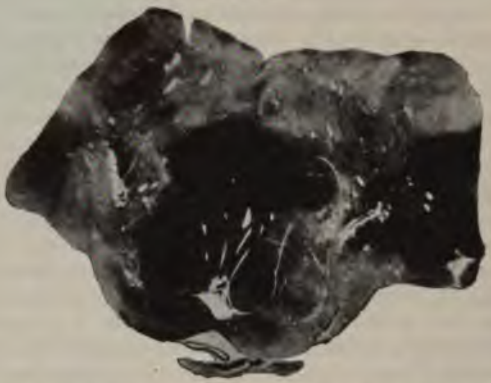


Multiple sclerosis. Section through crus. Unstained portions show distribution of sclerotic tissue. Weigert stain.

diffuse sclerosis has been found. Miliary sclerosis is a term that has been applied to minute patches of degeneration scattered throughout the nervous system, and almost microscopic in character.

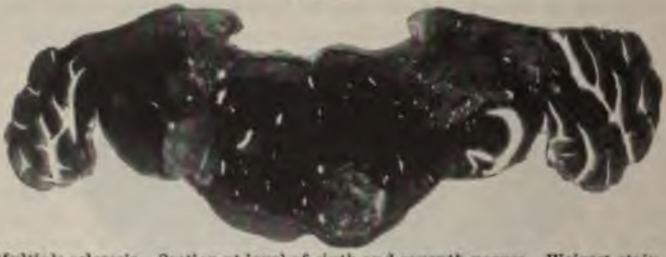
Pathology. Multiple sclerosis of the brain and spinal cord is one of the most striking pathological conditions to be observed in the central nervous system. On cutting into the parts, the grayish patches are visible to the naked eye in striking contrast to the tissues around them. They vary in size, from a line, or less, to an inch in diameter. Their consistence is firmer than that of the rest of the brain. It is impossible to understand what is the actual cause of the formation of these nodules of sclerotic tissue. It is strongly suggestive of the presence of some irritant or toxine in the blood, which stimulates the overgrowth of connective tissue about the smaller vessels. Under the microscope, we find the islets to be composed of fibrous tissue, which, gradually encroaching upon the nerve substance about them, cause wasting of the white substance of the fibres, though often the axis-cylinders can be observed penetrating the sclerotic patches. The bloodvessels in connection with these areas are thickened, as a rule, and there is nearly always a particular increase of connective tissue nearest to the vessels. While the system degenerations of the spinal cord are quite different from the disseminated variety of sclerosis, they are not infrequently found associated together in the same subject, and it is possible that they have, as their basis, a similar pathology. More than one observer has noted a resemblance be-

FIG. 227.



Multiple sclerosis. Section at mid-pontile region in same case as Fig. 226. Weigert stain.

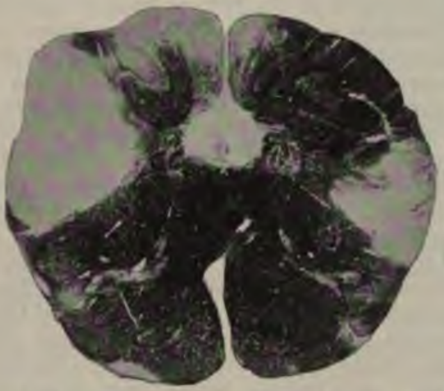
FIG. 228.



Multiple sclerosis. Section at level of sixth and seventh nerves. Weigert stain.

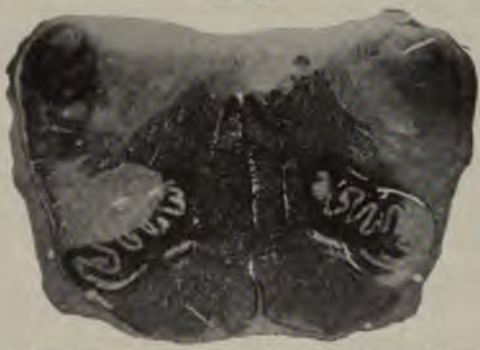
tween a diffuse cerebro-spinal syphilis and a disseminated sclerosis, as regards the character and distribution of the process, and this is also suggestive of an analogy in etiology.

FIG. 229.



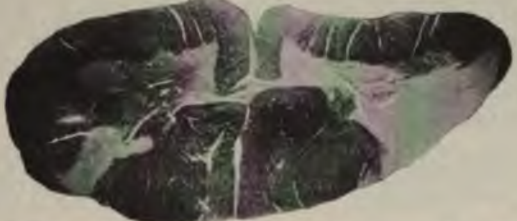
Multiple sclerosis. Section at upper level of motor decussation. Pals's method.

FIG. 230.



Multiple sclerosis. Section of medulla at level of tenth and twelfth nerves. Pals's method.

FIG. 231.



Multiple sclerosis. Section at level of seventh cervical segment. Weigert stain.

Course and Duration. As a rule, the disease is intermittently progressive, although sometimes the course of the disorder is quite gradual and uniform throughout. Remissions or arrest of progress may occur and last for long periods. The disease itself may go on for five to fifteen years, although five or six years is placed as the average duration. In cases in which the medulla is involved early, death may occur very soon. Usually patients die from weakness and exhaustion, due to the long continuance of a gradually increasing disease of the central nervous system, or from intercurrent diseases.

Diagnosis. The diagnosis in the typical cases is easy from the characteristic symptoms of tremor, weakness of the extremities, slow development, vertiginous attacks, nystagmus, spastic rigidity, peculiar speech, and exaggerated reflexes. There should be no difficulty in making a diagnosis between this disorder and paralysis agitans. In this latter disease the tremor occurs only during rest, is chiefly confined to the hands and fingers, and has an almost perfect rhythmic regularity of five per second. Occasionally there may be difficulty between cases of general paralysis of the insane and cases of disseminated sclerosis. In the former disease there may be an "intention tremor," very similar to that of the latter, and, while the differences in speech are marked to those familiar with the two types of speech developed, the practitioner who sees but few of these cases may at times be confused by their great resemblance. It must be remembered that the speech in sclerosis is of a stacatto-like character, the syllables being separated, while in general paralysis the speech is drawling and indistinct, very much like that of a drunken person. Any marked mental change would, of course, be in favor of general paralysis. The real difficulty in diagnosis is between the atypical forms of insular sclerosis and disorders like Friedreich's ataxia, locomotor ataxia, bulbar palsy, chronic meningitis, and hysteria. These fine distinctions can be made only after careful study of the whole condition and of the points mentioned under the headings referring to these other diseases.

Prognosis. The prognosis is, of course, grave as regards the curability of the disorder, but some hope of a possibility of improvement or of a remission may be held out. The improvement sometimes is quite remarkable, and, hence, the prognosis cannot be as absolutely bad as in the systemic sclerotic processes which go on in the spinal cord. Where the medulla or the nerves arising from it seem to be affected the prognosis is much more serious.

Treatment. There is very little, in a therapeutic way, which can be done for this disorder. We should employ nerve tonics, such as arsenic, quinine, and nitrate of silver; iodide of potassium and mercury should also be used with some hope of improving the condition. Arsenic has been recommended to be employed hypodermically. Solanine has also been recommended for diminishing the tremor, but probably the benefit derived would be more than counterbalanced by the bad effects of this powerful drug. Certain hygienic measures are of service, and hydrotherapy and electricity may at times be used. Doubtless as much good can be derived from hydrotherapy as from any ordinary general form of treatment. The life should be regular in every respect, and the patient should be kept as quiet as possible.

NOTE. The microphotographs illustrating Dr. Peterson's chapters were taken by Dr. Leaming from sections belonging to Drs. Van Gieson and Peterson.

CHAPTER XXIII.

PARETIC DEMENTIA.

By F. X. DERCUM, M.D.

The disease we are about to consider is variously known as paretic dementia, general paralysis of the insane, paralytic dementia, paresis, and as progressive general paralysis.

In a work upon diseases of the nervous system, an affection presenting such profound pathological changes as paretic dementia deserves detailed consideration. It differs widely from the ordinary psychoses in the degree in which such changes can be demonstrated. The pathological process is widespread, involving both brain and cord and even the peripheral nerves. It is accentuated in the brain, but in all cases distinct spinal lesions are present, and in not a few these antedate the cerebral disorder. Changes in the peripheral nerves suggesting those seen in neurotabes have also been found by a number of observers, *e. g.*, Déjerine, Bevan Lewis, and Campbell.

The affection is important because of its serious and progressive character. It is a disease in which a gradual change ensues in all of the mental and physical powers, a change which is degenerative in nature, and the recognition of which at an early period is of the utmost importance, not only to the patient, but to his friends and relatives.

Etiology. Certain factors outweigh all others in the etiology of paretic dementia. These are for the most part directly exciting causes. There are, however, secondary and extraneous elements which increase the liability or tendency to the disease, and it is therefore convenient to divide the various causes into predisposing and exciting.

PREDISPOSING CAUSES. Among the predisposing causes are heredity, time of life, sex, race, occupation, social position, and the unmarried state.

The family history of paretics is of some significance, because of its occasional neuropathic character. However, the importance of heredity has been somewhat exaggerated. Indeed, Mickle's[1] studies justify the conclusion that in the whole group of mental diseases taken together heredity is relatively more frequent than it is in paresis taken separately. Even in cases where heredity can be traced, it is not insanity or paresis that is transmitted, but rather a general tendency to organic and functional nervous disease, apoplexy, epilepsy, paralysis, etc. With this view the larger number of writers are in accord. Krafft-Ebing maintains that the predisposition to paretic dementia is usually acquired and not hereditary. One curious fact, however, should be stated, and that is that an hereditary element can be more frequently traced among female paretics than among males. As allied to heredity, Regis points out that consanguinity constitutes a predisposition to paresis, as it does to other nervous affections.

Paretic dementia is most frequently met with between the ages of thirty and fifty-five years. It is quite rare before twenty-five. It is very infrequent after sixty. Taking it all and all, the period between thirty-five and forty seems to be the one in which the greatest liability to the disease obtains. This period was formerly regarded as being from forty to fifty, but there can be no doubt that the larger number of cases reported within recent years occur before forty. In general terms the age at which paresis occurs, thirty to fifty-five, corresponds to the most active period of life, a period in which the greatest strains have to be borne and in which the greatest wear and tear takes place. Exceptionally, however, it occurs much earlier; thus cases of eighteen and sixteen years have been reported. Wigglesworth[1] has placed on record two cases aged fifteen, a boy and a girl. He also collected five additional cases in which the ages ranged from twelve to sixteen. All of these cases assumed a type of primary progressive dementia. In only one case (Clouston's) were delusions of grandeur present. The greater number were girls. Bristowe[2] reports the case of a boy, aged thirteen years, and Toulouse[3] cases of eleven and twelve years.[4]

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[1] Mickle on "General Paralysis of the Insane," 1886.

Experience has abundantly demonstrated that paretic dementia occurs much more frequently among men than among women; and this, when we consider the special exciting causes, is not surprising. The relative proportion of the sexes affected by the disease varies as to country, social position, and as to rural and city life. We should expect, therefore, the proportion to vary considerably. In Germany, for instance, the average is somewhere between eight to one and four to one. In France the proportion of women is distinctly higher, but still variable, while in our own country the proportion in the New England asylums is about four males to one female, in the New York asylums (exclusive of the city of New York), about nine to one, and in some of the Western asylums, such as those of Michigan, thirteen to one (Stearns).[5]

Paresis occurs very infreqently among women of the upper class, and, in keeping with this fact, we find almost without exception that in public asylums the proportion of female paretics to male paretics is much greater than in private asylums.

In rural populations, again, we not only find the total number of cases of paresis less, but also a comparatively slight disproportion between the sexes. For rural populations in France, Regis states that it is only one and a half times more common in men than in women. In our own country, according to Stearns, taking the States of Maine and Vermont, which contain no large towns, we find the proportion of female paretics one to three. In cities, paresis is not only relatively frequent, but the disproportion between the sexes is much increased. Especially is this true of the higher classes of society. According to Regis, for instance, it is thirteen times more frequent among men of the upper class than among women of the same class. Among the laboring population of large cities, however, this disproportion again falls, being only three times more common in men than in women. These facts are exactly in keeping with what we know of the principal exciting causes of the disease. Men, on the average, undergo far greater mental strains in the early or middle period of life than women. As we would naturally expect, where this strain is most disproportionate, *e. g.*, in the upper social class, men suffer much more frequently, while in the lower strata of society, where the strains of life are more equalized, the proportion

[1] Wigglesworth: British Med. Journ., 1893, i. 635.

[2] Bristowe: British Med. Journ., 1874. i. 1125.

[3] Toulouse: Gaz. des Hôpitaux, 1893, lxvi. 909.

[4] Whenever such exceedingly precocious paresis is met with we should always be suspicious of some such potent factor as hereditary syphilis. In both of Wigglesworth's cases, and also in that of Bristowe, there was a history of injury to the head.

[5] Stearns: Mental Diseases, Philadelphia, 1893.

of women affected rises. We have also reason to think that it is more common in those women who have entered into competition with the male sex in occupations ordinarily carried on by the latter. This supposition is borne out by the fact that paresis is distinctly on the increase among women. The menopause cannot be regarded as a predisposing factor of great importance, though it must be admitted that cases not infrequently occur at that time. Out of forty-one female cases recorded by Westphal,[1] eight are ascribed to the climacteric, a rather unusual number. Gestation exercises little or no influence. Krafft-Ebing, however, states that paresis sometimes follows repeated and severe labors.

Paresis is far more common in Western Europe and America than elsewhere, although this statement is open to a number of qualifications. For instance, while paresis is very frequent in England, it is so rare in Ireland as to be almost unknown. While rather frequent in Germany, it is almost unknown in Scandinavia, inhabited by a kindred race. It would appear that, under certain circumstances, when the Anglo-Saxon, or, in a larger sense, the Teutonic race, are stimulated to unusual exertion, paresis occurs among them. The studies which have been made in our own country in reference to this point, by Spitzka and by Stearns, are in conformity with this view. Spitzka, for instance, found that in a pauper insane asylum in New York City the Anglo-Saxons headed the list in numbers. Next followed in order Celts, Germans, and negroes. These facts are largely in keeping with the characteristics of the races, those of Anglo-Saxon blood being, for the most part, engaged in feverish business activities and burdened with great and exhausting responsibilities. The phlegmatic character of the German and the indifference of the Celt explain their secondary positions. It is, however, somewhat difficult to account for the relative infrequency of paresis among Hebrews, as they are, for the most part, a race exceedingly active both in business and professional life. Regarding negroes, Berkley[2] found that of seventy-four patients, five, or 6.7 per cent., suffered from paresis, a much larger proportion than he found among the white patients, three hundred and thirty of whom presented only three paretics, or less than one per cent.

EXCITING CAUSES. Among the exciting causes of paretic dementia, one stands out prominently above all others. It consists of those great strains of the nervous system implied by overwork, overexertion, intense and prolonged worry, terrible disappointments, serious and apparently hopeless reverses of fortune—in short, all causes that tax the intellectual and emotional faculties to the uttermost. In this list we should also include such profoundly disturbing causes as fright and excessive nervous shock. Often a number of causes of overstrain are present. Not infrequently do we find that a man engaged in an active and exhausting business which taxes his energies excessively does not develop paresis unless to this already great strain there be added some profound emotional disturbance, as a sudden and depressing grief or domestic trouble. In a small number of cases physical overexertion is to be regarded as a cause. Sometimes there is a combination of both psychical and physical overstrain. In keeping with the above facts we find that the number of cases of paresis occurring among business men and among those who live in the intense excitment of political strife is relatively high; whilst those who lead the more quiet lives of farmers, stock-raisers, and the like furnish a comparatively small number. These facts are also in keeping with the many predisposing causes which we have already considered, such as the frequency of paresis in the most active periods of life, namely, from thirty-five to fifty, and also its greater frequency among the male sex, especially in the middle and upper classes.

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[1] Westphal: Charité Annalen, 1893, x. 719.

[2] Berkley: Johns Hopkins Hosp. Bulletin, No. 34, October, 1893.

The majority of writers have laid great stress upon alcoholism as a factor. Proof is, however, lacking that alcohol of itself brings about paretic dementia. On the contrary, the symptoms of alcoholic dementia differ in important points from those of paresis. Evidently the abuse of alcohol can only be regarded as a factor secondary in importance. However, that it acts as a powerful adjuvant in the causation of paresis, when the factors of intellectual and emotional overstrain exist, there can be no doubt. Certainly no danger is greater than a resort to stimulants by persons passing through such strains. A mere physiological hyperæmia of the brain under the use of alcohol may become pathological and determine the onset of paresis. Considered from this standpoint, the dangerous nature of alcohol under such circumstances can readily be understood. On the other hand, we are liable to err regarding the extent to which paresis can be attributed to alcohol. The vast majority of paretics, by very reason of their disease, commit excesses of all kinds in the prodromal and early stages, and under these circumstances the abuse of alcohol is not so much a cause as an effect and symptom.

The same statements are also true of the sexual excess which patients present and which various writers enumerate among the causes of paresis. We find, from Mickle's[1] studies of the reports of the commissioners in lunacy (England), that out of four thousand two hundred and eighty-four cases, the disease was ascribed to sexual excess in one hundred and nine, *i. e.*, only 2.5 per cent. The importance of sexual excess as a factor is open to still further question. In the first place, the age at which paretic dementia is prone to occur is not the age of sexual excess. The period of sexual excess ranges from early youth up to thirty years of age. Rarely is it found at other times. Paresis occurs much later—at a time when both men and women are sobered by the severe realities of life. Doubtless in the majority of cases sexual excess is to be looked upon as an effect of the disease and not as a cause.

A very important factor in the causation of paretic dementia is trauma of the head. Among the four thousand two hundred and eighty-four cases collected by Mickle, as many as two hundred and eighty were ascribed to accident or injury. Concussion of the brain seems to lessen its power of resistance, perhaps affects directly the vasomotor control of its larger vessels, and thus predisposes it more readily to attacks of congestion. As a rule, the development of paresis after a severe blow upon the head is gradual, many months or even years intervening, although the interval is in rare cases much shorter. Sometimes the interval is so long that the trauma is often looked upon as a predisposing rather than as a directly exciting cause.

Sunstroke, or prolonged exposure to great furnace heat, is also an important factor. In ninety-seven of the cases collected by Mickle, sunstroke was assigned as the cause. It is very probable that sunstroke acts very much as does concussion of the brain, namely, by predisposing the organ to hyperæmia and by lessening its power of resistance.

Syphilis as a cause of paresis has been much discussed. Undoubtedly it is a powerful factor. According to Graf, as many as 40 per cent. of paretics are victims of syphilis, and Mendel gives the percentage as 75. According to Rieger, quoted by Krafft-Ebing, the subjects of syphilis are from sixteen to seventeen times more liable to paretic dementia than others not so affected. According Regis,[2] syphilis exists in seventy to ninety cases in every one hundred in general paralysis. Hougberg[1] found syphilis in 75.7 per cent; Bannister[2] in 89 per cent. Whether syphilis is an active factor in the production of paresis or is only a predisposing cause has been much disputed. Several possibilities suggest themselves. It is possible, first, that paresis is the outcome of the late action of the toxine of syphilis, a parallel instance of such supposed action being furnished by locomotor ataxia. This parallelism is enhanced by the fact that an ataxia comparable to ordinary locomotor ataxia now and then precedes the development of paresis (paretic dementia of the ascending type). It is possible, secondly, that a nervous system profoundly exhausted by the infection of syphilis breaks down more readily under the intellectual and emotional strains which play so important a part as exciting causes of paresis. It is further significant that, for the most part, paresis in syphilitic subjects is a late development. In Hougberg's[3] cases, eighty-one in number, the onset occurred in from five to nineteen years after infection.

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[1] Loc. cit.

[2] Manual of Mental Medicine, 1894, transl. by Bannister.

When in a syphilitic subject true gummatous infiltration of the cortex occurs, *i. e.*, of the membranes and of the vessels, the case is not one of paresis, but of syphilitic dementia. (See Chapter XXIV.) A possible and a serious error in regard to syphilis should here be pointed out, and that is, that, due to the sexual excesses and concomitant exposure to specific infection in the earlier stages, the patient may acquire syphilis *subsequently* to the actual onset of the disease. In such a case, of course, syphilis could not be regarded as the causal factor.[4]

Among other causes to which paresis is rarely attributed are lead-poisoning and the excessive use of tobacco. Inasmuch, however, as the symptoms of lead encephalopathy differ from those of classical paretic dementia, it is probable that the place which it occupies in etiology is that of a predisposing rather than a directly exciting cause. The same remarks apply, in all probability, to tobacco, attention to which as a cause of paresis was especially called by Guislain.

Paresis is rarely if ever to be attributed to acute illness, such as fevers, pneumonia, and other severe diseases. The latter are much more prone to cause other forms of mental derangement. Occasionally paretic dementia is met with subsequent to various insanities. It cannot, however, be regarded as standing in any relation to these insanities. In a case presenting such a history, the paresis must be regarded as an affection *de novo*. The same statement is true of the relation of other nervous disorders to this disease. Paresis is never met with, for instance, as the outcome of neurasthenia or hysteria.

Symptoms and Course. Both the symptoms and the course of the disease are extremely variable. Throughout the clinical picture, however, there is discernible a progressive mental and physical enfeeblement. As might be expected in a disease the changes of which are widespread and diffuse, there are present various psychic anomalies, many of which are common to other psychoses, and also various physical anomalies, many of which are common to other degenerative nervous diseases. Further, these symptoms, both mental and physical, are as a rule, at first so slight as frequently to escape recognition. Gradually, however, they become more and more pronounced until with time they are evident even to the lay observer. When once established the degenerative changes, save with one or two possible temporary interruptions, steadily deepen until they finally become incompatible with life.

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[1] Hougberg: Neurolog. Centralbl., 1894, p. 279.

[2] Bannister: American Journal of Insanity, 1893–4, p. 477.

[3] Loc. cit.

[4] The relation which the two sexes bear to paretic dementia as caused by syphilis appears to be different. Thus, in 148 cases of paresis in women recorded by Westphal (loc. cit.) syphilitic infection was probable in 48, a much smaller proportion than is usually found among men. In this connection a curious personal observation made by Morel-Lavallée is interesting. Of five men infected from the same source three developed general paralysis; two died of cerebral syphilis; while the woman remained perfectly well, married, and had two sound children.

Because of the very gradual and progressive character of the disease, it is separable into a series of stages or periods. We recognize at once the fact that there is in every case an initial period which distinctly antedates the fully developed disease, and to this the name of the "prodromal stage" has been given.

The disease begins in a manner so extremely gradual that it is rarely if ever possible to fix more than approximately the time of its inception, and similarly the various stages pass for the most part insensibly one into the other. It is important at the outset to recognize that this separation into stages is largely artificial, and, keeping in mind the further fact, already alluded to, that both the symptoms and the course of the disease are extremely variable, it is not surprising to learn that medical writers frequently differ as to the number and arrangement of these stages. However, this is true rather of the fully developed disease than of the prodromal period. The existence of the latter is, as a matter of course, recognized by all writers. To this prodromal or initial period, as it is better termed, we will now give our attention.

INITIAL PERIOD. In the majority of cases we are unfortunately dependent upon relatives and friends for an account of the earliest symptoms. However, the facts that are obtained, though often fragmentary, are of the greatest value. They are for the most part sufficient to indicate that various changes, both mental and physical, have taken place in the individual. In a general way, for instance, it has been noted that the patient has not been well for some time past. Often the friends maintain that the patient's appearance and manner have changed, that he no longer attends to his business as carefully, or no longer does his work as well as formerly. Often he looks badly. His face may be unusually pale, or, on the other hand, may exhibit an unaccustomed flush. He may look tired, often sleepy. Frequently his face seems to lack expression. Physically he may seem weak. His attitude, his movements, his walk may suggest a general loss of tone and vigor. Not infrequently he complains of a sense of fulness, pressure, or constriction about the head. Sometimes there are rheumatoid pains referred to the legs, to the arms, or to the back of the neck or trunk. Occasionally these pains are neuralgic in character, and indeed at times lightning-like pains resembling those found in the early stages of locomotor ataxia are present. Often the patient complains of headaches. Sometimes the latter occur in paroxysms of great severity. The pain, instead of being diffuse, may be referred to some special region, as the occiput or the brow. Occasionally the headache simulates an attack of migraine, the pain being felt with great intensity not only in the supra-orbital and adjacent regions, but especially in the eyeball of the affected side. Sometimes cases are met with in which this pain is so severe as to suggest, for the time being, an acute attack of glaucoma. Frequently ringing in the ears, sparks before the eyes, and muscæ volitantes are complained of. Attacks of giddiness and vertigo may also occur, and sometimes the patient complains of a dazed or stunned feeling in the head.

Mentally the patient presents symptoms which at first suggest, and indeed are sometimes remarkably like, those of neurasthenia. He cannot do his work without making an unusual effort. Work, especially mental work, soon becomes distasteful and even painful. Inability to fix the attention for any length of time upon an ordinary affair of business or other everyday matter, also, becomes evident. In conversation it frequently becomes necessary to repeat to him the simplest statement a number of times. There is distinct feebleness both in the ability to apprehend and in the ability to remember. Unusual forgetfulness is especially observed about the smaller details of life. A previously precise and methodical man forgets to wind his watch, to mail his letters, or, in dressing, forgets some article of attire, such as a collar or a cravat. It is further significant that recent events are soon forgotten even when the latter are of a character as ordinarily to impress themselves upon the patient, *e. g.*, the death of a friend or a business transaction conducted only a few days before. It is also in keeping with these facts that the patient often repeats the same statement or tells the same story a number of times in succession, being himself unconscious of the repetition. Such facts as these are of course of significance only if they be in striking contrast with the former peculiarities of the individual.

In numerous ways the patient may reveal other changes. For instance, his sensibilities, both intellectual and emotional, become blunted. Things which formerly interested him greatly, appeal to him less forcibly or perhaps not at all. An intellectual man devoted to some ethical or æsthetic pursuit loses all interest in the latter and may become entirely indifferent to it. Similarly his sense of obligation to his family, although formerly regarded in the most exalted light, is much lessened. The news of the sudden illness of a beloved child is heard with indifference; the caresses of wife and children no longer meet with their former response. His daily vocation, no matter how pressing, appeals to him in a much lessened degree, and at the same time he betrays numerous errors of judgment about the commonest affairs of life. His business capacity becomes much impaired. Errors and confusion begin to appear in his accounts. Engagements are neglected and business transactions badly conducted.

Hand-in-hand with the general blunting of the emotions and of the intellectual powers, the grosser animal qualities rise to the surface. A man formerly temperate in both eating and drinking now eats and drinks excessively, showing evident satisfaction, in this, to him, unwonted and coarse gratification. Soon he becomes indifferent to his personal attire, often appearing in the presence of his family or strangers with his clothing improperly adjusted, and in other ways manifests a distinct loss of the sense of the proprieties. Often, indeed, he makes careless and indecent exposures of his person. His speech, too, loses its former nicety and refinement and often becomes coarse and vulgar. Often he is obscene, and sometimes he attempts liberties with the female servants of his household or with other persons of the opposite sex in whose company he may happen to be.

Gradually the signs of the threatening dementia become more and more pronounced. The occasional lapses of memory soon increase to serious gaps, and the attendant dazed and confused condition of mind, together with the feebleness of the will, render the patient less capable than ever of conducting his affairs. It is not surprising that every now and then disaster and bankruptcy should under these circumstances occur, or that errors of accounts and neglect of obligations should make his affairs the subject of litigation.

There is also a change in character and disposition, which may be so marked as to be almost like a complete change in personality. Sometimes he is apathetic, abstracted, and dull. More frequently he is restless, irritable, and excitable. He becomes angry at slight causes, but falls utterly to react in a normal manner to events of real consequence. Thus Spitzka cites the case of a patient who threw a knife at his servant because she took his plate away before he had, as he alleged, finished dining, and who heard unmoved a few hours later of the collapse of a large business undertaking which involved a loss to him of over a hundred thousand dollars.

In addition, the patient is excessively egotistical, and this feeling is often accompanied by a fictitious sense of physical well-being and power. He talks extravagantly about himself and his affairs. He plans great enterprises, undertakes great projects, and generally succeeds in making absurd and useless expenditures. Thus a physician bought a large number of bird cages, a laboring man ordered an enormous quantity of flowers, while a third patient, a woman, purchased a great number of miscellaneous and incongruous articles without assignable object.

Together with this intellectual deterioration there is also a distinct impairment of the moral nature. The patient may tell silly and absurd lies without object or purpose. He is prone to commit theft, often stealing things for which he can have no possible use; indeed, theft is every now and then the first overt act by which general attention is drawn to the patient's condition. At times this thieving and lying has its origin in loss of memory and absentmindedness. At other times it is plainly the outcome of a blunting or perversion of the moral faculty, as, for example, when theft is perpetrated by means of a forgery.

Various sleep disturbances, as a rule, also make their appearance. The patient may suffer from more or less marked insomnia, and sleep, when it does occur, may be disturbed by disagreeable dreams. On the other hand, during the day more or less somnolence may be present. The patient is apt to fall asleep at unusual times and places; for example, at his office and at hours usually devoted to active business. The somnolence is especially noted after eating, when it may be almost impossible for the patient to keep awake.

The tendency to excess in drinking, already noted, gradually becomes more marked. Sexual excess also becomes more pronounced. The previous indifference to exposure of the person now gives place to active erotic excitement, the patient making no effort to conceal his condition, and often committing almost incredible excesses, as, for instance, in a case cited by Mickle, in which the patient, in the space of one week, "engaged, it is said, in about fifty acts of marital coitus with full seminal ejaculation on each occasion." Sometimes the erotism is so great as to lead to the commission of violent acts, as in the case of a patient who attempted to rape his own daughter. In some cases, however, it should be stated, early loss of both sexual desire and power occurs.

In his movements the patient reveals that he is awkward. His gait is often sluggish and unsteady. Mechanics, formerly skilful in certain movements, lose their dexterity. There is generally, also, more or less marked awkwardness and hesitation in speech; sometimes there are attacks of transient aphasia. More or less myosis, inequality of the pupils or other motor symptoms referable to the eyes, fibrillary tremors of the tongue, irregularly recurring twitchings of the facial muscles, or fine tremors of the hands, may also be present. Tremor is also apt to be revealed by the handwriting. The handwriting, in addition to being somewhat tremulous and jerky, may also reveal occasional errors, such as the elision of letters, syllables, or words, or unwonted errors in grammar. These peculiarities are, however, much more noticeable in the fully established disease. Indeed, it not infrequently happens that the motor symptoms as a whole are very insignificant in the prodromal period.

Among the various physical symptoms, the patient may also present anomalies of the knee-jerks. These are usually exaggerated, though sometimes much diminished and even lost. When this is the case spinal symptoms, such as are found in locomotor ataxia, are generally present, and have for some time preceded the cerebral symptoms. In other words, diminution or loss of knee-jerks is apt to be present in the so-called ascending form of the disease. When, on the other hand, the knee-jerks are exaggerated, spastic symptoms in the legs may be present. It is important, however, to bear in mind that in many cases of paresis, in the initial period, the knee-jerks are not perceptibly changed. The condition of the cutaneous reflexes is also very variable. In about 50 per cent. of the cases these reflexes appear to be normal. However, they are often lost, and especially is this true of the cremasteric reflex. With the latter there is sometimes found associated a beginning atrophy of the testicle. In a very small number of cases the cutaneous reflexes are found exaggerated.

At any time during this initial period the patient may suffer from attacks closely resembling apoplexy or ordinary epilepsy. They are not, however, by any means always present. When such seizures do occur in this period they are more apt to be apoplectiform than epileptiform. After a few premonitory symptoms, such as increased restlessness, excitement, and sleeplessness, and perhaps flushings of the head, the patient suffers from a loss of consciousness, the attack being accompanied by a sudden though temporary loss of power over one-half of the body. Sometimes the resulting hemiplegia is somewhat persistent. Occasionally marked weakness of both sides of the body results, though usually this symptom is more pronounced on one side than upon the other. Less frequently the seizures, instead of being apoplectiform, are like those of epilepsy, the patient having a convulsive attack, generally accompanied by a more or less marked loss of consciousness. To a detailed consideration of these convulsive seizures we will presently return. Suffice it here to say that after such an attack the various symptoms of the disease become accentuated. Occasionally the initial period terminates abruptly in an apoplectiform or epileptiform seizure, the patient passing at once into the fully developed stage of the disease. We must remember, however, that sudden seizures are by no means present in all cases, and that, under these circumstances, the transition from the initial period to the developed disease is so gradual that we are unable to determine when they merge one into the other.

The duration of the initial period, as a rule, is from one to three years, though it is sometimes much longer. Cases in which the initial period is very short—*e. g.*, several weeks—are exceedingly rare and are really open to question. The difficulty of fixing the duration of this period is increased by the fact that the progress of the disease is not always steady and uninterrupted. Temporary recession of both physical and mental symptoms now and then occurs, and under favorable conditions such a recession may extend not only over several days and weeks, but over much longer time. To this fact is doubtless due the extraordinarily long duration of the initial period observed in some cases. In addition, the course of the initial period is often irregular; while certain symptoms, such as tremor or ataxia, may recede, others, such as loss of memory and blunting of the moral faculties, may steadily advance. At other times the reverse obtains, mental symptoms receding and physical symptoms persisting.

The reader must not conclude from the above description of the initial period that all of the symptoms detailed occur in every case or that they even occur with an equal degree of severity. Some of the symptoms may be suppressed, while others may be unusually prominent. However, no matter how or when occurring they are always indicative of the essential feature of the disease; namely, persistent and unmistakable loss of psychic and physical powers.

It is important to state that the symptoms often vary considerably during the day. The patient who in the afternoon or evening presents more or less marked anomalies of judgment, of memory, of will power, or of movement, *e. g.*, ataxia and tremor, or, whose face has been flushed, who has been dull, heavy and sleepy, may the next morning be comparatively himself. Often during these early morning remissions, as they may be called, the patient vaguely and sometimes vividly realizes his unfortunate condition; but as the day advances the flush, the somnolence, the thick and hesitating speech, the tremor of the lips, the forgetfulness, again reassert themselves.

Frequently, as the initial period progresses, the patient becomes hypochondriacal and melancholic. He becomes filled with vague forebodings and ill-defined fears. This mental state may come on spontaneously, but sometimes it is the direct result of the partial recognition by the patient of his condition. Mental depression is of such frequent occurrence that Mendel has erected it into a separate stage of the disease.[1] He regards it as a second period, or as the first stage of the established affection.

The fact upon which we have already dwelt, namely, that the symptoms are extremely variable, is noticeable not only in the initial period, but especially in the fully developed disease. This has led to differences not so much in the description of the symptoms as in the division of the disease into stages. We have just alluded to the fact that in some cases the hypochondriacal symptoms in the latter part of the initial period are so marked as to have led Mendel to erect out of these symptoms the first stage of the fully developed disease. For practical purposes, however, since this condition of hypochondriasis is sometimes absent, it is best to consider it as a part of the initial period. The arrangement adopted by Krafft-Ebing appears to be the most natural, inasmuch as it is in accord with our general experience.

The initial period eventuates in three different ways: *first*, and most commonly, in a condition in which the symptoms of dementia already noted in the initial period have become more pronounced, but to which there is now added an expansive or a maniacal mental state; *secondly*, and less frequently, in a condition in which, as before, the symptoms of dementia are more pronounced, but to which there is now added a depressive, hypochondriacal or melancholic mental state; and, *thirdly*, and least frequent of all, a condition in which there is simply a steady increase in the symptoms of the dementia without the addition of any expansive or depressive mental phenomena, the disease resembling in its course a simple progressive dementia.

The period of the fully developed disease may be divided into a number of stages. Inasmuch as the disease is slowly, and for the most part, steadily progressive, the transition of these various so-called stages into each other is usually very gradual, and the separation of the disease into stages is really artificial. However, for practical purposes it is convenient. Roughly speaking, the period of the established disease in the expansive and depressive forms consists of *first*, a stage in which the expansive or depressive mental state is at its fullest development; *secondly*, a stage in which the dementia has increased to such an extent that well-marked delusions no longer exist, and in which at most mere traces of the former expansive or depressive mental state are found; and, *thirdly*, of a stage in which the dementia has become so pronounced that psychic life is almost extinct. It will serve our purpose best to consider the special symptoms of the various forms of paretic dementia separately. We will, therefore, turn our attention first to the expansive form.

FIRST STAGE OF THE ESTABLISHED DISEASE.

Symptoms of the Expansive Form. As just stated, this stage most frequently begins gradually, so that it is impossible to say when the initial period has ended and the first stage of the disease begun. We must remember, however, that at times it is ushered in suddenly by an apoplectiform attack.

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[1] Die progressive Paralyse der Irren, Berlin, 1880.

We notice at once that many of the symptoms presented in the initial period are now more marked than before. Mental failure, for instance, is more evident; memory, especially for recent events, is feebler than before, and this is likewise true of attention, judgment, and will power. Especially noticeable, also, is the increase in the loss of the power on the part of the patient of appreciating the actual circumstances in which he is placed or to recognize properly his own relations to his business or to the people about him. These evidences of progressive dementia are not, however, the most striking features of this stage. New symptoms are now added, and they are so common as to be characteristic. The tendency to boastfulness and occasional exaggeration, noted, perhaps, in the initial period, now becomes very marked.

Delusions most extravagant in character make their appearance. The patient believes himself to be a person of great importance, to be possessed of extraordinary physical strength, or, what is most common, he believes himself to be the possessor of enormous wealth. If the patient be a woman, she is endowed with great personal beauty, is blessed with an extraordinary number of children, or is favored by more than the usual number of lovers and husbands. A characteristic of these *delusions of grandeur*, as they are termed, is that they are imperfectly systematized, that they are often feebly held, and that they are always grossly improbable. They are, further, extremely variable. The patient who tells us to-day that his wealth was the result of some great invention, to-morrow tells us that he received it as a bequest, or he makes no effort to account for it at all. The patient who to-day asserts that he is worth two hundred thousand dollars, may to-morrow assert that he is worth several billions, or the next day but a fraction of this amount. In like manner does the substance of the delusions change. To-day he is a king or emperor, to-morrow a great physician, a great judge, or the richest man in the world. Some of the delusions, however, may be more persistent than others and slightly more systematized. This is particularly true of the delusions of wealth. Many of these expansive ideas have no connection at all with each other, and they all betray great inherent weakness. The following case will serve as an illustration:

R. J. B., admitted to the Philadelphia Hospital February 10, 1895; white, aged fifty-one years, salesman.

Family history negative as to mental and nervous diseases.

Personal history: Patient had a severe blow on the back of the head in 1876, which left him with a headache for several years. Has also been a steady drinker, frequently committing excesses. In 1887, after a drinking bout, his friends say that he "acted crazy" for a week or ten days.

Has for some time past been indifferent to the wants of his family. Within five months developed wild schemes and delusions as to money-getting, wealth, etc. Pawned anything he could find at home; talked extravagantly, said he stopped runaway horses, etc. Gave checks; signed his mother's name to checks on the Centennial Bank, where some years ago he had an account. Claimed to have inherited vast estates. Abandoned his religious belief and joined the new order of the "Third Christians."

At the time of admission the patient was asked the following question: What is your business? He replied: "I am a manufacturer of the first character of ladies' shoes, having been in the business for twenty-five years. Our firm is a queer combination, me a Friend, two Jews, and a Dutch Roman Catholic, so we never discuss religion. Our firm does a business of over $600,000 a year, the profits being 28 to 30 per cent. We make nothing but the very best class of shoes, silk linings, and many have either gold or silver buttons." Says that he has not accumulated much from business, as he has had to have his hand in his pocket continually to keep his brother's head above water. "I have, however, been fortunate in having been 'left' $6,000,000 in the following way: In January, 1877, while strolling through the Park, I caught the runaway horses of a gentleman, and, no doubt, saved his life. He took my name and address, and said sometime I would hear from him. Four months ago he died, leaving me $3,000,000, and there was a codicil which said that if not satisfied I should ask for more, and so I asked for $3,000,000 more. I also own two charitable hospitals—one for children and one for adults—which I have endowed for $1,000,000 each;" and thus, in a quiet, self-satisfied manner, he speaks of his great wealth and the number of relatives he intends to assist financially. He states further that he "was Governor of this State; in 1873, Mayor; at the present time he is United States Senator, and has been recently nominated for Select Council." Says that he has "always had the biggest majority of any one in this city," and has "a large following," and thinks he ought to take the position of Councilman to halt two men who are robbing the city. Prefers not to give the names of the two men at present. He "is defending one in a suit, and while defending him as a lawyer he will do as little to help him as possible." In another minute he tells us that he graduated in medicine at Harvard, in Berlin, and at Paris; that he has practised medicine for eighteen years. Since sixteen years of age "has been an elegant singer and player."

In appearance he is sleepy looking, nervous in action, and yet calm in mind. Hardly recognizes his surroundings, calls the hospital the "Hughes Academy," but really does not know. His whole demeanor during his mental examination was that of a man who believed exactly what he said.

February 12, 1895. His "delusions of grandeur" are, if possible, growing greater, but he is gradually adapting himself to the discipline of the hospital; sleeping and eating well.

February 15th. At 5.45 last evening was determined to leave the hospital, caught up a chair and struck the window with it, breaking a pane of glass. When the attendant endeavored to quiet him he attacked the latter with the chair. Was, however, finally quieted.

March 5. After the outbreak above mentioned the patient was transferred to Ward 5 (acute ward), and placed in bed. He has been perfectly tractable since, and has in every way submitted to the discipline of the ward. After one week in bed he was again permitted to be up and took his place in the ward day-room. His delusions have grown in vastness, so that now he estimates his wealth at $150,000,000, $50,000,000 of which he made "in as many minutes." He is still not cognizant of his surroundings. Shows no discontent with his detention.

Somatic condition: Face, expressionless, pale. Eyes, partial ptosis, pupils small, unequal—the right larger; reaction to light imperfect; accommodation normal. Tongue, slightly tremulous. Knee-jerk, slightly exaggerated. No ankle-clonus. Cremasteric reflex feeble. Heart, vessels, lungs, and abdominal viscera negative.

In keeping with the expansive mental condition, the bearing and expression of the patient indicate satisfaction and contentment. Often great pleasure and happiness are depicted on his countenance. The expression, however, may be modified, as we shall presently see, by such factors as the partial effacement of the normal wrinkles and folds of the skin, slight facial inequality, tremor, and twitching.

It not infrequently occurs that in this form marked delusions are absent, the expansive state being merely indicated by the patient's manner and expression, and by his oft-repeated statement that he is feeling very well and that he was never better in his life. In other words, an exaggerated sense of physical well being is at times the only indication of the expansive condition.

Occasionally during the expansive stage, the exaltation increases to such an extent that the patient may for short periods be in a condition closely simulating ordinary mania. There is marked excitement and restlessness. Sometimes the patient is destructive, and his language foul and conduct indecent. Such a maniacal condition is usually brief, though sometimes it is prolonged. It is important also to state that occasionally a sudden maniacal outbreak seems to replace the apoplectiform or epileptiform seizures seen in other cases. These maniacal outbreaks are in such instances followed, like the seizures, by a marked increase of the dementia. It is further a noteworthy fact that cases of paretic dementia presenting marked excitement or maniacal attacks run a comparatively rapid course.

Sometimes the mania is replaced by an intense motor excitement, the so-called "silent excitement" (Mickle), in which the patient is continually moving his limbs, pulling restlessly at his bed-clothing, or pushing at the objects about him, and at the same time resisting interference from his attendant, even when this is necessary for his immediate wants. Hours may be spent in this restless moving, pulling, pushing, shoving to and fro.

Little by little, as the case progresses and the dementia becomes more marked, the delusions of grandeur become gradually less clear and less coherent until finally they are represented merely by fragmentary ideas. The patient then passes into the next stage of the disease, that of simple dementia.

Symptoms of the Depressive Form. Instead of the expansive mental state supervening upon the initial period, the patient passes in this form into a state of marked mental depression. Delusions of both physical and spiritual ills make their appearance. Sometimes hypochondriasis, at other times melancholia is most pronounced. Occasionally hypochondriacal ideas are the direct outcome of the mental depression observed at the close of the initial period. The patient then has delusions of some hopeless physical disease; he may believe, for instance, that his blood has become congealed; that his viscera have undergone decay; that his bones are broken in many places; that he has lost his arms or lost his legs; that he cannot eat, because he has no longer a mouth, or that his bowels have become hopelessly obstructed. Sometimes these ideas are replaced by delusions of "belittlement" (Mickle), in which the patient believes himself to have wasted away, grown smaller, become dwarfed, etc. In other cases, again, the patient suffers from ideas of impending evil, delusions of persecution, of poisoning, or that he can no longer be "saved" (that is, in a spiritual sense). Just as the delusions of grandeur are extremely shifting and poorly systematized, so it is with these ideas of spiritual and physical ills. Further, in the larger number of cases of the depressive form, hypochondriacal and melancholic ideas are intermingled, though it is usually the case that one or the other group of delusions is uppermost. As in true melancholia, suicidal attempts may be made, but these for obvious reasons are infrequent and rarely successful.

It is important to add that hallucinations are more marked in the depressive than in the expansive form. The patient hears voices, and may base his delusions of persecution upon this symptom. Hallucinations of taste and smell are also present, and may in turn serve as the basis of delusions of poisoning. Finally, the depressive form of paretic dementia appears to be somewhat more frequent among women than among men.

In its course this form is progressive, like the expansive form, though, as a rule, much more slowly. Like the expansive form, again, it may be interrupted now and then by epileptiform or apoplectiform seizures, or it may pass gradually into the second stage of the established disease, namely, that in which the depressive delusions are forgotten, and nothing but simple dementia remains.

Rarely, the expansive and the depressive mental states alternate with each other, either by sudden transitions, or separated by an interval in which no delusions are present. This fact has given rise to the expression of "the circular form" of paresis. Several such alternations may take place in the course of the disease. The condition is, however, easily differentiated from ordinary "circular insanity" by the accompanying dementia, which is always more or less marked and progressive. The mere occurrence of a form of paresis in which expansive and depressive mental states alternate is evidence that the distinction between the various forms we have described cannot be considered as absolute. Indeed, to go further, it is by no means an uncommon experience to find, in the expansive form, traces of depressive delusions or even short periods in which mental depression, distress or fear is uppermost.

As in the maniacal or expansive form, attacks of intense motor excitement, the "silent excitement" already mentioned, may occur. Again, it sometimes happens that just as in the expansive form there is present merely a feeling of heightened physical well being, without well-marked delusions, so in the depressive form there may be present merely a vague feeling of depression and of illness. In rare cases again, the symptoms resemble those of a stuporous melancholia or a simple stuporous insanity. According to Mickle, the physical signs are in such instances at first absent or slight, though later, and generally after a remission, the physical signs may slowly increase,

Symptoms of the Simple or Uncomplicated Form. Here both the expansive and the depressive mental states are wanting, and the disease resembles in its course a simple progressive dementia. Gradual loss of memory and of the other mental faculties becomes more and more marked. It can readily be understood that there is a time when the mental condition of all cases of paresis is the same. For instance, a case of the expansive form which has progressed so far that well-marked delusions have been effaced is in a condition indistinguishable from that of the simple or uncomplicated form. The latter occurs beyond a doubt more frequently among women. Indeed, paresis is, almost as a rule, milder in women than in men, and the dementia is apt to be of a quiet type.[1] The simple form, further, differs from the other forms by frequently appearing at a relatively early age. It is often excessively gradual in its onset, and its course so smooth that for a long time it may remain unrecognized.

General Symptoms Common to or More or Less Liable to be Present in All Forms of the Established Disease. When divesting the expansive and depressive forms of their special features, an underlying basis of clinical facts is revealed which is practically common to all forms. We have already noted how with the appearance of the fully developed disease mental failure becomes more pronounced. Memory, judgment, and will-power are feebler than before. All of the psychic anomalies observed in the prodromal period become exaggerated. So, too, the various physical symptoms, traces of which were seen in the prodromal period, become more pronounced. If we test the strength of the muscles we find that muscular power is now decidedly diminished. It is true that sometimes decided loss of strength does not appear unless the test by the dynamometer be repeated at short intervals; rapidly induced fatigue is then noted. Tremor may also be present, especially when the patient makes a voluntary effort. Generally the movements are jerky and spasmodic. Motor disturbances are, as a rule, most marked in the face. Irregularly recurring twitches of the facial muscles, twitching or tremulousness of the lips or of the muscles about the eyes and forehead, are striking symptoms. The various folds about the face are less evident than in health. Especially is this the case with the naso-labial fold and the lines about the forehead. In addition the face may seem slightly puffed or tumid, that is, fuller than normal; the features may seem flabby and coarse. Sometimes one half of the face distinctly droops or is more frequently disturbed by spasms and tremors than its fellow, so that the two sides appear to have a different expression. When asked to show the tongue, the patient may protrude it by jerky and irregular movements.

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[1] Mickle, loc. cit.; Elkins, Lancet, 1894, p. 1495.

Myosis, which may have been noted in the prodromal period, is now as a rule marked; or, there may be present unequal dilatation of the pupils. Reaction of the pupils to light is either sluggish or is altogether lost. Sometimes other difficulties, such as diplopia, partial ptosis, and other conditions pointing to affections of the ocular nerves are met with.

As may be inferred from what has been said in the account of the prodromal period, the gait is very variable. The latter may still present only a slight departure from the normal. On the other hand, it may be somewhat ataxic or spastic; sometimes, indeed, it partakes of both of these features, and in such cases the elements of an ataxic paraplegia are present. At other times it is atypical. In those cases in which the spinal symptoms are pronounced or in which they precede[1] the cerebral symptoms, abnormalities of gait are more evident. Uncertainty of movement may also be noted in the hands, especially when the patient attempts to button his clothes or to pick up an object. The knee-jerks and other tendon reactions may reveal a condition similar to that met with in the prodromal period; they may be exaggerated, normal, diminished, or lost. It can very readily be understood that the knee-jerks in paresis are a very variable factor; that if spastic symptoms predominate they are increased, and that if ataxic symptoms are in excess they are diminished or lost.

The awkwardness, thickness, and hesitation in speech, present perhaps in the initial period, is in this stage apt to be pronounced. It is noted when the patient attempts to speak that the twitching of the lips and of the facial muscles becomes more marked. The patient stammers, stutters, and pronounces the words imperfectly, often slurring certain syllables.[2] Sometimes syllables are omitted altogether, or there are almost constant breaks or halts between syllables and words. Various factors contribute a share in the production of these phenomena. Some of the symptoms indicate defective action of the cortical centres, the third left frontal and other convolutions. There may be more or less verbal amnesia, and the speech may be constantly arrested by the inability of the patient to recall or frame the necessary words. Frequently also the wrong word is used. The tremulousness and ataxic movements of the various organs concerned in articulation—the tongue, lips, and palate—account in part for the imperfect enunciation. As might be expected, this is especially noticeable in words containing dentals and labials. The syllable containing them may be slurred altogether, or various of the labials or dentals may be substituted for each other, as for instance, a *b* for a *p*, a *d* for a *t*. Generally the speech of paresis is slow, though, when marked exaltation and excitement exists, the words may follow each other with great rapidity.

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[1] The spinal symptoms may simulate locomotor ataxia, lateral sclerosis, combined sclerosis and rarely amyotrophic lateral sclerosis.

[2] Sometimes bulbar symptoms are precocious as well as pronounced, and then give rise to the so-called "bulbar form."

In studying the speech phenomena of this stage we should remember that though the psychic and other symptoms may be pronounced, the speech defect may still be comparatively slight. As a rule, however, defects of articulation can be elicited by asking the patient to pronounce words which require nicety of adjustment of the tongue and lips, for example, "truly rural," "Popocatapetl," and the like.

In keeping with these facts the handwriting is also decidedly changed; it is shaky and irregular, and the patient makes in addition the grossest errors of spelling and of grammar. Elision of letters, of syllables, or of words, occurs with frequency, while the paper is often smeared and blotted. The writing, when it can be interpreted at all, deals disconnectedly with the delusions of the patient. Sentences are incomplete, words run together, and dates and signatures are omitted.

Sensory symptoms are not, as a rule, pronounced in this stage of the disease. However, now and then, there is distinct blunting of the cutaneous sensibility; and this goes hand-in-hand with the diminution or loss of the cutaneous reflexes observed in so many cases. Actual anæsthesia appears to be rare. Various disturbances of the special senses may also be noted; for example, diminution of visual power, color-blindness, amaurosis, or, on the other hand, visual hyperæsthesia. Similar conditions may be noted as regards the senses of hearing and smell. Hallucinations of vision and of hearing, as has already been pointed out, are not infrequent, especially in the depressive form of the disease.

SECOND STAGE OF THE ESTABLISHED DISEASE. Little by little, the various physical and mental symptoms of the preceding stage become more and more pronounced. Conversation becomes more and more difficult, owing not only to the anomalies of speech, but also to the steadily increasing dementia. Many of the previous speech symptoms are accentuated. The words follow each other slowly and are badly put together. The speech is drawling, halting, and stumbling, and at the same time the quality of the voice becomes changed. It may become hoarse, lower in pitch, and may sound hollow and rough, or perhaps indistinct and weak. Less frequently the pitch is raised. Often the voice is monotonous, all of the words being equally intoned. These symptoms are doubtless due to weakness and irregularity of action of the muscles of the larynx. Sometimes, and especially after convulsive and apoplectiform attacks, aphasic symptoms, more or less pronounced, make their appearance, and these may not be confined to mere motor aphasia, but may also include word-deafness. (See page 437.)

Gradually the delusions of grandeur or of belittlement and depression vanish, and only on occasion do they recur, and then in a fragmentary manner. Finally they are altogether lost. In numerous ways the patient shows that his appreciation of his surroundings is more imperfect than before. In every respect there has been an increased diminution in his sensibilities. If he walks at all he stumbles and staggers from weakness and ataxia. Occasionally his steps are short, and he moves as though his limbs were more or less rigid. Movements of his arms are ataxic and jerky, The truncal muscles are weak and their action is irregular. The patient, as he sits in his chair, lolls forward, or frequently to one side. He now fails to evacuate the bladder or the bowel at will, and he frequently becomes extremely filthy. The expression of his face is now one of marked dementia. The various folds and wrinkles of the skin are more or less effaced. The features are coarse and flabby, and the expression is vacuous and indifferent. Occasionally tremors and spasmodic twitchings distort the features, but they seem to correspond to no emotion of the patient. The two sides of the face are sometimes unequal, but decided facial palsy does not exist. Occasionally, also, there is automatic grinding of the teeth—a true masticatory spasm. The pupils, as in the previous stage, respond feebly or not at all to light. They may still be small and contracted, though not infrequently they are in this stage dilated, as a rule unequally. If asked to protrude the tongue, the patient frequently fails to comply. At other times, it is protruded partially and with jerky movements. The patient's habits and manner of eating become more gross than ever, and inasmuch as the fauces and pharynx are blunted, he is sometimes in danger of choking. Digestion is not much disturbed, though sometimes diarrhœa is present.

Bed-sores are apt to form, also boils, carbuncles, blebs, herpes zoster, or the peculiar hemorrhagic formation known as hematoma auris. At various times during this period, apoplectiform or epileptiform seizures, resembling those occurring in the preceding or even the prodromal period, may be observed. In fact, their recurrence at this stage is rather frequent. These attacks are, as before, not followed by any permanent loss of power, but for a number of hours following such a seizure there may be a more or less marked paresis of one side, or perhaps a temporary aphasia. Jerkings and twitchings confined to one or more limbs may be observed for some time after such a seizure. Sometimes the attacks terminate fatally.

THIRD STAGE OF THE ESTABLISHED DISEASE. Finally the patient becomes hopelessly bed-ridden. Voluntary movements are either not attempted, or attempted without evident purpose. They are more irregular, more shaky, more ataxic than before. Locomotion, if it be at all possible, is attended with the utmost difficulty. The legs, especially in the non-ataxic form, are now markedly rigid, and in many cases severe contractures of both arms and legs make their appearance. The legs become flexed over the abdomen, adducted or firmly crossed, while the arms become flexed and drawn over the chest. These cases often present a peculiar picture of distortion. Bed-sores, if not present before, make their appearance now. Other trophic changes, also, blebs, boils, hæmatomata, herpetic eruptions are the rule. The skin has a dirty hue and a greasy feel. The loss of control over the sphincters is now pronounced, and this condition adds greatly to the difficulty of nursing the patient. In addition, he can hardly swallow without the risk of suffocation. The mental faculties are completely, or almost completely abolished. At times some trace of cerebration is observed, but this is all. From now on, until life terminates, the existence of the patient is purely vegetative. Tuberculosis, diarrhœa, inflammation of the bladder, or disease of the kidneys finally ends the picture.

Summary of Symptoms. Although the symptoms of paretic dementia vary greatly, the underlying and essential features are always the same. A brief review will soon convince us that we have everywhere to deal first with gradual loss of function, and secondly with various perversions of function. In other words, we have, first, a *quantitative*, and secondly a *qualitative* change. For convenience it is well to divide the various symptoms into (*a*) psychic, (*b*) motor, (*c*) sensory, and (*d*) general somatic, trophic, and visceral symptoms.

(*a*) PSYCHIC SYMPTOMS. Here the initial symptoms are those indicative of loss, and this fact is especially noticeable as regards those faculties which are among the latest acquired in the development of the individual or in the evolution of the race. It is seen, for instance, in the loss of the æsthetic faculty, in the loss of the sense of the proprieties, of the sense of shame, and of the sense of obligation to family and to friends. The same fact is also evident in loss of memory, loss of will power, loss of judgment, and the loss of power to appreciate properly the current events and the current demands of daily life.

At the same time that these losses are observed, or subsequently, various other changes qualitative in character make their appearance. Prominent among these are the perversions of the moral sense which we have already considered. The patient may lie, steal, deceive, and commit forgery or be guilty of other immoral and illegal acts, such as destruction of property, incendiarism, burglary, and even murder (Mickle). We should remember in this connection that crime committed by paretics is characterized by feeble and erratic motive, by aimlessness and transparency. Either no attempts whatever, or absurdly ineffectual attempts at concealment, are made. Many minor crimes, also, arise not so much from the perversion of the moral nature as from simple loss of memory and loss of appreciation of the environment. This is evidently the case in many small purposeless lies and thefts, the patient in the one instance merely forgetting what has actually occurred, and in the other mistaking the belongings of others for those of his own. This is also usually the case with minor errors of accounts and with mistakes made in paying out or in changing money. The grosser acts sometimes have their origin in the expansive or depressive delusions of the patient.

By reason of the blunting of the higher faculties, the inhibition of the lower and coarser tendencies is lessened, and in consequence various excesses are committed. We have already spoken of the alcoholism so frequently observed in these cases, as well as of the tendency to sexual excess. The latter may lead to improper proposals, attempts at rape, adultery, bigamy, marriage with prostitutes, and various forms of abnormal sexual gratification.

In addition to loss and perversion of function, other psychic anomalies, hallucinations, illusions, and delusions make their appearence. The hallucinations may consist *first*, of anomalies of the general "body sense," the cœnæsthesis, the "Gemeingefühl" of the Germans, and *secondly*, of anomalies of the various special sensations.

The heightened sense of physical well-being or of the opposite condition of physical ill-being, is best interpreted, in the opinion of the writer, as an hallucination of the cœnæsthesis or body sense, that vague feeling of existence to describe which we have no special English word. Hallucinations of this sense may also take the form of various anomalies of consciousness. The patient, for instance, may feel as though he possessed a double personality, or as though his personality were changed. He may feel as though his former personality were passed and gone, and he may speak of himself in the third person; or the feeling may be such as to give origin to the delusion that he has died.

Hallucinations of the various special senses are not infrequently present. Visual and auditory hallucinations predominate. They appear to be present in an almost equal degree. They are found, according to Mickle, in about 40 per cent. of the cases. Tactile, gustatory, and olfactory hallucinations are present in about 12 per cent. The various disorders of the muscular sense are also to be relegated, in great part, to the field of hallucinations. The fictitious sense of great muscular strength, or, on the other hand, the sense of inability to move are, properly speaking, hallucinatory. As a whole, hallucinations are met with to a greater extent in the depressive form than in the expansive form of the disease. Not infrequently, as might be expected, they are linked with the various delusions of the patients.

As regards the illusions of paresis, it is difficult to separate them from the various hallucinations. That, however, they play an important part, there can be no doubt. Especially is this the case with the sense impressions derived from the various viscera and structures of the body. Visceral sense impressions, it must be remembered, enter normally, but slightly, if at all, into the field of consciousness. On the other hand, in paretics they frequently enter, not only largely, but are incorrectly interpreted. Many of the delusions as to the bodily condition apparently owe their presence to these illusions of the visceral-sense impressions. This is notably the case in the depressive form of the disease, in which, as we have already stated, delusions of bodily and visceral ills are very frequent.

The delusions of paresis have been considered in connection with the various forms. Suffice it here to say that for their general character they are dependent upon the presence of the expansive or depressive mental state, while for their special character in a given case they appear to be largely dependent upon the presence of general and special hallucinations. The supervention of the expansive or depressive mental state is doubtless closely related to, if not dependent upon, hallucinatory conditions of the cœnæsthesis, the general sense of bodily and psychic existence. Further, we have also learned that these expansive or depressive mental states frequently become exaggerated, so that mania, hypochondriasis, and melancholia, in various forms, are simulated. We see at once that it is unnecessary to suppose, as Baillarger has done, that paretic dementia is made up of two different elements, a dementia and a superimposed mania, or melancholia. Indeed, the "dual theory," as it is called, adds rather confusion, than clearness, to our conceptions of paresis.

(*b*) MOTOR SYMPTOMS. The motor symptoms may be summarized briefly as general weakness, tremor, twitching, awkwardness and jerkiness of movement, localized paresis, ataxia, abnormalities of gait, and contractures. To these are to be added the speech disturbances and the various apoplectiform and epileptiform seizures. Many of these phenomena, the reader will remember, are only indicated in the initial or prodromal period, and are not infrequently imperfectly developed, even in the first stage of the established disease, though in due course they inevitably attain their full development.

The *speech disturbances* are made up of various difficulties of articulation and of defective action of the motor speech-centre, naming-centre, etc. To these phenomena there are added, as the case advances, difficulties due to the increasing dementia. The ideas which the patient tries to convey become more and more fragmentary. As the disease advances other symptoms may be added—*e. g.*, word-deafness, word-blindness, etc. The abnormalities of phonation which we have mentioned also make their appearance in time. The hand-writing, in turn, presents peculiarities resembling those of the speech. The tremor, ataxia, and jerkiness are variously shown in the formation of the letters, whilst the elision of letters, syllables and words, and the fragmentary arrangement of the sentences in like manner indicate cortical involvement.

Seizures. The gradual and progressive course of paretic dementia may be interrupted, as we have seen, by sudden convulsive attacks or paralytic seizures. Sometimes these attacks resemble ordinary apoplexy. At other times they strongly resemble epilepsy. No sharp lines can, however, be drawn between the so-called apoplectiform and epileptiform attacks, as convulsions and paralysis may be present in both. The apoplectiform attacks, however, are characterized, as a rule, by more or less marked loss of consciousness, accompanied by some form of paralysis, most frequently a hemiplegia, and by relaxation of the sphincters. In an epileptiform attack consciousness may or may not be lost, but there is present a more or less marked convulsion, which may or may not be followed by local paralysis. As a rule, the apoplectiform attacks occur either during the initial or prodromal period or in the first stage of the established disease, although they may occur at other periods. The epileptiform convulsions predominate in the second and third stages of the established disease, although, like the apoplectiform convulsions, they may occur in the earlier periods. It is not unusual to find in a given case that apoplectiform seizures occur first, and are later replaced by epileptiform seizures.

The seizures of paretic dementia may come on suddenly and without warning. At other times they are preceded by a more or less marked increase of symptoms, especially insomnia, restlessness, excitement, flushing of the face, tremor and twitching of the muscles, and sometimes by vertigo. They present also all degrees of severity, being sometimes exceedingly slight and sometimes profound. Thus the epileptiform convulsions vary from attacks which present merely the severity of a petit mal to attacks in which the patient is for many hours in the status epilepticus. The majority of the epileptiform attacks, however, resemble an epilepsy of modern severity. The convulsive movements may be widely diffused over the body—that is, may be general—but more frequently they involve especially the face and arm, or the face, arm, and leg of one side; that is, they are more or less unilateral. Sometimes the entire seizure is limited to one limb or to a single group of muscles, and it may thus simulate a Jacksonian epilepsy. Again, it is not infrequent for a convulsion to start in one hand, one side of the face, or in a leg, and only later on become general. Further, the attacks do not recur as does epilepsy ordinarily—*i. e.*, one attack now and then—but the seizures come on, as it were, in *groups*, though also at irregular intervals. One seizure is rapidly followed by another, until the patient has a series of attacks, with but short intervals between them. Such a group of seizures may extend over several hours or days. Having occurred, they may recur very shortly, or after a more or less prolonged interval, or perhaps never again. In the slighter epileptiform seizures—those, for instance, resembling petit mal—consciousness may not be appreciably affected. If the attack be of marked severity, however, consciousness is apt to be lost. It should also be stated that those attacks in which a series of seizures occur are apt to be followed by paralysis of the parts that were most violently convulsed. It is not infrequent to note distinct paralysis of the face and arm and, to a less degree, of the leg of one side. Sometimes conjugate deviation of the eyes and head is noted. At other times, though rarely, crossed or alternate hemiplegia is found; that is, upon one side there may be ocular paresis, dilated pupil, strabismus, ptosis, etc., and upon the other paralysis of the leg and arm. It may also happen that during a series of seizures the limbs of one side may be in a condition of tonic spasm.

Paralysis, it must be remembered, does not necessarily follow an epileptiform seizure. Further, the degree of paralysis is very variable. It is frequently, but not always, related to the intensity of the seizure. It is most marked immediately after the attack. Subsequently it gradually disappears. Convulsive jerks, and twitchings may persist for some hours, and even days, in the affected limbs. In the typical apoplectiform attack convulsive movements do not occur. However, it is not infrequent to find some indications of their presence. The hemiplegia following an apoplectiform attack persists, as a rule, for several days; sometimes traces of it never altogether disappear. At times a temporary aphasia is present. Both after apoplectiform and epileptiform attacks the patient is dull, heavy, somnolent, and usually presents a rise of temperature. The thermometer may indicate a rise of 4° or 5° F. above normal. Sometimes fever is noted for a brief period preceding the attack. It is also noteworthy that the temperature in the axilla of the paralyzed side, on the side which was most convulsed, is, as a rule, somewhat higher than the axillary temperature of the opposite side. Every now and then, after an apoplectiform seizure, bed-sore, acute decubitus, occurs on the paralyzed side.

It also happens that the seizures occur unaccompanied either by convulsive movements or by loss of consciousness, there being simply a sudden attack of paralysis. Again, instead of there being a clonic convulsion there may be a tonic spasm of the muscles of the head and trunk, the head being depressed into the pillow and the neck and shoulders raised, oposthotonus being simulated. Sometimes other fixed positions are assumed by the patient. These attacks are aptly termed tetaniform[1] seizures. The reader, further, will not be surprised when he learns that in some patients, more especially among women, the convulsive attacks occur which closely resemble hysteria, and these are known as hysteriform[2] seizures.

The various forms of seizures occurring during paresis cannot be sharply separated from each other. Besides, great variations may be met with. However, their influence upon the course of the disease is similar, no matter what their form. It is noted that after a seizure, all of the symptoms presented by the patient become accentuated. This is true not only of the tremor, weakness, and ataxia, but also of the various mental phenomena. Sometimes, as we will see, these seizures are the first sign of the terminaton of a remission.

(*c*) Sensory Symptoms. Cutaneous sensibility is rarely much modified in the early periods. Sometimes pricking and formication of the skin are complained of. Hyperæsthesia is every now and then noted. Later on, however, there is a distinct lessening in cutaneous sensibility, the patient paying but little attention, in the advanced stages, to pricks, pinches, bruises, or injuries of the surface. This loss is, as a rule, more marked about the feet and legs than elsewhere, and more marked about the arms than about the face. These statements appear to be true not only of tactile sensibility, but also largely of the sensibility to pain and temperature.

The sense of taste is, probably, judging from the actions of the patient, sooner or later lost or perverted, as witness the eating of filth and excrement. Smell also appears to suffer in a similar way. As regards hearing, hyperæsthesia may be noted in the earlier periods, and, later, more or less marked loss of hearing.

Vision also suffers. Examination shows that there is a more or less marked loss of vision both for form and color. Indeed, amblyopia, loss of color sense, or amaurosis may be noted early in the case. In a patient recently under the observation of the writer partial reversal of the color fields was noted during the prodromal period. Examination of the fundus of the eye reveals in the advanced stages more or less degeneration and atrophy of the optic nerve. The pupils are, as we have seen, during the early periods apt to be contracted and spastic, though later on dilated, sometimes equally so. Frequently they are oval or ovoid, or present other irregularities of shape. Generally the iris is either sluggish or absolutely immobile to light stimulus. Various external ocular palsies may be noted giving rise to some form of strabismus, or sometimes to a partial ptosis. Nystagmus[3] has also been observed.

In addition to the various disturbances of general and special sensibility there are, it will be remembered, various abnormal sensations present in paresis, such as the dazed and confused feelings in the head, the headache, the neuralgia and ataxic pains of the initial period, and also the various abnormal visceral sensations noted in the established disease.

(*d*) General Somatic, Trophic, and Visceral Symptoms. The circulation, in the larger number of cases of paresis, presents an increased arterial tension. Examination of the heart generally reveals an accentuation of the second sound. The pulse-rate is not markedly changed. Rarely it is slower than normal. More frequently it is slightly increased.

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[1] Mickle: loc. cit.

[2] Loc. cit.

[3] Ballet: Progrès Méd., 1893, 2 s. xvii. 433.

The respiration presents no special change, save that in advanced cases disordered rhythm may now and then be observed—a rhythm which may closely resemble that of Cheyne-Stokes respiration.

Digestion as a rule is well preserved. Decided gastric or intestinal symptoms are absent, save perhaps in the third stage when diarrhœas may occur. Often, too, in the third stage the movements contain undigested food or there may be evidence of mucous colitis or ulceration of the bowel. Hemorrhages from the bowl also occasionally occur.

The secretions are more or less changed. This is especially true of the urine. The latter presents a condition which is apparently related to the altered nutritive processes which obtain in the patient. From the studies that have been made, it appears that during the earlier stages, in keeping with the general increased activity and restlessness of the patient, the urea and chlorides are increased in amount. Later on, as the dementia and the inactivity increases, these constituents diminish in amount. This is notably the case in the last stage of the disease. The phosphates present a variable condition; they are often increased in the melancholic and lessened in the exalted and maniacal phases of the disease. Diminution is more marked in the second and third stages. The urates also appear to undergo diminution with the other solids of the urine as the disease advances. Albumin is found infrequently. It is, however, at times, discovered after a convulsive seizure. Very rarely sugar has been found. Klippel and Servaux have also found peptone, and at times acetone.[1]

The function of perspiration is also modified in various ways. Sometimes there is a dryness of the hands and other portions of the body, or there may be a clammy sweat, or the skin may have a greasy feeling. Sometimes excessive sweating occurs, and when this is the case it is generally local or unilateral. Sometimes the skin of certain regions looks dull and darkened in hue. This is every now and then observed about the temples and forehead.

The saliva seems sometimes to be increased. However, the drooling observed in advanced cases does not depend upon an increased secretion. The latter is merely apparent.

Various general nutritive changes occur in paresis. In the prodromal and first stage of the disease the patient frequently loses in weight, though later on, as he becomes less active, increase in weight occurs, the patient accumulating a soft, flabby fat. In the latter part of the second stage and in the third stage, however, he again loses in weight. Numerous exceptions to this rule obtain. An examination of the blood reveals a moderate degree of leucocytosis with reduction in the percentage of hæmoglobin, changes which cannot be considered as of much significance.

The temperature in paresis, though the disease is essentially afebrile, may present various fluctuation from the normal. Thus it is noted that a rise of temperature generally occurs at the time of and following a convulsive or apoplectiform seizure or a maniacal paroxysm. Slight seizures, however, may occur without any rise taking place. Again, an occasional rise may occur independently of any seizure or disturbance whatever. The tendency to rise of temperature is seen more especially in cases pursuing a very rapid course. Peterson and Langdon,[2] who have studied the temperature in twenty-five cases, conclude that when unusual variations occur in paretics their cause must be sought for in conditions other than the paralytic dementia itself, for example, pneumonia, bed-sores, etc.

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[1] Gaz. Méd., 1894, No. 34.

[2] Journ. Nervous and Mental Dis., 1893, xx. 740.

We have just alluded to the general appearance assumed by the skin in speaking of the perspiration. In addition to various signs of dryness and atrophy, other changes due to perversion of the sweat or oil glands, local trophic disorders, blebs, herpetic eruptions, and ulcers may make their appearance. They occur, as we have seen, more frequently in the second and third stages of the diseases. The bed-sores, which we have mentioned, may be due to the pressure of the buttocks or other portions of the body upon the bed, but more frequently they are trophic in character and occur independently of pressure. In this connection, also, we should mention the perforating ulcer which is now and then found on the ball of the foot. It is a deep trophic ulcer, which is also found in other organic affections, more especially locomotor ataxia More or less marked evidences of vasomotor weakness may be presented by the skin. Thus *tâche cérébrale* is sometimes very readily elicited. If a stroke be made with the finger over the skin, a red streak and sometimes slight swelling follow. At other times this vasomotor weakness is so great as to lead to the actual escape of blood from the vessels. The skin of paretics and the tissues generally bruise very easily, so that subcutaneous and other ecchymoses are not uncommon. Not infrequently punctiform hemorrhages are noted in the skin, and at other times purpuric spots and blotches. Sometimes, indeed, the extravasation is more marked and is accompanied by swelling, as, for example, about the ankles, knees, popliteal spaces, and elbows. The mucous membranes also show evidences of the same vasomotor paralysis in the later stages, and hemorrhages may occur from them, *e. g.*, epistaxis, hæmatemesis, hemorrhage from the bowel and metrorrhagia. At times, and doubtless owing to the same loss of vasomotor control, hæmaturia may be noted, or hemorrhagic extravasation may occur on the surface of the pleura or in the substance of the lung.

Among the most interesting angio-paralytic phenomena met with is hæmatoma of the ear. Without apparent cause, extravasation of blood takes place in the fibrous tissue and beneath the skin of the auricle, and may become very extensive. The swollen ear is dark or reddish-blue in color, though at times, and especially in a recent case, the color may be much lighter. As a rule, after the extravasation has reached its limit, reabsorption takes place, followed by more or less deformity of the cartilage of the ear. In rare cases, where the exudation is enormous, rupture may take place. In other instances, again, suppuration may occur, though this also is rare. Curiously enough, the left ear is more frequently affected than the right, and males suffer more frequently than females. Othæmatoma occurs, it must be remembered, in other insanities and in other nervous diseases, and, indeed, may occur independently of either of these conditions.

Trophic changes are also observed in some of the deeper tissues; thus the muscles may bruise very easily, or spontaneous hæmatomata may occur in them just as they occur elsewhere. General muscular wasting may also take place, and, indeed, is not infrequent in the later stages of the disease. However, in rare cases, true muscular atrophy may occur in various situations due to lesions within the cord.[1]

Trophic changes occur also at times in the bones. The ribs and long bones generally may become very brittle, so that slight falls or blows result in fractures. Trophic changes in the joints are also occasionally met with, the changes being in every way similar to those found in locomotor ataxia (see p. 639), and, like the latter, are doubtless dependent upon lesions of the spinal cord. We find them, as may be expected, in cases of the ascending type, that is, in which the spinal antedate the cerebral symptoms. Extensive disorganization and deformity may ensue without the least symptom of pain.[1]

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[1] Joffroy: Bulletin Méd., 1894, viii. 533.

Remissions. In many cases of paretic dementia, periods occur in which the patient is comparatively free from his delusions—periods in which he seems to have made, for the time being, more or less of a recovery. This is one of the most interesting facts connected with the disease. Remissions are exceedingly important from a diagnostic point of view, especially when we reflect that they may occur in the prodromal period. It not infrequently happens that a patient in this period presents signs of the disease in the afternoon or evening, but presents nothing that is conclusive the next morning. At such times an examination may reveal no symptoms, with the possible exception of certain physical signs, such as slight tremor, inequality of the pupils, and the like. Even these may be in abeyance. As evening appears, the symptoms again return and may be pronounced. Similar in character to these brief diurnal remissions observed in the prodromal period are the more prolonged remissions noticed in the established disease. These may vary from a few days to months, or even years. On examining a patient in a period of remission various symptoms become apparent which prove that although better he is not well. It is found, for instance, that tremor of the lips or inequalty of the pupils, though less marked, persists; and that the speech, though not much improved, is not the speech of a man in health. Similarly, the mental faculties may betray slight weakness or impairment. An interesting fact in regard to remissions is that the psychic phenomena may recede while the physical signs remain pronounced. It is said that the reverse also occurs; that is, more or less marked disappearance of the motor with persistence of the mental symptoms.

The occurrence of a remission in a patient may bring before the alienist a difficult problem. The question always arises, "Should such a patient be dismissed from the asylum in which he has been confined?" Basing our opinion upon the fact that the remission is always more apparent than real, that the patient although better is far from well, and that the disease will sooner or later resume its progressive course, dismissal from the asylum should be resisted, unless the period of remission has persisted for a year or more. Even then the patient should be committed to the care of an attendant or a faithful relative, and constant supervision should be practiced. Mendel thinks the patient should be retained in the asylum three or four years. It must be remembered that such patients are utterly unfit to withstand even the ordinary strains of life, and that by improper management the period of remission may be suddenly terminated, the disease then resuming its sway with renewed intensity. Furthermore, these patients are as little to be trusted in transacting business or performing some serious duty, *e. g.*, the making of a will, as they were during the height of the disease. In other words, a paretic in the period of remission cannot be regarded as possessing legal responsibility.

In connection with remissions a curious fact remains to be stated. Not infrequently they follow and are apparently caused by severe traumata, acute illness, or profuse suppuration. Even recoveries have been attributed to such factors. Among them we may mention erysipelas, burns, abscesses, and fractures.

Duration. A number of factors influence the duration of paretic dementia. First among these is the form which the disease assumes. It is well known that the expansive form pursues a more rapid course than the depressive form, while the simple demented form pursues the slowest course of all. Secondly, the duration is markedly influenced by the sex of the patient. It is decidedly longer in women than in men. Thirdly, all factors of a violently disturbing character, such as apoplectiform and epileptiform seizures, especially if the latter be severe and frequently repeated, as well as the occurrence of maniacal attacks, greatly hasten the course of paretic dementia. Fourthly, all factors that subdue and quiet the patient, such as asylum life, and, especially, the occurrence of remissions, more or less prolong the disease. Visceral complications, according to their nature and degree, also, of course, influence the duration.

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[1] The writer is unable to confirm the statement of Regis that the xyphoid appendix becomes "depressed, elongated, and incurved toward the abdomen." A large number of cases examined at the Philadelphia Hospital failed to reveal conditions other than the variations met with in normal individuals. These are admittedly considerable.

It is difficult, for the above reasons, to make average statements as to the duration of the disease. Suffice it to say that males generally die within two or three years, females within three or four years, while the great majority of all cases die within five years.[1] Cases are occasionally met with in which the symptoms pursue a furibund course, the patient dying at the end of a few months. Even here, however, we should bear in mind the difficulty of determining the time at which the disease actually began. The duration may in reality be longer than at first appears. Again, cases are occasionally reported in which the course is exceedingly long. In these instances it not infrequently happens that the initial symptoms are spinal, and that the latter antedate the actual development of the cerebral symptoms by months and years.

Pathology and Pathological Anatomy. As already stated, paretic dementia is an affection which, more than any other of the insanities, presents tangible lesions. The lesions affect at times the skull, always the membranes, and the brain itself. It is exceedingly probable, however, that even here they are terminal in character.

With the exception of rare cases dying from intercurrent disease at a very early period, the changes are both gross and microscopic. Among them is a chronic leptomeningitis, which is, however, distinguished from other forms of meningitis by the fact that the inflamed and thickened membrane is adherent to the cortex. When the attempt is made to strip off the pia arachnoid a portion of the cortex is always removed with it, the lesion being really one of meningo-encephalitis. These changes in the soft membranes are generally most pronounced over the parietal and frontal lobes. The other portions of the brain appear to suffer at a slightly later period. The change is sometimes accentuated over the occipital lobes. Sometimes it is irregularly distributed, but this is the exception.

Chronic inflammatory changes are not limited to the soft membranes, but in a large number of cases they also involve the dura. The latter is frequently thickened. Sometimes the internal layer suffers alone. Sometimes both layers are affected. The change in the internal layer is not limited to mere thickening. It is frequently accompanied by spontaneous hemorrhagic exudations. The latter frequently recur, and are often so extensive as to give rise to large cysts. This condition is known variously as hæmatoma of the dura mater, arachnoid cyst, or hemorrhagic pachymeningitis. It has already been discussed (see page 351). If the change involves the external layer of the dura we notice, upon removing the calvarium, that it is very adherent, and, further, on examining the cut surface of the bone, we are apt to find that its density is very much increased; in other words, that there has been more or less loss of diploic structure. It may also be increased in thickness, and sometimes this bony overgrowth is accentuated in certain places, so as to give rise, here and there, to exostosis. Even the scalp may take part in this general thickening of tissue, so that it seems more dense and tougher than normal. Exceptionally, instead of osseous hyperplasia, we may find atrophy.

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[1] In Hougberg's cases, one hundred and seven, the duration varied from three months to seven years. Loc. cit.

The surface and the meshes of the pia arachnoid are œdematous. The convolutions are shrunken, especially over the motor and adjacent areas. The fissures are open and sometimes gaping. The disease process does not by any means limit itself to the cortex. It invades the brain tissue as a whole. We notice that the latter is softer than normal, and that the cut surface is more moist than it should be. Here and there also we see gaping spaces, in which bloodvessels lie loosely, and when we turn to the ventricles we find more or less evidence of chronic inflammation of the lining membrane; that is, an ependymitis. The surface of the ventricles is velvety or granular, while the cavities are enlarged and contain an excess of cerebrospinal fluid. Not infrequently they are much dilated.

As might be expected, loss of substance is readily demonstrated on weighing the brain. For instance, in thirteen autopsies made by the writer at the State Hospital at Norristown, the average weight of nine male paretic brains was merely 41.8, and of four female paretic brains only 37.1 ounces.

Early in the history of the case these great nutritive changes appear to be accompanied, or perhaps initiated, by marked disturbances in both the vascular and lymphatic supply of the brain. The vascular appear to precede the lymphatic changes, and we have every reason to believe that they consist in a passive paralytic hyperæmia. Soon both bloodvessels and brain tissue are profoundly affected. Changes appear in the connective and neurogliar elements. Not only do the nuclei in the capillary walls increase, but the nuclei in the immediate neighborhood of the vessels multiply. This is also true of the larger elements of the neuroglia, the cells of Deiters, which become so numerous and so enlarged as to attract special attention. Their appearance is so striking that they have received a characteristic name, that of "spider cells." Very frequently a direct connection between the vascular sheath and a spider cell can be traced.[1] As a matter of course such a chronic inflammatory process means sooner or later interference with the perivascular spaces; and this is the more significant when we remember that these perivascular spaces are the only lymph channels within the brain.

As a result of the general proliferation of the connective tissue and neurogliar elements and of the consequent lymphatic obstruction, and also primarily, the nerve-cells sooner or later suffer. They exhibit such evidences of change as granular and pigmentary degeneration, atrophy, and loss of cell processes. What the obstruction of the lymph path means to the nerve-cell, we can perhaps understand when we reflect that the lymph space within which it lies, the periganglionic space, is merely tributary to the perivascular space, and if the latter be obstructed dilation of the periganglionic space must sooner or later supervene. Like the nerve-cells, the nerve-fibres undergo atrophy and destruction especially in the region of their cortical distribution.

As might be anticipated from the paralysis of the limbs, which becomes more and more marked as the disease progresses, and which is so often accompanied by a late oncoming contracture, we find in many cases evidences of a descending degeneration in the motor pathways. This is every now and then seen well marked in the lateral columns of the spinal cord. In those cases of paresis, on the other hand, which begin with symptoms resembling locomotor ataxia—the ascending form—we find degenerative changes in the posterior columns. The cord may also reveal other degenerative changes, *e. g.*, relative increase of the neuroglia and connective-tissue elements, thickening of the pia and arachnoid, and of the vessel walls, though these are rarely if ever so marked as in the brain. Degenerative changes are also noted in the basal ganglia, in the medulla, and even in the cerebellum. In many cases they are also found in the peripheral nerves. They are noted in the optic nerve during life, and have also been observed in other cranial nerves, though it must be admitted rather rarely. Similar changes have been found by various authors in the spinal and even in the sympathetic nerves.

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[1] The lymph-connective system of Bevan Lewis.

The facts here briefly enumerated, while very suggestive, leave the actual cause of paretic dementia an open question. That some other cause than simple nervous overstrain is at work there can be no doubt. If nervous overstrain were the essential factor it is difficult to understand why a neurasthenia—in one of its graver forms perhaps—should not ensue rather than this strange disease. We cannot avoid the fact that the ordinary result of overwork, even when the latter has been very marked, is in reality a simple neurasthenia, although this may be terminal in form (see page 77). Again, on the theory of simple nervous overstrain, it is difficult to explain the relative immunity of certain races, as, for instance, the Jewish. It is not improbable that in the production of paresis, agents are at work akin to those which probably produce locomotor ataxia and other system or tract degenerations, and though our knowledge in this field is as yet extremely limited and is confined to a few facts, such as the relation of cord degenerations to pernicious anæmia and various cachexias, enough is suggested to make it plausible that profound disturbances in the constitution of the blood, chemical rather than morphological (for example, the presence of some autotoxine or of the toxine of some pre-existent infectious disease as syphilis), may be the initial cause. The theory of a toxine is adopted by Bannister,[1] who holds that paretic dementia is a toxine disease, the toxine being generally the syphilitic poison; that it acts directly on the brain and that syphilis is therefore not a predisposing but an exciting cause. A similar view is held by Morel-Lavallée.[2]

Two opposing theories are held at present in regard to the nature of the pathological process: first, that the disease is primarily an interstitial inflammation, and, second, that it is primarily a parenchymatous affection. The first view has been advocated especially by Mendel. He holds that the disease begins in the vascular apparatus and rapidly involves the neuroglia, the result being a destruction and alteration of the true nerve elements. Mendel's reasons (as summarized by Dagonet[3]) are as follows: First, the dilatation of the capillaries with thickening of their walls and proliferation of their nuclei; second, the result of his experiments upon dogs. (Mendel[4] some years ago produced chronic hyperæmia in the brains of dogs by fastening them on a revolving table, with their heads toward the periphery, and subjecting them so fastened to a series of rapid revolutions for a number of minutes daily. The experiments having been continued for a number of weeks the animals were killed and revealed adherent membranes, thickening and infiltration of vessel walls, and proliferation of the elements of the neuroglia.) Third, the initial symptoms are such as suggest vascular disturbances, for example, vertigo and apoplectiform attacks.

The view that paretic dementia is primarily a parenchymatous disease is held by a number of observers, among them Tuczek and Dagonet,[5] Schütz,

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[1] Bannister: American Journal of Insanity, 1893-94, p. 477.

[2] Revue de Méd., Fevrier 1893. p. 139.

[3] Ann. Méd. Psych., Paris, 1893, 7 s. xvii. p. 395.

[4] Neurolog. Centrabl., May, 1884.

[5] Loc. cit.

Joffroy,[1] and Carter.[2] Tuczek and Dagonet, for instance, believe that the primary lesion is in the nerve-fibres without any antecedent inflammation. Schütz[3] also takes this view and holds that the disappearance of the fibres is to be considered analogous to the system diseases of the cord, for example, degeneration of the lateral or posterior columns. If paresis has existed for a long time, the vascular and interstitial changes become marked and obscure the primary lesions. Joffroy believes the sequence of the pathological changes in general paralysis to be as follows: disintegration of the myelin of the nerve-fibres, secondary vascular changes, and finally neurogliar hyperplasia.

Many facts relative to changes in the cord and also of the peripheral nerves are in keeping with the view that paresis is primarily a parenchymatous disease. Of special value are the studies made by Klippel.[4] Klippel states that the cord is almost invariably affected, and quotes in support of his statement the observations of Tuczek and Fuerstner. In twenty-two of Tuczek's cases only one presented no lesions in the cord; in Fuerstner's cases, 118 in number, cord lesions were present in all. In his own cases, Klippel found the cord also affected. His general conclusions are as follows: First, if a line be passed transversely through a cross-section of the cord, through the canal and cutting the cord into equal halves, the lesions, as far as the white matter is concerned, are situated in the posterior half—in the crossed pyramidal tracts with diffusion into neighboring areas and into the posterior columns. Second, the changes in the lateral columns are generally less marked than in secondary degeneration following cerebral lesion. Third, the posterior columns (although the changes vary) may present the distinctive alterations seen in tabes. Fourth, regarding the gray matter, the lesions of the horns are frequent and may attain a marked degree. Fifth, all lesions are more marked in the cervical and dorsal than in the lumbar region. Sixth, the lesions present a certain degree of diffusion, but the areas which escape are quite limited to the anterior and antero-lateral columns. They have a systemic character in that the uninvaded portions are always the same and that the portions involved constitute in their ensemble a physiological system. Klippel considers the histological process in the cord to consist, first, of a degeneration and absorption of the myelin; secondly, congestion and exudation; thirdly, secondary inflammation. He separates the pathological processes into, first, foci of myelitis produced directly; secondly, dystrophy of nervous elements which is dependent upon the brain lesions; then follow the vascular and connective-tissue changes.

The case reported by Joffroy,[5] in which there was present atrophy of the left hand, is also important, there being atrophy of the large cells of the left anterior horn of the cervical cord. The absence of the changes in the white matter of the cord makes this lesion necessarily a primary one. Interesting in this connection are also the observations of Schütz, who observed disappearance of the nuclei of the hypoglossal and facial nerves.

Regarding the pathology of the tabetic form Joffroy[6] records an interesting autopsy. The latter revealed lesions of the posterior columns, but not those of tabes. The posterior roots were but slightly affected, and there was atrophy of the cells of the anterior horns and Clark's column. Marie[7] points out an important difference between the spinal lesions of the tabetic form of paretic dementia and the lesions of locomotor ataxia; namely, in the former they rise within the cord, *i. e.*, are endogenous, in the latter they begin in the posterior roots, *i. e.*, are exogenous. Joffroy maintains that true tabes complicates general paralysis but rarely. In fact, he regards cases that suggest such a coexistence as merely general paralysis with tabetic symptoms.

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[1] Bulletin Méd., 1894, viii. p. 532.
[2] Brain, xvi. p. 393.
[3] Quoted by Dagonet, loc. cit.
[4] Klippel: Archiv. de Méd. Expériment. et d'Anatomie Patholog., 1894, vi. p. 75.
[5] Loc. cit.
[6] Neurolog. Centralbl., 1894, p. 664.
[7] Gaz. des Hôpitaux, 1894, lxvii. p. 55.

Regarding the frequency of the disease of the posterior columns in paresis, the statistics of G. Renaud[1] seem conclusive. Among 482 cases, it occurred in twenty-five, and of these fourteen presented typical tabes.

Changes in the peripheral nerves and muscles have recently been studied in twelve cases by Campbell.[2] He found frequently changes in the nucleus of the vagus, in the ascending root of the fifth, and rarely in the phrenic nerve. In the spinal nerves he found interstitial and parenchymatous lesions. The anterior roots and the posterior roots between the ganglion and the cord presented constant changes. The spinal ganglia were, as a rule, normal. The muscles showed fatty degeneration and atrophy and hyperplasia of the sarcolemma and the connective tissue. On account of the changes in the peripheral nerves Campbell seeks to establish a relation between general paralysis and the primary intrinsic toxæmic group of multiple neuritis.

The relation of the lesions of paresis to the symptoms seems clear as regards the motor, sensory, and psychic losses, *i. e.*, the palsies and dementia. Regarding the expansive and depressive mental states, "the hallucinations of the cœnæsthesis," it is perhaps not going too far to suppose that they are the outcome of toxic substances circulating in the blood—substances having their origin in deranged metabolism of the tissues.

The epileptiform and apoplectiform attacks of paresis may be explained in various ways. Thus, Mickle[3] holds to the view that fluctuations in brain pressure represent the factors in paralytic attacks—fluctuations called forth by the hindered outflow of the lymphatic fluid. This will happen the more easily the more intensely the cortical functions are damaged by the diffuse disappearance of nervous tissue. Neisser[4] holds that the paralytic attacks depend upon degenerations of the cortical areas. Mendel,[5] on the other hand, thinks that the cause is disturbance of the circulation. Mendel's view certainly accords best with the clinical findings.

Finally, regarding the remissions of paresis, it is not improbable that they are the result of the opening up of the perivasular spaces and the temporary re-establishment of the lymph outflow.

Prognosis. The prognosis of paretic dementia is uniformly unfavorable. A few cases of recovery, it is true, have been reported. The suspicion is, however, always justified that such cases are either instances of mistaken diagnosis, that the patient really suffered from brain syphilis, or that a very prolonged remission was mistaken for a recovery. The disease from its very nature is essentially progressive, and a fatal termination practically inevitable.

Diagnosis. In the very beginning of the disease the symptoms sometimes bear a superficial resemblance to those of neurasthenia. However, the actual psychic *losses* of paresis are never met with in the latter disease. In paretic dementia, in addition to mere weakness and irritability, there is actual loss of memory, loss of the moral sense, loss of the æsthetic sense, loss of judgment, of the sense of the proprieties, of shame, etc. No such thing, for instance, as a careless exposure of the person is seen in the neurasthenic. There is no silly lying, no thieving, no eroto-mania, or any of the other numerous stigmata of paresis.[6]

When the symptoms have become established the diagnosis of paretic dementia can, as a rule, be made with ease; but every now and then cases are met with in which the differentiation from diffuse syphilis of the cortex is very difficult and indeed sometimes impossible. However, both in their general features and in their detailed symptomatology these diseases present important differences. These differences may be enumerated as follows:

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[1] Quoted by Marie, loc. cit.

[2] Journ. Ment. Sci., 1894, xl. 177.

[3] Brain, 1893, xvi. 50.

[4] Neurolog. Centralbl., 1894, p. 666.

[5] Ibid. discussion.

[6] For the differential diagnosis of neurasthenia the reader is referred to Chapter II., p. 77.

First. In paresis the course of the disease is in the main gradual and progressive. Though here and there it may be interrupted by seizures or perhaps by a remission, it is steadily onward toward a fatal termination. In syphilis, on the contrary, the course is very irregular. Frequently the very first symptom that is noted is not change of character or altered mental action, but an ocular palsy or a hemiplegia, while mental symptoms appear only later on. Again, even when the mental symptoms have been established they may suddenly recede and marked improvement may take place, to be followed after some irregular interval of time by a renewed accession of symptoms. On the other hand, spontaneous arrest and disappearance of certain symptoms may occur together with the sudden appearance of others entirely new. As opposed, then, to the more or less progressive course of paresis, let us repeat it, we have in syphilis a course that is decidedly irregular. At times, indeed, the changing and shifting of the symptoms is so marked as actually to suggest hysteria.

Second. In syphilis symptoms almost invariably occur pointing to focal and more or less limited lesions of the brain. In paresis the signs point rather to diffuse involvement. On the side of syphilis, we note such symptoms as a marked dilatation of one pupil, a marked strabismus, a complete ptosis, or it may be a total facial palsy of one side.

Third. Whilst symptoms resembling the above sometimes occur in paresis, the palsy lacks the accentuation or completeness seen in syphilis. Total palsy of an ocular muscle, such, for instance, as seen in complete ptosis, is rarely if ever met with in true paresis. In the latter disease the lid droops rather than drops.

Fourth. In syphilis these palsies are characterized by the suddenness with which they make their appearance, and by the fact that they are temporary, sometimes transient, in duration.

Fifth. In syphilis the physical symptoms as a rule suggest multiplicity of lesions; such, for instance, as the association of left hemiplegia with aphasia.

Sixth. Some of the detailed motor symptoms so common in paresis are but rarely met with in syphilis. This is especially true of tremulous and ataxic movements. In syphilis, for instance, there is no twitching or ataxia of the muscles of expression. Tremor of the lips and tongue is seldom observed, and when met with is much less pronounced than in paresis.

Seventh. The speech disturbances of the two diseases present important differences. In paresis they are apt sooner or later to be pronounced. In syphilis, on the other hand, speech difficulties may be but slightly marked. Again, in paresis we have not only disturbances of the cortical centres, but also tremor and ataxia due to involvement of the centres in the medulla.

Eighth. Syphilitic dementia and paresis also differ notably as regards the character of the delusions. In syphilis mental depression, hypochondriasis, and melancholia are the rule, while the expansive mental state is the exception. In paresis, as we have seen, the reverse obtains. Again, even when expansive delusions do occur in syphilis they are apt to be less extravagant, better systematized, and, taken all in all, more plausible. This brings us to the recognition of another fact, and that is, that in syphilis dementia is, as a rule, less pronounced than in paresis.

Among the various special symptoms in which differences exist between paresis and syphilitic dementia we should first mention headache. This in syphilis is persistent, diffuse, and nocturnal. In paresis, on the other hand, headache is apt to come on in a few isolated attacks of great severity, and sometimes resembling ophthalmic migraine.

Secondly. In syphilis optic neuritis may be observed at a relatively early period, pronounced in type and pursuing an acute course. In paresis the eye-ground changes are both late in onset and chronic in character.

Other factors may prove of value in enabling us to come to a conclusion in doubtful cases. If, for instance, in a given case there is a history of bladder symptoms, particularly of feebleness of expulsion, at a relatively early period, the indication is (see Chapter XXIV.) that the lesion in the cord is of specific origin. Especially significant is it, if these symptoms have spontaneously disappeared, *i. e.*, have been temporary in duration. These bladder signs, the value of which we are just beginning to recognize, are frequently among the earliest indications of nervous syphilis. Further, we are also assisted by a history of comparatively recent specific infection. This, of course, points to syphilis. In paresis, when a history of infection is present, it is invariably of long standing. Finally, we should remember, that while paresis is practically limited to a certain period of life (see Etiology, page 667), cortical syphilis may be met with at almost any age.

While syphilitic dementia and paresis undoubtedly differ in the ways above indicated, it cannot be denied that a residue of cases exist in which the diagnosis cannot be made except by observing the effects of antisyphilitic treatment and watching the progress of the case for a period. Even then, for obvious reasons, we may fail, for syphilitic dementia of long duration is attended by permanent cortical changes, so that improvement under treatment does not occur.

Again, when we reflect that the lesions of paresis and of syphilis, though essentially distinct, may and often do affect similar regions and similar structures, it cannot be surprising that the symptoms of the two diseases may so closely resemble each other that a differentiation is practically impossible. However, in the writer's experience, the more closely the principles here indicated are applied, the smaller is the number of doubtful cases.

As regards diseases other than syphilis, of which dementia may be a prominent symptom, *e. g.*, alcoholism, lead encephalopathy, tumor and other gross organic disease of the brain, it may be stated that the differentiation can, as a rule, be readily made by bearing in mind the characteristic features of these affections. For a consideration of these the reader is referred to Chapters V., XIII., and XVI.

Treatment. The treatment of paretic dementia separates itself naturally into the management of the initial period and of the well-developed disease. The patient who presents some of the suspicious symptoms of the initial period should at once, if possible, be withdrawn from his ordinary occupation and surroundings. All work, physical and mental, should be absolutely stopped. All sources of worry, annoyance, care, or excitement must be avoided. A rest-cure, partial or complete, should be instituted. If the patient has lost in weight, rest in bed for a long period should be insisted upon. Unfortunately, and especially with men, this radical plan cannot, as a rule, be carried out, and we are generally compelled to adopt a system of partial rest treatment. For details of this measure the reader is referred to the chapter upon Neurasthenia.

A number of symptoms demand special attention. The paretic, in the initial period, is frequently a sufferer from insomnia, and to combat this symptom some mild hypnotic should be used. Paraldehyde in doses of from 20 to 30 minims may be given at bedtime, or double this quantity may be administered, suspended in thin mucilage, by the bowel. A drug which is, however, of especial use in this condition, and which unfortunately has met with but little recognition, is antipyrin. This, if used in sufficiently large doses (ten, fifteen, or even twenty grains every four hours), produces a most beneficial effect upon the excitement. Trional in doses of gr. xv. to gr. xx. is also serviceable. Sponge-baths before retiring, hot foot-baths, or brief immersions of the entire body in a hot bath, often has a most beneficial effect in favoring sleep. Tonics, nutrients, and reconstructives generally are, of course, indicated, though little good is traceable to their use.

As already indicated in discussing remissions, improvement occasionally follows surgical procedures. Tuke and Claye-Shaw have noted improvement after trephining over the parietal region. The operation was originally proposed with the idea that in paresis there was present an increased intracranial pressure. Blisters, setons, and issues to the scalp and back of the neck have also been followed at various times by improvement.

Though the ultimate outcome of paresis is unfavorable, it is very probable that remissions can be brought about by a properly directed treatment. The remissions which we at times see in cases admitted to the hospitals are due to the rest, quiet, regular feeding, and the monotony of institution life. General principles must guide the treatment. The same care that is used in the management of other demented patients is to be used here. The same watchfulness is necessary. It is worth while mentioning that in his hurried gulping of food the paretic is every now and then in danger of choking.

Sooner or later he becomes bed-ridden. Because he can no longer feed himself, he must be fed by the attendant, and here it must not be forgotten that, because of the benumbed condition of the fauces and epiglottis, food may readily enter the trachea and give rise to inspiration pneumonia. Again, the bed-sores, which develop sooner or later, require constant attention. We must remember, too, that most of the sores are really not due to pressure, but are trophic in character, as they frequently occur in situations in which the skin has at no time been subjected to pressure. The importance of cleanliness and the difficulty of obtaining this end in cases in which paralysis of the sphincters exists need only be mentioned.

It is important, however, to allude to a remarkable method of treatment of these trophic sores of the insane which has been instituted in the Philadelphia Hospital by the chief resident physician, Dr. Daniel E. Hughes. Corrosive sublimate and other antiseptic washes are abandoned. The sore is simply thoroughly washed with warm water and castile soap, and then thoroughly rinsed. Following this a liquid preparation of beef, Bovinine, is poured over the surface of the ulcer, whilst pledgets of lint are also saturated with the same material. The whole surface is then carefully covered as in ordinary surgical dressing. On removing the latter in the course of a day or two great improvement in the sore is observed. Granulations spring up with rapidity and soon reach the general level of the skin. In due course they assume an epithelial covering. The repair brought about by this novel method appears to be quite durable. The tissue does not seem to be any less resistant than the neighboring skin. Should it break down anew, the treatment is repeated as before. This novel and remarkable method should certainly be adopted in hospitals generally. It appears applicable, furthermore, to trophic sores of all kinds.

CHAPTER XXIV.

SYPHILIS OF THE NERVOUS SYSTEM.

By F. X. DERCUM, M.D.

In other portions of this volume various diseases due directly or indirectly to syphilitic infection are considered. It is the object of the present chapter to present a *résumé* of syphilis of the nervous system and especially to call attention to the general principles which must guide the practitioner in diagnosis and treatment.

The lesions of syphilis are peculiar in that they are especially attended by a formative exudation or deposit. The extent to which this peculiarity may be present in a given lesion varies, however, greatly. Thus a specific inflammation may be so slightly formative as almost to resemble a simple inflammation, or it may be attended by so large a plastic exudation as to lead to extensive new formations. The latter is most frequently the case. These new formations, gummata, or gummatous exudations, as they are termed, closely resemble in their intimate structure, when recent, ordinary granulation tissue. This tissue may develop rapidly and as quickly disappear, and this cycle of rapid growth and rapid retrogression may be often repeated. However, if the new formation persists for any length of time it may become the seat of various degenerative changes, chronic in character. Thus in an old gumma we occasionally observe caseous degeneration. If a gumma be at all large, a number of foci of caseous change may be seen. Fibrous degeneration may also take place, and this may be so complete that after a time nothing but a welt of connective tissue remains of the former syphilitic deposit. Very frequently both caseous and fibrous changes take place in the same gumma, the caseous degeneration taking place in the interior and the fibrous upon the surface.

Syphilitic inflammation affects primarily the mesoblastic tissues of the nervous system—that is, the membranes and the bloodvessels. It is extremely probable that even when an isolated gumma is found deep within the nervous substance, for example, in the centrum ovale, that it has had its origin in the wall of some vessel or in some extension of the pia mater. The membranes may undergo either diffuse inflammation, or, what is more common, more or less extensive local deposits of gummatous material may take place. In the vessels, similarly, we may have either diffuse thickening of the walls or local deposits somewhat resembling atheroma. Syphilitic inflammation of the dura is generally diffuse, while in the pia arachnoid local gummatous deposit is more apt to take place. This is true alike of the membranes of the brain and of the spinal cord. Gummata, though occurring in all situations, are more common at the base of the brain than elsewhere. They frequently involve the cranial nerves and pons, though they rarely attack the cerebellum. When involving the walls of the arteries, they may lead to thrombosis and to consequent softening of the nervous structures supplied by the vessels. At other times rupture and hemorrhage may result. Sometimes aneurisms are formed. Not infrequently one of the larger arteries of the base, such as the middle cerebral or the basilar, is thus affected.

The symptoms to which the above-described changes give rise are varied and manifold. They depend in each particular case upon the location and extent of the lesions. The latter are radically different from those observed in the various degenerative diseases which also occur in syphilitic subjects. Thus in locomotor ataxia we have an extensive degeneration of certain tracts or systems of fibres (see p. 640); that is, a slow progressive change which takes place in the posterior columns of the spinal cord, and which, while it may occur in a specific subject, is at no time specific[1] in character. This fundamental distinction between true syphilitic processes affecting the nervous system and tract degenerations occurring in syphilitic subjects is of the greatest importance. The latter appear to be due to the profound depression of nervous nutrition seen in so many syphilitics, or possibly, as Strümpell supposes, to some toxine, itself a product of the syphilitic germs, whatever they may be. These degenerative diseases also occur, it must be remembered, in non-syphilitic patients.

Syphilitic lesions of the nervous system occur both in the acquired and in the inherited forms of the disease. When they occur in the acquired form they are frequently present as late manifestations. Indeed, it has been the custom to classify the symptoms of nervous syphilis as among the tertiary symptoms. The patient generally presents a history of several years intervening between the initial sore and the outbreak of nervous phenomena. The interval is at times exceedingly long—ten, twelve, and fifteen years being not unusual. Gowers mentions nineteen years as one of the longer intervals observed by him. The writer himself has observed one of twenty, another of twenty-nine, and Wood speaks of even thirty years. A period of from three to ten years is an interval, however, more commonly met with. Although nervous syphilis is, as a rule, a late manifestation, numerous instances are on record in which it has occurred very soon after infection. Thus in speaking of syphilitic thrombosis, Gowers mentions an interval of but six months, and another, less certain, of three months. Wood has placed on record a case of two months and eight days, and in Manchon's collection of cases of precocious syphilis intervals of two and even one month are given.

Regarding the frequency of precocious nervous syphilis, *i. e.*, syphilis occurring very early after infection, the statistics of Fournier are interesting, although they refer purely to spinal cases. This writer collected 71 cases of spinal syphilis. In 8 of these the symptoms appeared during the first year, 18 in the second year, 10 in the third, 10 in the fourth, 17 in the fifth to the tenth, and 8 from the tenth to the twenty-fifth year. In Savard's figures, quoted by Boulloche,[2] there were 26 cases in which the outbreak of symptoms coincided with secondary manifestations, 7 which appeared during the transition period, and 35 in the tertiary stage. Precocious spinal syphilis appears to be somewhat more common than precocious cerebral syphilis. There are no special exciting causes which determine these precocious attacks. In only 7 out of 56 cases collected by Gilbert and Lyon[3] was the onset ascribed to exciting causes.

Inherited syphilis may act in various ways. First, it may so affect the nervous system of the fœtus as to seriously influence its development. This is especially seen in an arrest of growth and development of the brain, many cases of idiocy being directly traceable to this cause. Secondly, lesions identical in character with those of the acquired form may be present in the newborn or may manifest themselves at a later period. If not present at birth they generally make their appearance in early childhood. However, contrary to the usual belief, nervous symptoms due to hereditary syphilis may make their appearance late. They may not be noted until the child is seven or eight years of age, or may even be delayed much longer. Cases from seventeen to twenty-three years of age have been placed on record. In one instance of twenty years the diagnosis was confirmed by an autopsy by the writer. Charcot[1] records the case of a woman, aged thirty years, who was seized with focal epilepsy and in whom acquired syphilis could be excluded. The diagnosis was based upon optic neuritis dependent upon syphilitic basilar meningitis, specific choroiditis, and characteristic headache, linked with focal symptoms; from the early history of the case it appears that symptoms of hereditary syphilis first manifested themselves at seven and again at fourteen years of age. Not only does hereditary syphilis occasionally make its appearance late, but it sometimes manifests itself in unusual and unexpected ways; thus Nolan[2] reports a case of paretic dementia in a boy of eighteen years of age, the subject of inherited syphilis. Though such cases are rare, it is important to remember the possibility of their occurrence. (See also p. 668.) Evidences of inherited syphilis should be sought for in all unusual or anomalous cases of organic nervous diseases in childhood, early youth, and even the beginning of adult life. We should remember, however, that the probability of specific disease diminishes with the length of time intervening between birth and the outbreak of symptoms.

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[1] The terms specific and syphilitic are here used interchangeably.
[2] Boulloche: Ann. de Dermatolog. et Syphil., 1891, 35, ii. 753.
[3] Gilbert et Lyon: Archiv. gén. de Méd., 1889, ii. 404, 536, 662.

Etiology. In the present state of our knowledge it is impossible to explain why syphilis in one instance should attack the nervous system and why in another the latter should escape. It may be that causes that impair the vitality or the resistance of the nervous system invite or predispose to the occurrence of specific lesions. Among such causes various writers, especially Heubner, place excessive brain-work, excesses of all kinds, long-continued strain, whether mental or physical, and profound shock and fright. In some instances trauma also appears to be an exciting cause. Thus blows upon the skull may lead indirectly to syphilitic disease of the bone or even of the membranes of the brain; in other words, in persons who have had syphilis, the lesions produced by trauma may assume a specific character. Lastly, it is not improbable that persons who are hereditarily neuropathic suffer more frequently from nervous syphilis than others.

Symptoms. As already stated, syphilis produces its peculiar pathological changes primarily in the membranes and bloodvessels. These changes are generally widespread and but rarely limited. The symptoms, therefore, in a given case are generally such as point to a number of lesions, or to some widely diffused pathological condition, rather than to a single focus of disease. While this is true, it must be remembered that syphilitic exudations occur most frequently in certain situations, and, as a consequence, certain groups of symptoms are met with more frequently than others. However, the fact that the involvement of the nervous system is frequently generalized or multiple in character cannot be too strongly insisted upon. To see, for instance, brain and spinal syphilis associated is one of the most frequent of occurrences; so much so, indeed, that this association when present constitutes one of the diagnostic features of the disease.

Numerous writers have at various times attempted to set up clinical types of brain or spinal syphilis, but each of these types or symptom-groups has been found by increasing experience to be inconstant and variable. It occasionally happens that, in a given case, at one time cerebral symptoms predominate and at another spinal symptoms, and the same variability is noted throughout the entire symptomatology. Nothing, for instance, like a fixed type of cerebral syphilis can be said to exist. A case that at one time presents the symptoms of basal syphilis may at another present signs of cortical involvement. In the same way, we find that no one set of symptoms can be said to represent syphilis of the spinal cord. Similar groups of symptoms are all that we meet with—nothing approaching a fixed clinical type.

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[1] Charcot: Bull. méd., 1891, v. 131.

[2] Nolan: Journ. of Ment. Sci., 1893, xxxix. 217.

For practical purposes it will be best to consider the symptoms according to whether the lesions predominate in the brain, in the cord, or in the peripheral nerves.

CEREBRAL SYPHILIS.

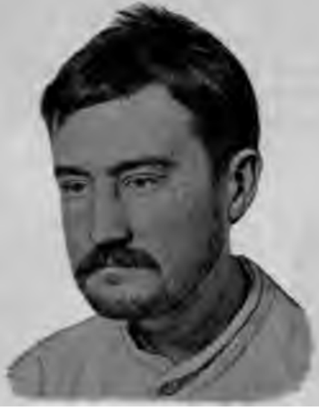
Syphilitic deposits take place either at the base or at the convexity of the brain, and in these situations give rise to well-recognized trains of symptoms; that is, in one group of cases basal symptoms predominate, and in the other vertical symptoms. In a third group, instead of either of these lesions, there is a widely diffused inflammation of the membranes and cortex; the symptoms differ largely from those of either of the other forms in that they present psychic anomalies. Frequently they closely resemble those of paretic dementia.

General Symptoms. We will consider first briefly the symptoms common to or liable to occur in all forms of brain syphilis. Among the most striking and most important of these is headache. This is dull in character and as a rule diffuse. Occasionally it is referred to the vault of the cranium, at other times to the base; it may even be referred to some limited area, though this is comparatively rare. Like other headaches dependent upon organic cause, it is constant and subject to but little variation. At times, however, marked exacerbations occur, most frequently at night, though sometimes in the morning. The symptom is of special value only when associated with others. It may precede more definite symptoms by weeks or months. Not infrequently it is accompanied by giddiness and vomiting.

Next in importance are disturbances of sleep. In the early period of cerebral syphilis the patient frequently suffers from insomnia. Often this insomnia is accompanied by much irritability, and both symptoms are frequently prodromal to some apoplectic or other outbreak. At times the insomnia is caused by the intense headache; at other times it seems to be due to meningeal irritation. The opposite condition, that of somnolence, more or less pronounced, is even more frequent than insomnia. It is noticed, for instance, that a patient will retire early, will sleep through the entire night and perhaps late into the day, and, notwithstanding this increased amount of rest, will often fall asleep while at work, especially if the occupation is sedentary. Somnolence may indeed be so pronounced as to unfit the patient altogether for his business. In some cases, the patient seems, while awake, to be but half conscious, and sometimes he acts as though he were under the influence of a drug. Often he can only be awakened temporarily, or he may act automatically, very much as do intoxicated persons. He may leave his bed, void the urine, evacuate the bowels, or perform some other long-accustomed act mechanically. In short, every possible phase, from great insomnia, which is comparatively rare, to almost continuous sleep, is met with. Somnolence, it should be remembered, often precedes thrombosis of the vessels.

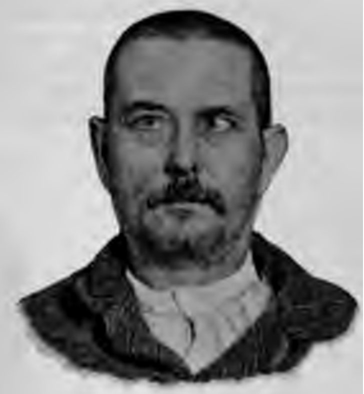
That other symptoms indicative of general cerebral disturbance should be present is not surprising. The patients are frequently apathetic. Often there is a distinct loss of memory with slowing of thought and speech. General mental failure variable in degree is also noted. There is often a slight smoothing out of the lines and wrinkles of the face, such as is seen in a more pronounced degree in dementia.

FIG. 232.



Facial expression in a case of brain syphilis. Patient apathetic and somnolent. (Philadelphia Hospital.)

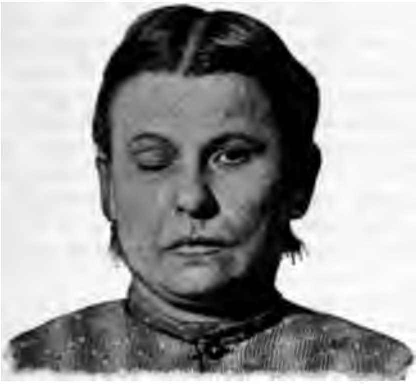
FIG. 233.



Paralysis of left abducens in a case of alternate hemiplegia of syphilitic origin. (Philadelphia Hospital.)

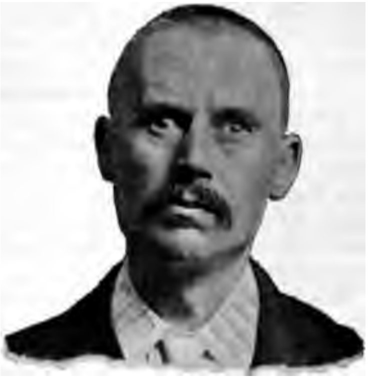
Special Symptoms. SYPHILIS OF THE BASE. As already stated, gummatous exudations take place far more frequently at the base than at the vertex. They are prone, further, to occur in the most central portions of the base, namely, in the neighborhood of the optic chiasm, the interpeduncular region, and adjacent portions of the middle and posterior cranial fossæ. That in brain syphilis, therefore, we should have involvement of cranial nerves is not surprising. The latter may be more or less imbedded in the exudation and suffer both from pressure and from infiltration of their sheaths, or the vessels supplying

FIG. 234.



Ptosis in a case of alternate hemiplegia of syphilitic origin. (Philadelphia Hospital.)

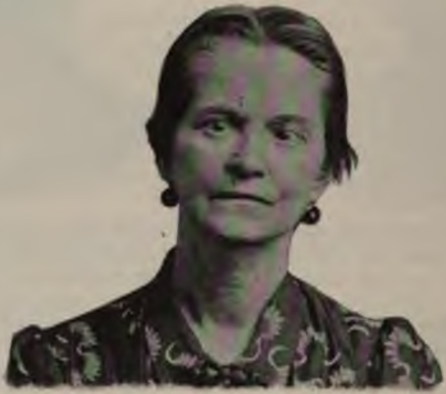
FIG. 235.



Paralysis of internal rectus and dilated pupil; partial internal and external ophthalmoplegia of syphilitic origin. No hemiplegia. (Jefferson Hospital.)

their nuclei of origin may be the seat of gummatous deposit, with subsequent thrombosis and softening. As a consequence numerous special symptoms are present. It is very common, for instance, to meet with unequal pupils, ptosis, strabismus, and other symptoms indicating interference with the oculo-motor nerves. Affections of the optic nerves are also exceedingly common, as is shown especially by abnormalities in the visual fields. The latter are apt to be more or less irregular and contracted, but often distinctive forms of field disturbance are present. Thus a specific exudation at the base may so affect the chiasm as to give rise to a blindness of both nasal halves of the retina, that is, a bitemporal hemianopsia; or it may so involve an optic tract on either side as to produce a lateral or homonymous hemianopsia. Not infrequently the loss of vision becomes so great that total blindness supervenes. These facts, considering the frequency of syphilitic exudation in the region of the chiasm, are of the utmost importance. They are too often neglected.

FIG. 236.



Ocular palsies of both sides in a case of double hemiplegia. (Jefferson Hospital.)

While involvement of the optic nerve tracts and chiasm is most frequently indicated by changes in the visual field, optic neuritis is also frequently met with. We must remember that syphilitic meningitis and localized gummatous deposits may give rise to this symptom just as do other new formations. However, as Gowers has pointed out, the optic neuritis of syphilis is of the acute form, and rapidly becomes intense, differing in this respect from the neuritis present in tumors.

Not infrequently the olfactory nerves are attacked, with a resulting loss of the sense of smell. The trifacial nerve may also suffer. Anæsthesia and hyperæsthesia are rare, but neuralgic pains are occasionally met with. These owe their existence to involvement of the roots of the nerve or of the Gasserian ganglion in the gummatous exudation. (Oppenheim[1] mentions a case in which there was complete anæsthesia of one side of the face, with absence of the corneal reflex and a beginning neuro-paralytic keratitis. In the same case loss of taste on the same side of the tongue was also noted.) Less frequently we have involvement of the facial nerve with a consequent "complete" facial paralysis, that is, one embracing the entire peripheral distribution of the nerve. Involvement of the auditory nerve appears to be quite rare. Wood mentions deafness as occurring. The roots of other cranial nerves, such as the hypoglossal, the vagus, the spinal accessory, may also be attacked, though such an involvement is quite infrequent.

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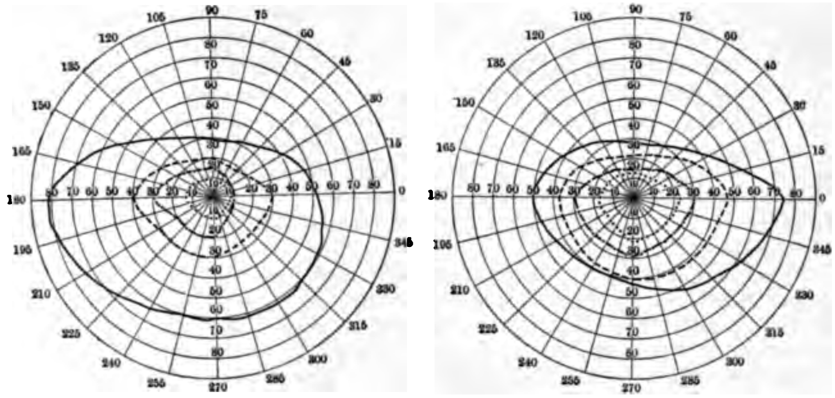
[1] Oppenheim: Syph. Erkrankung. des Central. Nervensyst. Berlin, 1890.

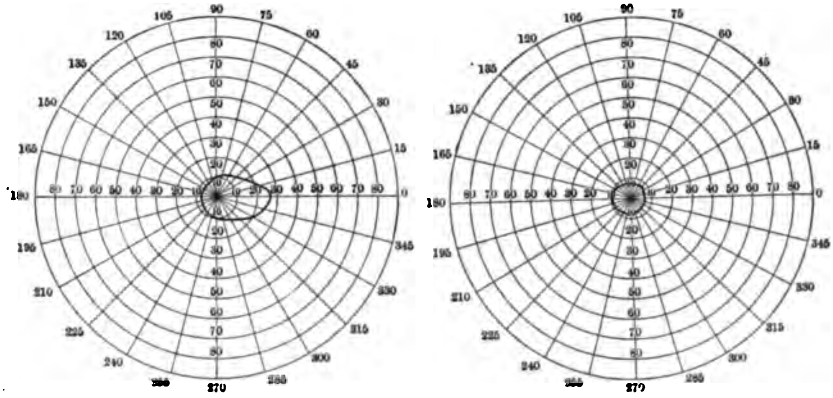
Just as the various cranial nerves may be involved in the exudation so may the vessels of the base. The arteries supplying the basal ganglia and capsules and those supplying the motor area, the middle cerebrals, are among the most frequent to be affected. It cannot be surprising that in such instances symptoms indicating gross lesions of the brain, hemiplegias, and other palsies are present. These are due either to thrombosis or to hemorrhage, thrombosis being the more common of the two conditions. The symptoms resemble in a general way those occurring from ordinary vascular disease of the brain. They present, however, certain important peculiarities. First, the history of the onset is seldom that of an ordinary apoplexy; the attack is rarely sudden. Much more frequently the onset is gradual and is attended by a preservation of consciousness. However, hemiplegia, the result of a non-specific thrombosis, may also be gradual in its onset, as may the hemiplegia of ingravescent hem-

FIG. 237.



Visual fields in syphilis of the base, showing marked contraction. (Infirmary for Nervous Diseases, Philadelphia.)

FIG. 238.



Visual fields in same patient as Fig. 237 six months later, showing extreme contraction.

orrhage. These peculiarities cannot, therefore, be considered as pathognomonic; but their presence in a given case should always suggest syphilis as a possible cause. Again, the hemiplegia, or rather the hemiparesis of syphilis, for the palsy is rarely complete, is apt to be transient or fugacious in character. A cerebral artery may have its lumen encroached upon by specific deposit, but the nature of this new-formed tissue permits the blood-current after a time to be more or less re-established, and, as a consequence, the palsy disappears or becomes markedly diminished. Later on, narrowing in the calibre of the vessel again occurs with a renewal of the paralysis, and this cycle is repeated until more or less permanent paralysis results. The hemiplegia of syphilis is further characterized by the association of other symptoms indicating basal lesions. Important among these are the various cranial nerve involvements, especially the oculo-motor. It is a common experience to see a hemiplegia of *one* side of the body with a dilated pupil or other evidence of oculo-motor palsy upon the *other* side. (See Figs. 233 and 234.) In other words, crossed or alternate paralyses are here very common. (See also Chapter XV., page 459.)

Again, a patient the subject of syphilis may not only have a hemiplegic attack upon one side, but later also a hemiplegia of the opposite side. The exudation at the base having become very extensive, involves in such a case the vessels of both sides of the base of the brain in succession. Other things equal, a history of double hemiplegia, one attack following the other, points to syphilis.

Among the interesting complications met with in specific hemiplegia is aphasia. This may follow the same laws as it presents when due to vascular disease from other causes, or it may be of the fugacious character just described. It is, important to remember that if aphasia be associated with a *left* hemiplegia, our inquiries should always be directed toward determining possible syphilitic infection. Indeed, quite a number of cases have been collected in which aphasia has been associated with left hemiplegia in syphilitic subjects. This is doubtless due to the frequency of multiple lesions in the latter.

Syphilis of the vessels of the base does not, of course, always result in hemiplegia. Every now and then involvement of the basilar and the vertebral arteries give rise to symptoms referable to the pons and medulla.

SYPHILIS OF THE CONVEXITY. The symptomatology, as thus far described, is more especially that of basal syphilis. When the disease attacks the convexity there are present, in addition to the headache, insomnia or somnolence, mental impairment, and other general manifestations, certain special symptoms dependent upon the particular location of the lesion. Thus, the occlusion of a vessel or a gummatous deposit in the motor area will give rise to characteristic focal symptoms; that is, there may be an arm, leg, or other limited palsy of the opposite side of the body, or the patient may suffer from epileptiform attacks, Jacksonian in type. Focal symptoms, sensory in character, are also occasionally met with, though they are decidedly less frequent. Again, in addition to or in place of focal symptoms, others may be present indicative of diffuse or multiple involvement of the membranes and vessels. It is needless to say that such symptoms point strongly to syphilis.

Epileptic seizures are frequently the outgrowth of a general nervous involvement, and in such cases lack a focal character. Like ordinary epilepsy, the attacks may be present either in the form of *petit mal* or of *grand mal*, and may closely resemble essential epilepsy. However, in some cases consciousness is not as completely lost as in the essential form. The latter, as a rule, makes its appearance long before adult life is reached. If, therefore, an epilepsy occurs at a relatively late period, the presumption is strongly in favor of syphilis. This general truth in regard to syphilitic epilepsy was first pointed out by Fournier, who says: "True epilepsy never begins at adult age, at mature age. If an adult man, above thirty, thirty-five, or forty years of age, is seized for the first time by an epileptic attack, and while in apparent good health, there are, I repeat it, eight or nine chances out of ten that this epilepsy is of a syphilitic origin." (See also Chapter XI.) Of course, other causes, such as lead and alcohol, and gross pathological conditions, tumors, traumata, etc., must be excluded.

Among the less frequent symptoms of brain syphilis must be mentioned polydypsia and polyuria. Even melituria and paroxysmal hæmoglobinuria have been recorded.[1] It is probable that in these cases the lesion involves the medulla or is in close proximity to it. Pierre, for instance, found a small gumma pressing on the fourth ventricle.

When brain syphilis is precocious it attacks the membranes and vessels in a manner in no way distinguishable from the ordinary form. However, Schmitt[2] some years ago stated that there is a precocious form of cerebral syphilis which manifests itself as a hemianæsthesia which may or may not be associated with hemiplegia. It may appear in the fourth month after infection, and yields only slowly to antisyphilitic treatment. A lesion of the arterial walls seems to be the pathological basis of the affection. This form must certainly be infrequent, as these statements have not been confirmed.

DIFFUSE SYPHILIS OF THE CORTEX, SYPHILITIC DEMENTIA. As already stated, no sharp distinction can be drawn between the various forms of cerebral syphilis, and it is not infrequent to find symptoms indicative of cortical involvement, such as dementia and other psychic anomalies, in both syphilis of the base and syphilis of the convexity. In addition there is a form clinically well-recognized in which mental symptoms predominate and constitute the most striking features of the disease.

In this group the general symptoms of cerebral syphilis already described (see p. 702) are also noted. Prominent among these is headache, which, as before, is dull, diffuse, and persistent. Often it is a headache that grows worse at night. Sometimes it is increased by pressure upon the skull. Somnolence, too, is also noted, and is apt to be excessive. Often it persists for very long periods, though not infrequently it is broken in upon by shorter periods of insomnia. Vertigo, present in the other forms, may also be a striking symptom here.

In addition to these general symptoms, various psychic anomalies make their appearance. The patient presents a change in character and disposition. It is noticed by his friends that he has become morose, irritable, and depressed, that his memory has become impaired, and that his capacity for intellectual labor has become diminished. He is easily tired out. His facial expression is dull and heavy, and his attitude is one of general relaxation. Most commonly the depression of the patient deepens, and is soon associated with melancholic or hypochondriacal delusions. Not infrequently these take the form of delusions of persecution. Sometimes the depression becomes very pronounced, and there may even be a tendency to suicide. Hallucinations of hearing, or of taste and smell, also commonly make their appearance.

In other cases, and by far the smaller number, instead of being in a condition of depression, the patient may be more or less exalted, and now expansive delusions, "delusions of grandeur" similar to those met with in paretic dementia (see p. 677), may be present. These, however, as a rule, are not quite so extravagant, not quite so variable, and a little better systematized than in the latter disease. In many cases, again, well-marked delusions are absent, a mild cerebral excitement being the only indication of the patient's condition. On the other hand, the excitement may increase until there is great restlessness, anxiety, agitation, violence, or a condition of actual mania. Indeed, we may meet with all degrees of violent cerebral disturbance, varying from an intense mania to an acute delirium.

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[1] Colleville: Gaz. Hebdom. de méd., 1890, 2 s. xxvii. 448.
[2] Schmitt: Congrès Internat. de Dermat. et Syph., 1889, 1890, 726.

The reader will see at once that syphilis may simulate both the expansive and the depressive forms of paretic dementia, and it is not surprising to find that the course pursued is at times similar to that of the simple and uncomplicated form of paresis; that is, there may simply be blunting of the faculties, slowness of thought, and impairment of memory. These symptoms may gradually deepen until, together with the somnolence so frequently present, stuporous conditions, coma, or a condition of sleep-drunkenness may be present.

As a rule, the symptoms are slow in development, but their course instead of being continuous is more or less interrupted. Intermissions occur, during which the patient remains in his previous condition or in which spontaneous improvement takes place. Sometimes a recession takes place which not only embraces individual symptoms, but sometimes the entire group of symptoms. However, after an interval, which is sometimes long or sometimes short, a fresh accession occurs. The course of the disease is also peculiar in that physical symptoms indicative of focal lesions occur early or even precede the mental symptoms. It is very common, for instance, to observe various ocular palsies, such as strabismus, dilated pupil, and ptosis. At other times, aphasia, hemiplegia, or loss of power in one extremity may be noted. These focal symptoms present the usual characteristics of syphilitic phenomena, in that they are sudden in onset, but, as a rule, temporary in duration. As in other forms, they are shifting and fugacious. Other symptoms noted, also, in the other forms of cerebral syphilis may make their appearance, such as amblyopia, amaurosis, or optic neuritis. The latter presents the peculiarities already mentioned (see p. 704). At times also the course of the disease is interrupted by convulsive attacks which may more or less simulate focal epilepsy. At other times seizures occur which are apoplectiform in type, and accompanied by a hemiplegia or other palsy, usually temporary. Less frequently there are attacks of cerebral excitement which may culminate in maniacal outbursts, or the patient may suffer from an hallucinatory delirium, the hallucinations being often accompanied by fear.

While the course of syphilitic dementia is exceedingly irregular, it not infrequently simulates paretic dementia, so much as at times to cause difficulty in diagnosis. Taken as a whole, however, the clinical picture of syphilitic dementia differs markedly from that of paresis. In addition to the peculiar course of brain syphilis just described and the palsies presenting the peculiarities of syphilis, we note that the expansive mental state so commonly met with in paresis is in syphilitic dementia the exception and not the rule. For the detailed differential diagnosis between these two diseases the reader is referred to Chapter XXIII., p. 696.

Other forms of insanity are occasionally met with in syphilitic subjects. They are, however, in the majority of cases an indirect outcome of the disease, and not due to lesions of the brain or its membranes. However, it is said that in some cases during the period of infection, that is, the febrile period, delirium and even mania may occur, but these phenomena are certainly rare. With the exception of syphilitic dementia the various morbid states met with are due to secondary and general causes. It is not infrequent to meet with syphilitic subjects who, as a result of the knowledge of their infection and the probable character of the disease, acquire hypochondriasis and sometimes melancholia (see also p. 712). Here heredity or predisposition to insanity is in many cases doubtless the determining factor. Indeed, similar conditions are sometimes met with in those who have led immoral lives and having suffered exposure are in constant dread of infection, or actually believe that they have been infected. This condition, known as syphilophobia, may even be observed in persons who have never actually suffered exposure, but who have merely been addicted to masturbation. Neurasthenic symptoms and general hypochondriasis are usually found in such cases.

SPINAL SYPHILIS.

As in cerebral syphilis, the virus of the disease expends itself mainly upon the membranes and bloodvessels, and, because the lesions are liable to be both extensive and variable, the symptoms presented are manifold. If the exudation be more or less localized, that is, if gumma be present, the symptoms are those of spinal tumor. If, as is more frequently the case, the exudation be more diffuse and involve also the bloodvessels, various symptoms referable to involvement of the cord itself, of the various roots of the spinal nerves, are present.

The exudation may involve all three of the membranes or may be limited to the pia and arachnoid. The latter may become more or less fused with each other and adherent to the cord. The bloodvessels are especially prone to suffer, their walls becoming infiltrated, and thus the blood-supply of various portions of the cord may be interfered with.

As in brain syphilis, no one "symptom group" exists which is characteristic of all cases of spinal syphilis. It is true, however, that certain clinical forms occur more frequently than others. This fact led Erb,[1] a few years ago, to describe a special complex of symptoms under the name of syphilitic spinal paralysis. While, as we will presently see, all cases of spinal syphilis do not conform to this description, it occurs with sufficient frequency to warrant special consideration. It is characterized as follows: Spastic paralysis of the lower extremities, markedly exaggerated tendon reflexes, low muscle tension, disturbances of the bladder, and but slightly marked disturbances of sensation.

The history, according to Kuh,[2] is somewhat as follows: It usually begins with a slowly increasing weakness and stiffness of the lower extremities, frequently accompanied by paræsthesias. These disturbances of sensation consist chiefly of formication, and are most frequent in the legs. Girdle sensations are also often mentioned, but the patient fails to accurately localize them. Other sensations, such as burning, velvety feelings, feeling as though the patient were receiving an electric current, trembling sensation in the legs or paræsthesias about the anus, and drawing sensations over the back and the legs, are also occasionally noted. These sensory disturbances are, not rarely, the only indications of a lesion of the sensory tracts. Occasionally, also, hyperæsthesia is mentioned as an initial symptom. Actual pains may occur in the beginning of the affection, but they are almost without exception of very slight intensity. Most frequently they occur in the vertebral column or in the sacral region. At times they are present in the form of a girdle. The most varied portions of the body may be the seat of pains. The breast, the intercostal region, the abdomen, the legs, the knees, the hips, the iliac fossa, the buttocks, etc. Serious objective disturbances of cutaneous sensibility appear never, or almost never, to occur. Slight sensory and motor disturbances not infrequently begin in one leg, and only later make their appearance in the opposite limb. More rarely the symptoms pursue an ascending course, that is, spread gradually from the feet upward to the region of the pelvic girdle. Now and then, too, we see in the beginning of the affection jerkings in the legs.

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[1] Erb: Neurolog. Centralbl., 1892, xi.
[2] Kuh: Deutsche Zeitschrft. f. Nervenheilk, 1898, iii. 359.

To the most important phenomena of this period belong the disturbances of micturition that are found in about one-half the cases among the early symptoms. It is characterized most frequently by a weakness of the detrusor urinæ, more or less marked. More seldom there exists in the beginning a weakness of the sphincter. In a number of cases bowel disturbances are also noted, constipation being twice as frequent as incontinence. Not rarely we note among the early symptoms an impairment of the sexual power which appears to be rapidly progressive, amounting to actual impotence. Very rarely, symptoms of sexual irritation, *e. g.*, priapism, are present. In one patient intense pains in the back were attended by sexual excitement, which at times increased up to ejaculation.

When the clinical picture is fully developed we find spastic paralysis of the lower extremities. It is present in all degrees. Very seldom only is a complete paralysis noted, and when this occurs it appears to be temporary in character. Usually we have a very well-developed spastic gait, associated with a relatively slight paresis. Further, in the early stage, the motor symptoms are unequal on the two sides, one leg being more affected than the other. In beginning or in incompletely developed cases the spastic paralysis may be more or less limited to one extremity.

A sharp contrast to the markedly spastic gait is formed by the muscle tension, which is remarkably low. The tendon reflexes of the lower extremities are always exaggerated. Almost constantly do we find an ankle-clonus present, very frequently also a patellar clonus. The deep reflexes of the arms are but seldom affected. The skin reflexes are not especially involved. Decubitus and cystitis occur infrequently, and then only in very severe cases and in the later stages.

According to Kuh, the disease has its origin in all probability in syphilis of the vessels of the cord in the dorsal region, the lesions in the cord being secondary and involving mainly the lateral tracts, and slightly invading the posterior columns. The recognition of this type is exceedingly important, as, according to Erb, marked improvement is apt to occur. Kowalewsky,[1] who has also studied Erb's symptom-group (for it cannot be termed a type), concludes that it is quite common, that it belongs to the ages of thirty to forty-five, and that it is most common in the male sex.

As is well known, ataxia is every now and then present in syphilis of the cord. At times this ataxia is associated with exaggerated reflexes, so that a condition is presented similar to that which is met with in ataxic paraplegia or combined sclerosis. In other cases, again, the ataxia may be associated with absence of the tendon reactions, and the clinical picture may still further resemble that seen in true locomotor ataxia in the presence of an Argyll-Robertson pupil.

When we recall the fact that the virus of syphilis expends itself mainly upon the membranes and bloodvessels, it is not surprising that most varied clinical pictures should present themselves. Not a single symptom can be taken as constant for all cases. Even the spastic character of the gait may be lacking. Instead of flaccidity contractures may be present and, under certain conditions, the knee-jerks may be absent. Indeed, Oppenheim[1] goes so far as to maintain that Erb's type is only a stage of meningo-myelitis. Many cases present symptoms of meningitic irritation and of involvement of the nerve-roots. The clearest and most comprehensive interpretation of the symptoms that has yet been presented is that of Sachs[2], who says: "If the infiltration start from the meninges it invades most frequently the lateral columns first, often at symmetrical points, and advances very slowly from white to gray matter. The intensity of the process is spent often upon the lateral columns, hence the frequency of the spastic symptoms. It may invade the gray matter, giving rise to sensory symptoms, sometimes to atrophic symptoms. It is very apt to recede from the gray matter, restoring sensation, the conditions of nutrition, and the normal condition of the bladder, but it seems to halt at the lateral columns, leaving the spastic symptoms intact for a very long time." How much the clinical picture of spinal syphilis may vary is shown by the occurrence of such phenomena as Brown-Séquard paralysis. Hertel,[3] Oppenheim,[4] and Gerhard[5] especially note the occurrence of this symptom. Gerhard, however, is of the opinion that Erb's type is an actual entity, at least is not always a mere stage of meningeal syphilis, as Oppenheim would make it. Finally, it is important to bear in mind that in spinal syphilis, as in brain syphilis, the same tendency to spontaneous remissions, followed sooner or later by renewed accessions, is found.

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[1] Kowalewsky: Neurolog. Centralbl. 1893, xii. p. 383.

The student should be impressed with the frequency of cerebral complications in spinal syphilis. This fact, which is well recognized, has been especially insisted upon by Oppenheim, Siemerling, and by Sachs. Indeed, the diagnosis of a given case of nervous syphilis should more frequently read "multiple cerebro-spinal syphilis" than either syphilis of the brain or syphilis of the cord, and, when we reflect upon the infectious nature of the disease and the consequent tendency to wide distribution of the lesions, this statement will not cause surprise.

Spinal syphilis is not infrequently precocious in its onset. Erb states that the type of spinal syphilis described by him appears in over half the cases in the first three years after infection—sometimes in the first year. Gilbert and Lyon,[6] however, in their studies of precocious spinal syphilis, tell us that the spinal cord may become the seat of spinal lesions from the third month after syphilitic infection, more frequently, indeed, from the third to the sixth month than later. As a pathological groundwork they distinguish a meningo-myelitis, which is either necrobiotic or which is attended by cellular hyperplasia, by diffuse sclerosis, or by localized gummatous formations.

Sottas[7] in discussing the pathology of spinal syphilis maintains that the condition is due, not to inflammation, but to a process of softening, that meningeal and vascular alterations first ensue which result in the obliteration of the vessels and consequent softening of the cord tissue, and that the sclerosis which results is due to a reactive inflammation. A similar view is adopted by Déjerine.[8] There is apparently never a true primary syphilitic myelitis.

Every now and then gummata are found in the cauda equina, and in such case give rise to symptoms similar to those which other tumors of the cauda produce. (See page 652.)

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[1] Oppenheim: Berlin. klin. Wochenschrift, 1893, xxx. 837.

[2] Sachs: Brain, 1893, p. 405.

[3] Hertel: Charité Annalen, 1890, xv. 214.

[4] Loc. cit.

[5] Gerhard: Berlin. klin. Wochensch., 1893, xxx. 1209.

[6] Loc. cit.

[7] Sottas: Compt. Rend. Soc. de Biolog., 1893, 9 s. v. 359.

[8] Déjerine: Compt. Rend. Soc. de Biolog., 1893, 9 s. v. 432.

SYPHILIS OF THE NERVES.

Cranial Nerves. Syphilis may attack the cranial nerves primarily, but more often, being exposed to syphilitic exudation, they become involved secondarily, or the minute vessels supplying their nuclei become occluded by gummatous deposit. Especially frequent is the involvement of the oculomotor nerve, as already stated. In such instances a dilated pupil, external strabismus, prominence of the eyeball, ptosis, or various derangements of vision may be present. Whenever such phenomena make their appearance as isolated symptoms without special cause, syphilis should be suspected. The sixth nerve may be similarly attacked, the symptoms then being internal strabismus and diploplia. The facial nerve is very infrequently affected; the palsy is generally a complete one, that is, it involves the entire distribution of the nerve, the orbicularis palpebrarum suffering along with the other facial muscles. The trigeminus also is every now and then involved, either at its roots or at the Gasserian ganglion, and in such an instance pain, intense in character—a severe and persistent neuralgia—is present. The optic nerves, as already stated, also frequently suffer. Involvement of the spinal accessory and hypoglossal is also occasionally met with. For a detailed description of the symptomatology of disease of these nerves the reader is referred to Chapters XXVI. and XXVII.

Spinal Nerves. Syphilis of the spinal nerves is excessively rare. As in the case of the cranial nerves, the various nerve-roots may be involved in syphilitic exudation, and thus special symptoms may arise. Single nerve-trunks may also be affected, but acute multiple neuritis, primary and directly due to syphilitic infection, is exceedingly rare, if, indeed, it ever occurs. As shown by Peret, Déjerine, and others, changes take place in the peripheral spinal nerves in many cases of locomotor ataxia. The lesion is, however, a degenerative one, and not due to direct specific inflammation.

FUNCTIONAL NERVOUS DISEASES.

Recently Kowalewsky[1] has studied the relation of syphilis to various functional nervous affections. Syphilis can doubtless produce nutritional disturbances which will not manifest themselves by any visible changes in the central nervous organs. These disturbances may be secondary, first, to changes in the blood; secondly, to changes in the tissues following energetic antisyphilitic treatment; thirdly, to changes in the bloodvessel walls, due to pathological processes; fourthly, to changes in the nerve elements resulting from the psychic shock arising from the knowledge of having acquired so terrible a disease; and, lastly, to alteration of the nervous tissues due to their permeation with the chemical poison of syphilis. The blood changes of syphilis probably begin on the first day of infection, increase progressively, and reach their acme during the secondary stage. Afterward the blood appears gradually to return to normal. These same blood changes are met with in inherited syphilis. Antisyphilitic treatment with mercury in large doses causes a diminution in the red blood-corpuscles, a loss in weight, and impairment of nutrition. In this way Kowalewsky explains the frequency of functional disturbances in tertiary syphilis, especially the occurrence of neurasthenia.

The alteration in the vessel walls commonly met with in tertiary syphilis is a periarteritis. In addition, as we have above pointed out, there are, particularly in the cerebral vessels, gummatous deposits. These changes necessarily influence exosmosis and endosmosis, and consequently the regularity of nutrition of the structures supplied. Under the influences of these conditions we find the development of temporary alterations in the nervous system, such as transient palsies, aphasias, etc., and also hysteria.

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[1] Kowalewsky: Archiv f. Psychiatrie, xxvi. ii. p. 552.

The moral influence of the knowledge of the existence of the disease is a powerful factor in depressing the nervous system. This has also been pointed out by Fournier. According to the latter, neurasthenia is apt to come on between the fourth and fifth month after the initial sore. It is especially frequent in women. It is not, however, limited to the secondary stage, but may also come on during the tertiary period. According to Fournier, it attacks especially the educated classes in the cities, and not the polyclinic patients. The neurasthenia in these cases is, of course, merely symptomatic, and does not differ essentially from the neurasthenia which makes its appearance in the course of other grave diseases.

The direct action of the syphilitic poison is probably a chemical one, and it may affect the function or the structure of the nerve elements. According to Kowalewsky, four so-called functional disturbances are probably attributable to this action of the poison, namely, neurasthenia, hysteria, chorea, and angina pectoris. The neurasthenia here alluded to is, of course, different from that due to the moral shock already mentioned. It is probable that in this form of neurasthenia, to which the term syphilitic neurasthenia should properly be restricted, the chemical composition as well as the molecular structure of the nerve-cells suffer change. Three factors may bring this about: first, insufficient quantity of nutritive material; secondly, a nutritive material altered in its chemical nature; thirdly, inadequate removal of the waste products. All three of these factors are present in syphilis. They are probably also responsible for the neurasthenia which we every now and then see in the subjects of hereditary syphilis. True syphilitic neurasthenia occurs most frequently during the height of the blood changes, that is, during the secondary period. This neurasthenia is distinguished from ordinary neurasthenia by the fact that it is successfully treated by ordinary antisyphilitic remedies. It differs, of course, radically from the pseudo-neurasthenia following the too free use of mercurials. It is probable that in many cases of syphilis the neurasthenia present is of mixed origin, that is, is in part due to the direct action of the poison, and in part to the moral shock. Instead of neurasthenia, hysteria may be developed, and it is not uncommon to find hysteria during the secondary stage. This was also pointed out by Fournier. Psychic and moral factors doubtless play a part in some cases. It is also important to remember that every now and then the children of syphilitic parents suffer especially from hysteria. Hysteria is essentially a degenerative neurosis.

Kowalewsky cites two cases of chorea attributed to acquired syphilis. Chorea appears to be quite rare as the direct result of syphilis, but, according to Kowalewsky, is frequent in the offspring of syphilitic parents.

Regarding angina pectoris, Kowalewsky believes that syphilis may produce it in three ways: (1) by exciting pathological changes in the heart wall, (2) by morbid changes in the vessels, (3) by alterations in the nerves.

While the relations of syphilis to functional nervous diseases are less clear and less striking than in the case of the organic affections, there can be no doubt that these relations are frequently of great practical importance, and in many forms of obscure functional nervous disorders the question of syphilis, either directly acquired or inherited, should be investigated.

INHERITED SYPHILIS. As already stated, the lesions of inherited syphilis are not distinguished by any peculiarity, save when occurring in the early developmental periods. Gross phenomena, such as various arrests of development entailing striking deformities, may then be produced. The pathological changes noted from time to time are most varied. Sclerosis of the cranium, pachymeningitis, leptomeningitis, ependymitis and hydrocephalus, softening and sclerosis of the brain, gummata of the brain, gummata of the cord, etc., are among the various lesions that have been described. It is not surprising that idiocy and epilepsy frequently accompany inherited syphilis. The various pathological changes we have summarized need no elaboration to explain this fact. (See Chapter XII.)

Friedmann[1] maintains that there is also a typical spastic paraplegia in children, apart from congenital spastic palsy, dependent upon hereditary syphilis. It is curable, but apt to recur. It is related to Erb's type of syphilitic palsy in the adult, and is perhaps its analogue.

SYPHILITIC SPONDYLITIS. Syphilis of the spinal vertebræ, that is, specific spondylitis, requires mention inasmuch as the symptoms presented now and then suggest disease of the spinal cord. Further, the recognition is of importance, as the suffering accompanying the affection is very great, and as our therapeutic measures, as a rule, readily control the progress of the disease. There is present deep-seated pain in the back, generally in the neighborhood of the dorso-lumbar juncture. Pain is also excited by various movements of the trunk, but especially by transmitted shock. If such a patient, while standing, raise himself upon the toes and then allow himself to fall heavily upon the heels, great pain is experienced. Torsion and flexion of the trunk likewise excite pain, though to a less extent. In addition, we have now and then symptoms indicating the involvement of various nerve-trunks. Thus, severe pain resembling a girdle pain, or shooting, darting pain, may follow the course of one or more of the intercostal nerves, or may pass along the ilio-hypogastric or the ilio-inguinal nerves. In one case which it was my fortune to see in consultation, the pain followed the distribution of the ilio-inguinal so closely as to suggest the pain often present in renal calculus.

In syphilitic spondylitis deformity of the spine is not present, as a rule, the suffering caused by compression or inflammatory infiltration of the nerve-roots being so great that that the patient seeks relief at a relatively early stage. Specific lesions of the vertebræ can hardly be confused with specific disease of the cord, but the distinction between it and syphilis of the membrane is not always easy.

Diagnosis. In many cases a history of specific infection as well as a history of secondary manifestations is readily elicited. Not infrequently it happens, however, that a clear history of syphilitic disease is wanting, and even exposure to possible infection may be denied. In such cases, we sometimes obtain a history of frequent miscarriages on the part of the wife, of stillborn children, of the death of children in early infancy, or other history suggestive of syphilis. Sometimes the patient admits the history of a sore, but denies all knowledge of secondary symptoms. This occurrence is indeed so frequent among polyclinic cases that many neurologists look upon this history as of itself significant. To say the least, a history of secondary symptoms is not at all necessary to establish the diagnosis of nervous syphilis.

Of equal if not greater importance than a history of infection are the following general diagnostic features: First, *multiplicity of lesions.* Syphilis gives rise to diffuse and multiple lesions of the nervous system. Therefore, symptoms which require for their explanation a number of independent foci of disease point to syphilis. Secondly, the *peculiar progress of the symptoms*, namely, intermissions followed by renewed accessions of symptoms. In this connection the fugacious character of many of the palsies is of great significance. This remarkable peculiarity is doubtless due, as Oppenheim points out, to the nature of the new formation, namely, a rapidly forming and, at the same time, perishable granulation tissue. The pressure or irritation caused by such a new formation must necessarily be very variable. It is not at all uncommon, for instance, to note marked variations in the character of the visual fields, showing that the optic nerve is being exposed to constantly changing pathological conditions. This is also especially true of the pupillary reactions and of the knee-jerks. Finally, there is *the general subacute character of the affection.* The symptoms of a gumma may make their appearance suddenly, but if the patient be questioned closely it will be found that various symptoms, perhaps slightly marked, preceded the sudden outbreak. In other words, while the symptoms often make their appearance suddenly, the growth producing them occupies some little time in its formation; but it is to be remembered that the course of a gumma is far more rapid than that of other tumors. Symptoms of brain tumor persisting with little change for months or years are incompatible with syphilis. Finally, in the detailed diagnosis of the position of the lesion the involvement of the various cranial nerves, the occurrence of Jacksonian or focal epilepsy, or of special palsies, must be taken into consideration.

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[1] Friedmann: Deutsche Zeitsch. f. Nervenheilk, 1892-93.

While the diagnosis of most cases of cerebral syphilis can be readily made, diffuse syphilis of the cortex presents special difficulties. It may simulate paretic dementia, and the differential diagnosis between the two affections may be almost impossible, especially as true paresis is itself often an outcome of syphilis. However, diffuse cortical syphilis pursues a course that when contrasted with true paresis is decidedly atypical. Unfortunately this fact is merely suggestive, as instances of atypical true paresis are not uncommon. However, in many cases of diffuse cortical syphilis there are present recent secondary manifestations in the skin, mucous membrane, hair, etc. The discovery of such signs at once settles the diagnosis. Finally, the occurrence of epileptic attacks, hemiplegias, or other palsies in a manner conformable to syphilis, also assists in the diagnosis. A history of comparatively *recent* specific infection, if present, is also of value; although a large number of paretics present a history of syphilis, the infection is generally of long standing. Paretic dementia, as already pointed out, belongs to the degenerative affections which are apt to occur in syphilitic subjects, and is not directly dependent upon specific inflammation.[1]

If cerebral symptoms are largely in excess, *i. e.*, if the case be one of cerebral or encranial syphilis, the existence of headache, characteristic sleep-disturbances, epileptic or apoplectiform attacks, vertigo, and the peculiar blunting of the mental faculties already described, assist us in making a diagnosis. Headache is generally, though not invariably, a marked feature. Exacerbations, as already stated, frequently occur. Gray[2] lays especial stress upon the tendency of syphilitic headache to periodicity, that is, to a tendency to recur at a certain time in the twenty-four hours; most frequently at or toward night, less frequently in the afternoon or morning. It owes its origin to pressure or irritation of the membranes, and may therefore be simulated by various forms of organic encranial disease, but here the characteristic sleep-disturbances come to our assistance. Marked insomnia, occurring usually at the outset and lasting for a few weeks, is insisted upon by Gray, who also considers as significant the sudden cessation of the headache and insomnia upon the appearance of paralytic or convulsive phenomena. Somnolence and the mental phenomena have already been sufficiently described in the symptomatology.

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[1] For the detailed differential diagnosis, see p. 696.

[2] Gray: Am. Journ. Med. Sci., 1892, ciii. 30.

If, in addition to the general symptons indicative of cerebral syphilis, there occur a hemiplegic attack which is incomplete and rapidly improves, that is, is evanescent or fugacious, syphilis is again indicated. Especially is this the case if the attack occur at a time of life preceding that at which degeneration of bloodvessels from non-syphilitic causes generally ensues. Gowers,[1] for instance, holds that if a hemiplegic attack occurs previous to forty-five years of age, a specific origin is indicated. Again, if instead of a hemiplegia or other palsy, there are present epileptic attacks, occurring for the first time in an individual over thirty years of age, syphilis is also suggested. Headache and sleep-disturbances, it should be remembered, are not always prominent features of cerebral syphilis, and specific epilepsies and palsies may be present when these symptoms are but slightly marked.

In the diagnosis of spinal syphilis we are, as before, to be guided by general principles. Pain referable to the membranes or to the nerve-roots, and various motor disturbances are significant. We have seen, in discussing the symptomatology, that the symptoms may simulate various tract degenerations of the cord, but they are always atypical. Thus, while syphilis of the cord frequently simulates the paraplegia of lateral sclerosis, muscular rigidity, so constant in the latter disease, is generally wanting. Further, bladder palsies, sensory disturbances, belt-like pains, etc., symptoms not present in lateral sclerosis, may be present here. This is also true when the disease simulates locomotor ataxia or combined sclerosis. An interesting instance of a case simulating locomotor ataxia occurred in a negro, a patient in my ward of the Philadelphia Hospital. All of the typical symptoms were present with the exception of the Argyll-Robertson pupils and the ataxic pains. Inasmuch as locomotor ataxia is practically unknown in the negro, and as the symptoms presented differed in at least two important points from those of typical locomotor ataxia, large doses of iodide of potassium were administered, with the result of a rapid and complete cure of the patient.

The fact of the atypical character of the cord symptoms in syphilis cannot be too strongly insisted upon. Thus it may simulate myelitis. However, as pointed out by Sachs,[2] there is not in syphilis a morbid process "which rapidly advances across the entire cross-section of the cord, involving all the symptoms due to loss of function of the various spinal systems. If the syphilitic disease be the result of a specific endarteritis of the vessels of the cord, we know that some and by no means all of these vessels are affected, and that the disease advances slowly from one group of vessels to another. If there be diffuse specific infiltration, it also invades, but very slowly, one part after the other. It has a remarkable tendency, too, to increase for a time and then to recede, whether as a result of treatment or not, and then possibly to increase with renewed force."

As illustrating the irregularity of the symptoms we should again mention the not infrequent occurrence of Brown-Séquard paralysis. In this connection also a case recorded by Beevor[3] is interesting. The patient presented *intra vitam* the symptoms of syringomyelia, but at the autopsy a syphilitic tumor was found on each side of the cord in the cervical region.

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[1] Gowers : Syphilis and the Nervous System, Philadelphia, 1892.

[2] Loc. cit.

[3] Beevor : Lancet, 1893, ii. p. 1252.

Recognition of the symptom group described by Erb is of importance because of the frequency of its occurrence, but that it by no means represents all cases of spinal syphilis cannot be too strongly insisted upon. The diagnosis should always be made upon general principles, and among these the following, formulated by Sachs,[1] may be considered as established: First, the relatively slight intensity of the disease, the palsies rarely being complete. Secondly, the wide distribution of the symptoms, indicating often simultaneous involvement of the cervical, dorsal, and lumbar enlargements; and, finally, the tendency to remissions and relapses.

Regarding the diagnosis of syphilis of the nerves, the facts already stated in discussing the symptomatology must be borne in mind. Important is it to remember that isolated disease of any of the cranial nerves, especially of the optic and oculo-motor, points to syphilis.

In many cases the diagnosis of syphilis is confirmed by the "therapeutic test." If the symptoms disappear after the administration of iodide of potassium or mercurials, the inference is often justified that the lesion is syphilitic. We must remember, however, that the iodides sometimes have a remarkable effect upon neoplasms that are non-syphilitic in origin. Among these we must especially mention gliomata. Further, should a negative result follow the administration of specific remedies, the non-syphilitic character of the affection cannot always be inferred, as the remedies are sometimes unable to arrest the progress of the disease. Finally, it must be borne in mind that the symptoms in a given case are often the result, not of syphilitic deposit, but of the secondary changes in the nerve-tissues themselves, permanent in character.

In studying cases of suspected hereditary syphilis, interstitial keratitis, notched or Hutchinson's teeth, and other signs should be looked for. A history of parental syphilis is of course of great assistance in making the diagnosis.

Prognosis. As we have just seen, the diagnosis of a syphilitic lesion in the nervous system does not necessarily imply a favorable prognosis. Not only may a gumma produce permanent changes in the nerve-tissue with which it is in contact, but it may also undergo retrograde metamorphosis itself; and when caseous and fibrous changes have once taken place, neither iodides nor mercurials can bring about its removal. Further, a symptom, *e. g.*, a hemiplegia, may be due to syphilis of the walls of a vessel, and in such case permanent changes, softening, etc., take place in the affected nerve-centres, changes which our remedies can in no way influence. The same is necessarily true when a hemiplegia is due to a hemorrhage occurring from a syphilitic vessel. In syphilitic epilepsies, also, it is extremely probable that the affection is often the outcome of vascular disease associated with permanent changes in the cortical tissues. In such cases, again, our remedies may fail to arrest the convulsive attacks.

As a rule, the prognosis in a given case is unfavorable in proportion to the length of time the symptoms have existed. A further unfavorable factor is the appearance of symptoms while the patient is under active treatment. It is probable in such cases either that the organism has become "accustomed" to or excessively tolerant of the iodides and mercurials, or that the attack of syphilis is of especial virulence. Frequently, however, especially if the patient be seen early after the appearance of symptoms, the prognosis is very favorable and very brilliant results are accomplished. Cerebral palsies, paraplegia, headache, somnolence, and other symptoms may under these circumstances rapidly disappear.

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[1] Loc. cit.

It often happens that great improvement takes place in a given case, but not absolute cure; for example, a palsy may disappear, but some residual weakness may remain; or headache and somnolence may vanish, but mental weakness and apathy may persist. Frequently, too, though the principal symptoms rapidly improve, special symptoms, such as result from the involvement of some of the cranial nerves, remain unchanged. Thus, there may be a residual inequality of the pupils, some strabismus, or some degree of ptosis. Finally, the age of the patient appears to influence the prognosis. Hjelman[1] thinks that after thirty-nine years the prognosis is not as favorable as in earlier periods. The interval between infection and brain syphilis he believes to be of no consequence, provided the interval be under ten years. After a longer interval than this the prognosis becomes proportionately unfavorable.

Treatment. The treatment of syphilis of the nervous system should be instituted at as early a moment as possible, and should always be of an active character. The remedies consist especially of the iodides and mercurials. Neurologists differ in reference to the relative value of these two classes of remedies. The iodides have, as a rule, a rapid action. However, the results, though they are often brilliant, may not be durable. The mercurials, like the iodides, cause the rapid absorption of syphilitic deposits, and it is observed that the effects of a thorough mercurial course of treatment are more persistent than if the iodides are used alone. Besides stimulating absorption, the mercurials appear to act in a manner antagonistic to inflammatory processes in general, and, further, it is not improbable that they have a destructive or antagonistic action upon the specific virus itself.

The objects to be gained in treating cases of nervous syphilis are, first, to remove the deposit or infiltration with the greatest possible rapidity, and, secondly, to make this result as lasting as possible. Reliance should, therefore, not be placed upon one drug to the exclusion of the other. It is the writer's custom either to use both together or to follow one by the other. A good plan is to administer the iodide of potassium in rapidly increasing doses until 20, 30, 40, or even 60 grains are given three times a day. For short periods of time, in urgent cases even, this quantity may be exceeded, as the tolerance to the drug is exceedingly great. At the same time that the iodides are being administered mercurials in some form or other should be added to the treatment. The writer's preference is the use of the protosalts, so guarded as to prevent purgation, or, what is better still, the cautious use of mercurial inunction. A small quantity of the ointment, say 20 grains, should be rubbed into the axillæ every day, and, should the patient prove tolerant to the drug, this amount may rapidly be increased to 30 or 40 grains or more. In urgent and desperate cases much larger quantities should be used. As soon as tenderness of the gums is noticed the inunctions should be absolutely stopped, but as soon as this symptom subsides they should be cautiously resumed and the patient kept under the influence of the drug off and on for a long time.

The question next arises as to how long an active course of antisyphilitic treatment should be pursued. If the case be amenable to treatment, improvement is noted at the end of several weeks. Sometimes the improvement is noted in a few days. More often it happens that, after a certain amount of improvement, the case reaches a standstill. Under these circumstances it is advisable to increase the amount of the iodide or the mercury administered. If under increased dosage no improvement takes place, it is probable either that the patient has become habituated to the medicines or that a maximum amount of recovery for the time being has been attained. Under these circumstances it is better to abandon the specific treatment altogether for a time and to administer, together with a liberal diet, reconstructive remedies, such as iron, the hypophosphites, cod-liver oil, and such tonics as strychnine and arsenic. We habitually underestimate the reparative powers of the nervous system. Very frequently, after specific lesions have been removed, the symptoms persist, but the nervous tissue, freed from pressure or other pathological interference, in the course of time, weeks or months, recovers, and the symptoms eventually disappear. This process can take place only if the interference has not been of such a nature as absolutely to destroy the parts affected.

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[1] Hjelman: Helsingfors, 1892.

In some instances it is advisable to return to the use of the iodides and mercurials after an interval of several weeks or months. Very frequently a persistent treatment, in which specific remedies, reconstructives, and tonics are alternated, is followed in the course of months, and sometimes years, by the most gratifying results. In two instances in the experience of the writer the maximum amount of improvement was only attained by this method at the end of three years. Both were grave cases of syphilis of the cord.

Persistence in treatment is especially indicated because of the well-known tendency of syphilitic lesions to recur. Gowers[1] states that every patient who has had syphilis should take at least eight years from the primary disease, or for five years from the last manifestation of it, a course of iodide for three weeks, twice a year.

The hypodermatic method of administering mercurials has nothing special to recommend it in the treatment of nervous syphilis, the most rapid impression being made by the method of inunction. However, when it is essential that the nature of the malady should be concealed, or when inunctions, because of hot weather, become exceedingly unpleasant, the hypodermatic method may be resorted to with advantage. One-tenth of a grain of bichloride of mercury, dissolved in 10 or 15 drops of distilled water, may be administered every second or third day. The injection is best made into the buttock or into the loose tissues of the back. "Gray oil" is also occasionally used. It consists of an emulsion of metallic mercury in lanoline, in the proportion of two to one; this is diluted for hypodermatic use in the proportion of three parts of ointment to one part of olive oil.[2] One to two minims may be administered at intervals of every second or third day. Sacaze and Magnol[3] state that in three cases of cerebral syphilis they obtained very prompt results from its use.

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[1] Gowers: Internat. Med. Mag., 1893, ii. 823.

[2] L. Wolf: "Hypodormatic Medication in Syphilis." The College and Clinical Record, May, 1893.

[3] Sacaze et Magnol: Ann. de Dermat. et Syph., 1893, 35, iv. 943.

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CHAPTER XXV.

DISEASES OF THE NERVES.

By WHARTON SINKLER, M.D.

THE diseases of the nerves are: Congestion or hyperæmia, inflammation, degeneration, new growths, and functional disorders.

HYPERÆMIA.

Hyperæmia may occur as the result of exposure to cold, injuries of various kinds, or compression. Rheumatism or gout may also give rise to congestion of nerve-trunks. Hyperæmia may occur as the result of disturbances of nutrition connected with the menopause, or with overuse of a part.

The symptoms of hyperæmia of a nerve are principally sensory. There is a sense of burning or numbness in the distribution of the affected nerve, and various forms of paræsthesia. The numbness and tingling may be so intense as to amount to positive pain; but in the majority of instances there is merely a sense of prickling and formication, such as occurs when the part is "asleep." Tenderness is sometimes found over the nerve-trunks, but this is rarely met with. A slight amount of muscular weakness is also present.

ACROPARÆSTHESIA (NUMB FINGERS, WAKING NUMBNESS. This disease may be considered in this connection, as it is probably due to hyperæmia of the peripheral nerves. It is met with commonly in women at about the climacteric, but it may occur in either sex. It occurs most frequently in women who have hard manual work, such as washing, scrubbing, cutting with scissors, and sewing. It can sometimes be traced to a gouty or rheumatic diathesis. In other cases there is no history of excessive use of the hands, but it appears to be connected with disturbances resulting from the menopause.

The symptoms are numbness and formication of the extremities, which first begin in the hands. Usually the numbness is felt when the patient wakes in the morning, and it soon passes off after the hands have been used. As the disease advances the numbness extends up the arms to the shoulders, and invades the lower extremities. It then comes on when the patient has been lying down only for a short time, and persists for longer periods. In aggravated cases the patient is awakened from sleep by the numbness, and is obliged to get up and walk about and rub the extremities before the discomfort is relieved. In some cases the numbness becomes painful, like the distressing sensations which occur in a limb which has been "asleep." In women there is frequently associated with the paræsthesia, general flushings followed by profuse sweating. Usually there is no muscular weakness or anæsthesia, but the fingers are clumsy, and the patient is unable to sew or button her clothes. In some cases the paræsthesia is confined to the ulnar distribution alone; but, in other cases, the scalp and ears are affected as well as the extremities. The symptoms are frequently accompanied by general nervousness and restlessness. The urine is often excessive in quantity, and contains an undue proportion of phosphates or urates.

Prognosis. The disease may last only for a few weeks or months, but relapses are frequent, and sometimes after a whole year there will be a return of the affection. Although the condition is often of long standing, it never runs into any serious disorder.

Diagnosis. The disease is to be distinguished from hysteria or organic disease of the brain or cord. In hysteria, the distribution of the numbness is generally localized or unilateral, and there are other evidences of hysteria; in organic diseases of the brain or cord, the numbness is either unilateral or confined to the lower extremities, and is associated with paralysis. In Raynaud's disease, there is spasm of the bloodvessels, producing pallor of the fingers.

Treatment. The most successful plan of treatment, in my experience, has been the administration of ergot in fairly large doses. Relief usually follows this remedy in a short time. Faradization and massage are useful adjuvants in treatment; and special attention should be paid to the general health. Salt baths and tonics are always of advantage; and change of air, when it can be obtained, is very desirable. After the acute symptoms have subsided, strychnine or arsenic, combined with iron and quinine, should be administered.

NEURITIS (Inflammation of the Nerves).

Neuritis is met with in two forms, inflammation of a single nerve or groups of nerves, and multiple neuritis, or a general inflammation of all of the peripheral nerves.

When inflammation of a single nerve occurs it is generally as a perineuritis, in which the outer sheath of the nerve is affected, or it may involve the connective tissue between the bundles of the nerve-fibres, constituting an interstitial neuritis. When the nerve-fibres are affected, as occasionally occurs, it is then called parenchymatous or degenerative neuritis. In simple neuritis one nerve-trunk alone may be affected, but two or more may suffer at the same time.

Neuritis may be acute, subacute, and chronic, and the inflammation may begin near the periphery of a nerve and extend upward—neuritis migrans.

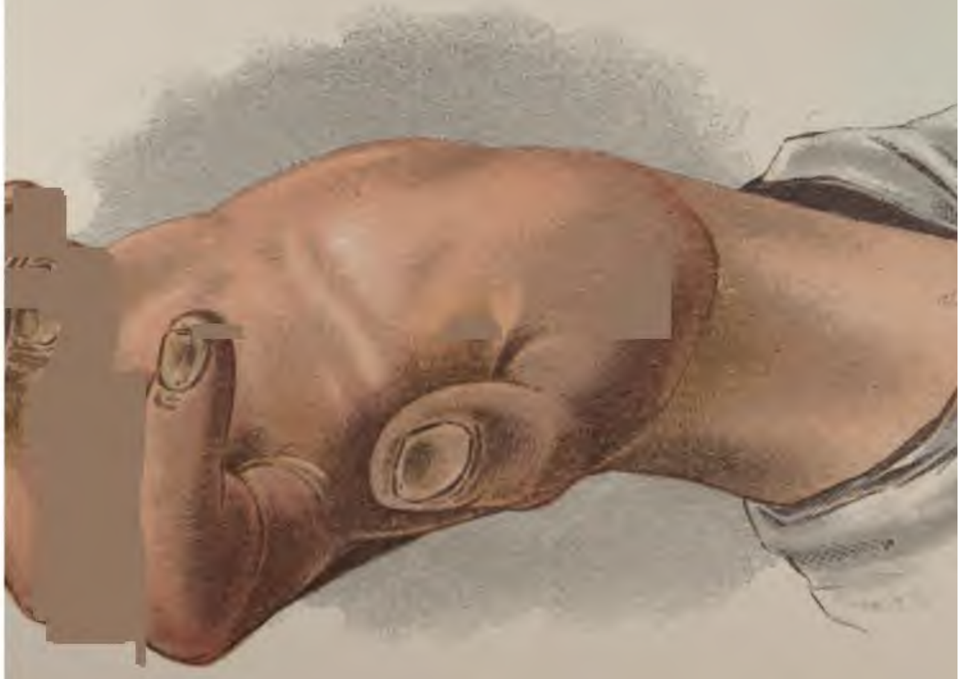
Etiology. Simple neuritis results most frequently from injuries, wounds, and contusions, or compression of the nerve-trunk, either from external pressure or from tumors and growths, which squeeze the nerve in a bony canal, through which it passes. Nerves in the neighborhood of joints are frequently injured by dislocations, and occasionally a nerve undergoes compression or contusion through extreme muscular action. In fractures a nerve is occasionally caught in the callus, and thus injured. Neuritis may also arise from an extension of inflammation in adjacent organs, as in arthritis. Cold and the rheumatic and gouty poisons are also sources of neuritis. Inflammation of nerve trunks may also be due to a cachexia, syphilitic or cancerous, and may also arise from leucocythæmia. Occasionally an injury of a nerve near its periphery will cause an ascending inflammation—neuritis migrans—which may extend as high as the plexus, from which it arises, and involve other nerves. Injuries of nerves from cuts with glass seem especially prone to be followed by ascending neuritis. In a patient whom I saw several years ago, the extensor tendon of the middle finger was divided by a broken pane of glass. The wound healed speedily and without ill effects, but the patient was unable to extend the finger on account of the divided tendon. An operation was performed to suture the ends of the severed tendon, and care was used to observe all antiseptic precautions. The tendon united in a short time, and the perfect use of the finger was restored, but a neuritis began at the point of incision and extended up the forearm and up to the shoulder, causing much pain and joint difficulties.

Pathological Anatomy. An acutely inflamed nerve is red and swollen, the bloodvessels are distended, and are plainly visible on the surface in the nerve-sheath. This condition is soon followed by œdema or sero-fibrinous exudation, and sometimes the exudation is jelly-like. Under the microscope leucocytes are found surrounding the vessels and infiltrating the sheaths of the nerves. Small extravasations of blood are also met with. When these changes are confined to the nerve-sheath the condition is known as perineuritis; but if it extends into the tissue between the bundles of nerve-fibres it constitutes an interstitial neuritis. In the latter condition leucocytes are found in the septa between the nerve-bundles, and may even be found in the fasciculi. These changes may extend the whole course of a nerve, but generally the entire nerve-trunk is not affected, and intervals of healthy tissue are found.

The nerve-fibres do not show much change in perineutritis unless the nerve has undergone compression through the swelling, and the fibres have in this manner been injured. In interstitial neuritis the fibres are frequently affected, but not invariably so. In parenchymatous neuritis the inflammation begins in the nerve-fibres, and the interstitial connective tissue is not much disturbed. In parenchymatous neuritis the changes in the nerve-fibres are the same as in degeneration; the medullary sheath is first swollen, then the myelin breaks up into segments, greater or less in size, and in time these become divided into small fragments. The masses filling the nerve-tube then become cloudy and granular, and the axis-cylinder is interrupted wherever the myelin is divided. The nuclei of the sheaths are increased. Later the axis-cylinder disappears entirely, the sheaths become empty and shrivel, and are replaced by connective tissue which contains much fat—the lipomatous neuritis of Leyden. The degenerative process often stops at the first node of Ranvier above the seat of the injury, but it may extend upward and through the whole length of the nerve. The process goes on to complete destruction of most of the fibres in the nerve, with, occasionally, a few unchanged fibres remaining. After a time regeneration begins. This occurs either from the development of new axis-cylinders and sheaths from the nuclei in the old sheaths or from the growth of axis-cylinders from the undegenerated part of the old nerve-fibre; the latter view is held by Ranvier.

Symptoms The symptoms of neuritis depend largely upon the function of the nerve which is affected. Pain is a prominent symptom in all cases, and there is more or less tenderness over the course of the nerve. In the case of a sensory nerve there is severe pain in the distribution of the nerve, with cutaneous hyperæsthesia and sometimes impaired sensibility. The pain is sometimes almost unbearable. In a case of median neuritis under my care the thumb, index, and middle fingers felt swollen and acutely painful, as if they were about to "burst." In inflammation of a mixed nerve there is a sense of pain, burning, or tearing in the course of the nerve, and the motor symptoms are also conspicuous. There is paralysis of the muscles supplied by the nerve. The degree of paralysis varies, according to the extent of the neuritis. In some cases loss of power is only partial, but in many cases, especially those in which the neuritis has resulted from injury or compression, there is a total loss of power. The electrical reactions are altered, and within a few days there is marked quantitative change to the faradic and galvanic

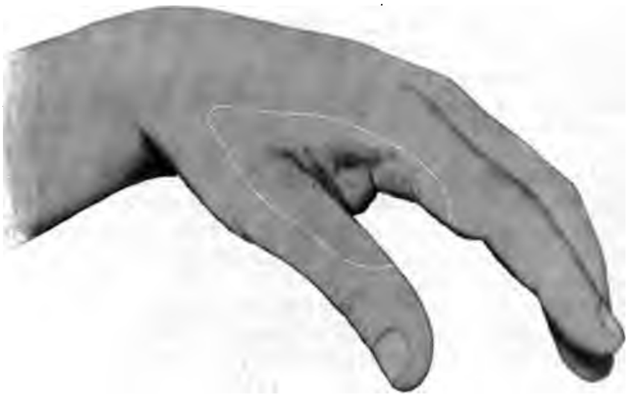
PLATE IV.



Glossy skin, result of neuritis of median, ulnar and radial nerves. (Mitchell.)

currents. After a short time there is complete loss to the faradic current, and the reaction of degeneration to the galvanic current is then present. (See p. 38.) Trophic changes are frequently met with, and the joints become swollen, inflamed, and the skin red and glossy. In the fingers there is often atrophy of the subcutaneous tissue, so that the fingers become thin and tapering to the extremity—the bone may be atrophied. The nails become brittle, rough, and ridged, showing transverse furrows, and the whole nail may be lost. (See Plate IV. The illustration shows beautifully the change in the skin and nails in a patient of Dr. Weir Mitchell, in whom a fracture of the forearm had injured the median, ulnar, and radial nerves, setting up a neuritis.) The growth of hair is altered; it may fall out or become coarse. Œdema often occurs in parts where there is much subcutaneous cellular tissue. In wrist-drop from musculo-spiral palsy there is usually a thick hump on the back of the hand (Fig. 239).

FIG. 239.



Wrist-drop from division of musculo-spiral nerve. The white line encloses an area of partial anæsthesia.

Vesicles, bullæ, or herpetic eruptions may form in the parts supplied by the affected nerves, as, for example, in herpes zoster, herpes frontalis, and herpes cruralis. In a case of neuritis which I saw, the result of division of one of the digital nerves by a piece of broken glass, a series of bullæ formed in succession on the finger. In another, a case of median neuritis, large blisters occurred on the index finger, and these were followed by ulcers, which healed slowly.

Among the nerves most frequently affected is the facial, which is liable to neuritis, from compression in the aquæductus Fallopii, in which case all of the muscles supplied by the nerve are completely paralyzed. The nerve may also undergo inflammation, as the result of cold draughts against the face or of rheumatic influences.

The musculo-spiral nerve is frequently the seat of inflammation, and the most common cause of this is injury. The palsy resulting from compression neuritis of the musculo-spiral nerve has been called "Saturday-night palsy" or "Sunday-morning palsy," because frequently a man who has been drinking heavily on Saturday night, sleeps with his head upon his arm, or he may sleep sitting up, with his arm resting against the back of a bench or chair, so that the musculo-spiral nerve is compressed. The musculo-spiral nerve may also be injured by sudden and violent action of the triceps muscle. Another way in which I have known this nerve to be injured is when a patient has been placed upon a narrow table for a gynecological operation, and the arm allowed to fall over so that the back of the arm just above the elbow rested against the sharp edge of the table. Lead-poisoning often attacks the musculo-spiral nerve alone, giving rise to a neuritis. Many years ago I called attention to the similarity between lead palsy and paralysis from musculo-spiral compression,[1] and expressed the belief that lead paralysis was due to a lesion of the peripheral nerves. The musculo-spiral nerve lies so superficially that when a person is exposed to severe cold, as in driving through a snow-storm, the nerve may become inflamed.

The median nerve is not infrequently the seat of neuritis, and when this occurs there is disturbance of sensation on the palmar surface of the thumb, fore-, and middle-finger. There is more or less anæsthesia in this region, depending upon the degree of neuritis; but in most cases the anæsthesia does not extend beyond the palmar surface of the thumb and first two fingers, although it is stated that there is also loss of sensation on the back of the last phalanges of those fingers, and also of the radial half of the ring-finger. This extensive anæsthesia does not occur except when there has been complete abolition of the function of the nerve. There is pain and tenderness in the course of the nerve, and pain in the thumb and first two fingers, which is frequently intense. In a case which I have seen, a tap on the end of the forefinger would cause an acute pain, running up the whole course of the nerve. The forefinger is often swollen, and trophic changes occur, such as I have already described. There is loss of power in the pronators of the wrist, which is usually incomplete, and inability to flex the first two fingers or to abduct the thumb. The median supplies the flexor communis, but the outer half receives branches from the ulnar nerve, so that the third and fourth fingers can be flexed.

The causes of median neuritis are exposure to cold, or direct violence. I saw recently a young man, who, in a wrestling bout, was grasped violently by his opponent about the junction of the middle and upper third of the arm. The median nerve must have been tightly squeezed by the ends of the fingers. When the patient was seen by me there was but little tenderness at the seat of the injury, but the nerve below the elbow was exquisitely sensitive, and there was paralysis of the flexors of the thumb and forefinger, with inability to abduct the thumb. There was anæsthesia on the palmar surface of the thumb, index, and middle-fingers to light touches. These fingers were the seat of such acute pain of a throbbing and burning character that the patient was unable to sleep at night. Another case of median neuritis was caused in a patient who had been cutting meat with a dull knife, so that great pressure was made upon the palm of the hand against the back of the blade. The neuritis extended up the forearm, causing much tenderness in the course of the nerve and anæsthesia in the region supplied by the nerve.

In ulnar neuritis there is pain and loss of sensation in the outer half of the third finger and in the fourth finger, with paralysis of the flexor carpi ulnaris, and the outer half of the deep flexors of the fingers, together with the intrinsic muscles of the little finger, the interossei lumbricales, and the adductor of the thumb. In cases of long standing of ulnar neuritis there occurs the typical "claw-like hand," owing to overextension of the first phalanges and flexion of the last two. Ulnar neuritis often results from pressure upon the nerve during sleep, or from direct injury to the nerve where it passes between the projecting bones of the external condyle and olecranon. Gowers speaks of a case in which there had been two attacks of ulnar neuritis following successive confinements. In a patient which Dr. H.

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[1] American Psychological Journal, November, 1875

R. Wharton kindly allowed me to see, the blow of a stick displaced the nerve from its groove behind the internal condyle of the humerus so that it lay on the outside of the condyle.

The circumflex nerve is quite frequently the seat of neuritis, either from injuries, like blows upon the shoulder, or cold and rheumatic influences. I saw a case of neuritis in this nerve in a professional athlete, who had been in the habit of allowing a bar of iron to be bent by a blow over his shoulder, in order to show his muscular development. Pressure during sleep, or during a prolonged illness, is sometimes the cause of inflammation of this nerve. Overuse of the arm may bring it on, as in the case of a girl whom I had under my care, who was continually turning sheets of paper for a press. Sensation may be impaired in the skin, and there is pain and rapid atrophy of the shoulder muscles. The shoulder-joint may become much relaxed, or false anchylosis may occur from contraction of the muscles, and in some cases adhesions about the joint take place.

Duration. An attack of neuritis may last from a few weeks to several months, and in some cases complete recovery in the function of the part may not be restored for years. The prognosis is said by some writers to be invariably good, but in many cases there is never restoration of the function of the nerve. In cases where the nerve has undergone injury from dislocation of the humerus, paralysis is prominent; and in some cases of facial neuritis the paralysis remains complete for years.

Treatment. The application of moist heat, such as fomentations and poultices, gives great relief to the pain and discomfort from an inflamed nerve. The part should be kept absolutely at rest for a few days, and counter-irritation, in the form of blisters or sinapisms, may be applied over the course of the nerve. In cases that are seen early the best method of treatment is the application of the ice-bag directly over the nerve. If the pain is intense and not relieved by local means, subcutaneous injections of morphine or cocaine may be employed. Small doses of salicylate of sodium and salol are useful in this condition, and the several coal-tar derivations, acetanilid, phenacetin, and antipyrin are useful in allaying the pain and formication. Galvanism is frequently of value in allaying the local discomfort. Passing a mild continuous galvanic current over the course of the nerve for a few minutes is usually followed by relief of pain for several hours.

The most important indication in the treatment of neuritis is to keep the affected part absolutely at rest. In a case of inflammation in the nerves of one of the extremities the limb should be placed on a well-padded splint, so as to insure absolute muscular repose. Turkish baths are sometimes of marked benefit, even where the neuritis is localized in a single nerve-trunk. In cases of gouty or rheumatic origin, a dose of blue mass, followed by saline purgatives and the free use of the alkaline waters, is indicated. In all cases particular attention should be paid to the general health, which is usually disturbed in some way. Alteratives and tonics are usually required. As soon as the hyperæsthesia and tenderness in the course of a nerve and other acute symptoms have subsided, the use of massage and electricity should be resorted to, in order to restore the functions of the nerve and muscles. Great care, however, must be observed in the application of these remedies. Faradization should, on no account, be employed as long as acute symptoms remain; and in the beginning of treatment the massage should be employed only by a person thoroughly skilled in the use of this agent.

MULTIPLE NEURITIS.

SYNONYMS. Polyneuritis; Peripheral Neuritis; Disseminated Neuritis.

The following classification of the different forms of multiple neuritis has been made by Ross:[1]

- I. Idiopathic form.
    1. Acute (Landry's paralysis).
    2. Subacute form.
    3. Chronic form.
- II. Toxic form.
    1. Diffusible stimulants: alcohol, carbon monoxide, bisulphide of carbon, dinitro-benzine, aniline.
    2. Animal poisons: diphtheria, typhoid and other fevers, septicæmia, syphilis, pneumonia, tubercle, malaria, beri-beri, leprosy.
    3. Metallic poisons: lead, arsenic, mercurial, phosphorus, and silver.
    4. Endogenous: rheumatism, gout, chorea, puerperal state, and diabetes.
- III. The dyscrasic form: chlorosis, marasmus, cancerous, and other forms of cachexia, vascular degeneration.
- IV. Sensory, vasomotor, and trophic neuritis.
    1. The neuritis found in ataxia (neuro-tabes peripherica).
    2. The vasomotor neurosis-erythromelalgia.
    3. Raynaud's disease.
- V. The irritative form of neuritis, in which spasm predominates over paralysis.
    1. Tetany.
    2. Professional hyperkineses.

Multiple neuritis is the term applied to the condition when many peripheral nerves are affected at once. It is a disease which has been recognized only during the past few years, although its existence was suspected by several writers. Dumesnil first described multiple neuritis in 1864; Joffroy made additional observations on the disease in 1879, which were confirmed by Leyden in 1880; and in 1881 Grainger Stewart wrote fully on the subject. It is only since the observations of the last-mentioned writer that general attention has been directed to the malady in this country. Since that time contributions to the subject have been almost innumerable.

Many years ago Graves indicated by his writings that he thought that many cases of paralysis might be due to diseases of the peripheral nerves; James Jackson, of Boston, described alcoholic paralysis in 1822, and Barwell, prior to 1870,[2] expressed the opinion that infantile paralysis was a lesion of the peripheral nerves.

Idiopathic multiple neuritis occurs at various ages, from infancy to old age, although the majority of cases occur in adult life. Among ninety cases collected by Ross most of the patients were attacked between the ages of twenty and forty years, but in one case the disease was said to have begun at two years, and another patient was sixty-seven when attacked.

I have recently seen a case of multiple neuritis in a lad of fifteen, in whom no exciting cause could be traced. The boy was a patient of Dr. Allison's, and was seized quite abruptly with general pain and slight loss of power in the extensor muscles of the limbs. There was tenderness over the nerve-trunks and muscles in the lower extremities. In three days there was complete loss of power in both arms and legs. The reflexes were all lost, but there was no impairment of sensibility. The patient states that he was unable to articulate distinctly on the day that the attack occurred, but the speech-disturbance was only transient.

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[1] "Peripheral Neuritis," James Ross, M.D., and Judson S. Bury, M.D., London, 1893.

[2] St. Thomas's Hospital Reports, N. S. vol. 1. p. 201.

There are more males than females the subjects of idiopathic multiple neuritis. In Ross's ninety cases, sixty-two were males and twenty-three females. It is likely that this is due to the fact that men are more exposed to the exciting causes of multiple neuritis than the other sex.

Acute polyneuritis may follow exposure to cold, or chilling of the body after being overheated, and it may come on spontaneously without any exciting cause being traceable. The onset is usually like that of an acute infectious disease. The attack is frequently ushered in with a chill, followed by pain in the back and limbs, with a rise of temperature to 103° or 104°. There is usually headache, loss of appetite and general malaise. Tingling and formication are felt in the feet and hands, and there is general muscular aching and tenderness. There is, at times, soreness throughout the entire limbs, the muscles and nerves being equally sensitive, so that it is not easy to distinctly define the nerve-trunks themselves.

Loss of power is manifested early in the disease. It usually begins in the flexors of the feet, and extends upward, as in Landry's paralysis. However, loss of power sometimes begins in the arms, and involves the legs secondarily. In severe cases paralysis is general in a few days, extending to the muscles of the trunk and the special muscles of respiration. The muscles atrophy rapidly and become soft and flabby. The deep reflexes are lost early, but the state of the skin reflexes vary, being preserved in some cases and lost in others. The electrical reactions of the muscles, however, do not undergo change except in severe cases and after the disease has lasted for some time. In the early stages the muscles respond to the faradic current, and this is a point to be borne in mind in the diagnosis between a peripheral neuritis and a spinal paralysis. The sensory disturbances vary in different cases; sometimes there are merely the subjective sensations of formication and numbness, with hyperæsthesia; but in other cases, in addition, there is a distinct anæsthesia. Sometimes the anæsthesia occurs only in circumscribed patches.

The disease has been divided into a motor and a sensory type, depending upon the most prominent symptoms of the attack. Ross believed that Landry's paralysis and acute multiple neuritis were one and the same disease; but other writers, like Hun, consider that they are two distinct diseases. The truth is, that in some cases of acute ascending paralysis the lesion is that of multiple neuritis, while in others there is cord disease. I have recorded a case of acute ascending paralysis[1] in which the patient died on the twelfth day after the attack. The symptoms corresponded very closely with those of an attack of acute multiple neuritis, but the autopsy showed distinct myelitis in the upper part of the cervical cord.

The course of the affection varies with the extent and intensity of the attack. A case may terminate fatally within a week from bulbar paralysis, or it may last five or six weeks before any material change takes place. The paralysis may remain for months or years, with atrophy and contraction of the muscles, but with gradual improvement as to muscular power.

ALCOHOLIC NEURITIS. This form of multiple neuritis differs materially in its symptoms from the idiopathic variety. It is the result of the excessive use of alcohol in any form. Persons who take small quantities of spirits frequently and steadily are more liable to alcoholic neuritis who drink to excess periodically. Drinkers of beer, howev affection, as well as those persons who drink gin and brandy constantly. Women suffer more frequently from alcoholic neuritis than men, there being three times as many cases in women as in men. This is probably because when a woman has once acquired the habit of taking alcoholic stimulants she is a more continuous "tippler" than is a man. Gowers suggests that there may be some difference in the nervous system of the two sexes which may explain why the peripheral nerves in women are more liable to alcoholic neuritis, and in men delirium tremens is more common.

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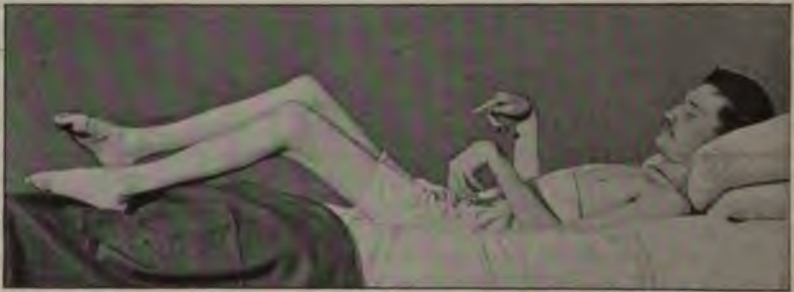
[1] Medical News, November 7, 1891.

The earlier symptoms of alcoholic neuritis are sensory disturbances, pain in the extremities, with sensations of pins and needles, and formication. There is a sense of numbness and blunted tactile sense, and sometimes there is hyperæsthesia. Soon motor symptoms begin, and the extensors of the feet and hands are primarily attacked, producing the characteristic foot-drop and wrist-drop. Foot-drop is as typical a symptom of alcoholic neuritis as wrist-drop is of lead palsy. Usually the lower extremities suffer first, but in some cases the extensors of the hands are affected first, and the paralysis may be confined to the upper extremities. In a case of alcoholic neuritis which was under my care, the first symptom which was noticed by the patient was weakness in the hands while driving. All of the limbs are not simultaneously affected, one leg may become almost completely powerless before there is much motor change in the other. With the extension of the disease there is increased tenderness over the nerve-trunks and muscles, and the sensory disturbances cause the patient great distress. In some cases the hyperæsthesia of the extremities, especially the soles of the feet, is so great that the patient is unable to bear the slightest touch; and the tenderness over the nerve-trunks and muscles and cutaneous hyperæsthesia is excessive. The muscular tenderness is a characteristic symptom of this disease. It is difficult to move the patient's limbs on account of the pain caused by every motion. In the onset of the attack the temperature is elevated, ranging from 101° to 103°, and there is tachycardia; the pulse being feeble and rapid, ranging from 100 to 150. The high temperature may last for four or five weeks. The capillary circulation is bad, the skin is of a dusky hue, and the finger-nails blue. Diarrhœa is not an unusual symptom, but there is no loss of control over the rectum or bladder. I have seen one case in which there were intestinal hemorrhages. In most cases there is a complication o some form of kidney disease. The paralysis may extend rapidly and is ofte complete in two weeks. The disease may terminate fatally in a few day but in most cases the progress of the disease is slow, and after an interval

FIG. 240.



Multiple neuritis. Double wrist-drop and double foot-drop. (LLOYD.)

several weeks recovery may begin. In fatal cases bulbar paralysis occurs, and paralysis of the diaphragm is always a grave symptom. The walk of a patient with alcoholic neuritis is characteristic. Owing to the foot-drop, he is obliged to lift his knees high, in order to allow the toes to clear the ground, producing the "high-steppage" gait. The tendon reflexes are lost early, but in many cases the skin reflexes are unchanged; occasionally the knee-jerk is preserved, but in those cases the quadriceps extensor has escaped paralysis.[1] The special senses seldom suffer. There is no disturbance of vision, hearing, or taste. Optic neuritis has been observed in a few cases, and Starr refers to two cases in which hearing was affected; but these exceptions merely prove the rarity of the implication of the nerves of the special senses. The facial muscles have been affected in some cases, but the muscles of deglutition are not involved except in fatal cases. The paralyzed muscles become soft, and atrophy very rapidly. The electrical reactions are changed. In some cases there are merely quantitative changes, a very strong faradic current being required to move the muscles. When the paralysis is complete, there is entire loss of muscular contractility to the faradic current, and a strong galvanic current is necessary to cause any reaction. Reaction of degeneration may not occur until late in the disease. Contractures are a common accompaniment of alcoholic neuritis; the claw-hand, and extreme extension of the foot from shortening of the tendo-Achillis are commonly met with. In most cases these deformities subside as recovery takes place, but in some cases they are permanent. Trophic changes are not often met with, but occasionally the skin of the extremities becomes red and glossy, and the nails are roughened; but bed-sores and ulcerations are rarely met with. Profuse sweating occurs in some instances, the limbs being bathed in perspiration.

Brain symptoms occur in most cases, but do not begin until the disease is well established. They may be present early in the attack, and it should be remembered that neuritis may begin during an attack of delirium tremens. A few cases of alcoholic multiple neuritis may escape brain involvement. The mental symptoms begin in the shape of hallucinations of various kinds; in some instances there are delusions of grandeur, but in other cases the patient is merely confused as to his surroundings and friends. He is seldom unhappy, but is apathetic except when disturbed. A common feature is for him to imagine himself in some strange place, and to believe that he is among strangers. The memory is greatly impaired, especially for recent events, and lapse of time seems to make no impression upon the patient. Insomnia is an almost constant symptom.

In some cases inco-ordination is a prominent feature of the disease, giving to it a close resemblance to locomotor ataxia. These cases have been called pseudo-tabes. Inco-ordination in gait, absence of knee-jerks, neuralgic pains, and "Romberg's symptoms" are points of similarity between the two diseases, but in multiple neuritis we find absence of the Argyll-Robertson pupil and muscular tenderness.

Duration. In mild cases the attacks last from a month to six weeks, but in severe cases which recover, the course of the disease may be one or two years, and complete restoration of motor power may not occur for years.

Gowers states that power returns first in the muscles affected last and least, but this is not always the case. Frequently there is a simultaneous return of power. In a case recently seen by me the liml [illegible] were affected successively: first the left leg became paralyzed, t[illegible] and afterward the arms. The right arm and left leg were most completely paralyzed. Recovery began in the left leg, which was the member first affected.

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[1] Maude and Clark have reported two cases in which the kne[illegible] a Medical Journal, Feb. 28, 1893.

Diagnosis. The history of alcoholism, with extreme sensory disturbance, and the extensor paralysis, together with the peculiar mental disorder, make but little difficulty in arriving at a diagnosis of alcoholic neuritis. In cases, however, in which, owing to the circumstances of the individual, one would not naturally suspect alcoholism, the diagnosis is sometimes not so easy. The groups of muscles affected and the peculiar mental phenomena should lead one to suspect alcoholic neuritis, and a careful search into the history of the case will then reveal the fact that the patient has been using alcohol in some form.

Prognosis. The prognosis in alcoholic neuritis is far more unfavorable than in other forms of this disorder. It depends, to a great extent, upon the age, the previous habits, and health of the patient. In cases in which extensive kidney, liver, or stomach disease is present, due to alcohol, the outlook is most unfavorable. In the class of patients brought into the public hospitals of large cities the mortality is great, but in private practice the patients usually recover. Death may occur within the first ten days from rapid involvement of the respiratory muscles, and in other cases the patient dies from pneumonia or pulmonary congestion. Paralysis of the vagus is another cause of death.

In cases which recover the convalescence is frequently slow, and it may be years before the muscular wasting disappears. In some cases the foot extensors remain permanently weak. The contractures may also remain throughout life, and the memory remains imperfect for a long time after the attack.

NEURITIS FROM CARBONIC OXIDE. In this country cases of multiple neuritis from carbonic oxide generally result from the inhalation of illuminating gas. It may occur, however, from being in a room in which charcoal or coal fire is burning, and to which oxygen has not sufficient access. In the early stages of carbonic-oxide poisoning there is a period of unconsciousness which generally, but not necessarily, precedes the symptoms of neuritis. There may be pain in the hypochondriac regions, and dyspnœa. Temporary mental symptoms are often present, such as depression and loss of memory, and the latter may persist for several months.

The symptoms of neuritis from carbonic-oxide poisoning are numbness, impairment of tactile sensibility, and pain in the extremities, usually over the affected muscles. There are local palsies which are irregular and variable in distribution, differing in this respect from other forms of neuritis. Occasionally there is wrist-drop and foot-drop, but in some cases the paralysis may be hemiplegic and in others paraplegic. Vasomotor and trophic changes are common. There is frequently redness of the skin, with œdema, and herpetic eruptions over the course of the nerve-trunks are common.

Multiple neuritis may also arise from other forms of diffusible poisons, such as aniline and dinitro-benzene, but the symptoms are not materially different from those already described.

MULTIPLE NEURITIS FROM LEAD. General peripheral neuritis may be due to lead-poisoning. The symptoms are those of polyneuritis, and the most typical features are those due to the local paralysis of the extensors of the wrists and feet. The deltoid and biceps muscles are more frequently involved than in other forms of neuritis. A characteristic feature, however, is the fact that in lead multiple neuritis the supinator longus and extensor ossis metacarpi pollicis usually escape paralysis. No one, however, has yet suggested a reasonable explanation of this fact. The sensory symptoms are either slight or entirely wanting.

Two types of lead neuritis are met with: one in which the affection resembles paralysis from injury to the musculo-spiral nerve, and in which the lesion is confined to the peripheral nerves; and in the other resembles progressive muscular atrophy in the extensive and general muscular wasting which occurs. In the latter form there is involvement of the ganglion cells in the anterior horns of the cord.

In lead neuritis there is sometimes pain and slight tenderness in the muscles and nerve-trunks, and occasionally some anæsthesia over the affected muscles. The pains, however, are dull and rather deep seated. In some cases there is a considerable degree of tremor present. The tendon reflexes are lost. There is no bladder paralysis, but the bowels may be obstinately constipated.

Diagnosis. The presence of a blue line upon the gums, the extreme anæmia and history of colic point to lead-poisoning. The sensory symptoms are less marked than in alcoholic neuritis, and the brain symptoms when present are different in character from those of the latter affection. The symptoms of lead encephalopathy are accompanied with stupor as well as delirium. Lead neuritis is to be distinguished from progressive muscular atrophy by the fact that the electrical changes are more marked, and by the absence of the knee-jerk, which is usually preserved in progressive muscular atrophy as long as there is a muscular fibre remaining. Furthermore, the history of the case is different.

ARSENICAL NEURITIS. Arsenical neuritis occurs from the absorption of arsenic into the system in various ways. The drug may be taken with suicidal intent, or it may be administered with the food with the view of poisoning. A patient whom I saw some years ago had been given quite large quantities of Paris green in his food. He had, as a consequence, extensive motor and sensory paralysis.

Multiple neuritis sometimes follows the medicinal administration of arsenic, and cases have been reported by Potts, Barrs,[1] and Osler.[2] Putnam has reported a most instructive case of multiple neuritis, in which arsenic was recovered from the urine. The cause of the attack was the administration of Fowler's solution in doses of five drops three times a day for six weeks.[3]

The symptoms of arsenical neuritis resemble those of lead and alcoholic neuritis to a considerable extent. The sensory symptoms are more marked, however, than in the former, and the mental symptoms characteristic of alcoholic neuritis are seldom present in arsenical neuritis. The pains in the limbs are of a shooting and darting character, and anæsthesia is frequently present. Skin eruptions and trophic changes are often met with. In doubtful cases an examination of the urine will reveal the presence of arsenic.

DIPHTHERITIC NEURITIS. The paralysis begins commonly in the palate, and is shown by changes in the voice and difficulty of deglutition, and liquids may regurgitate through the nose. In many cases the paralysis is confined to this part, but usually after a few days it extends to the muscles of the eyes, and the ciliary muscle is especially liable to be involved, giving rise to loss of power of accommodation and impaired vision. The arms are affected next in order of frequency, and then the legs. Of one hundred and seventy-one cases of diphtheritic neuritis collected by Ross,[4] the palate was the part first paralyzed in one hundred and twenty-eight; in seventy-seven cases the ocular muscles were affected; in sixty cases paralysis occurred in the arms; and in thirteen in the legs. Sensation was disordered in eighty-six of the one hundred and seventy-one cases.

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[1] British Med. Journ, February 4, 1893.

[2] Johns Hopkins Bulletin, April, 1893.

[3] Boston Med. and Surg. Journ., September, 1893.

[4] Med. Chronicle, December, 1890.

The paralysis is seldom complete, and is almost invariably bilateral. The muscles become flabby and relaxed at an early period. The knee-jerks are lost early; in fact, in some cases there is absence of knee-jerk even where no muscular paralysis can be detected. In severe cases the pneumogastric nerve is affected, giving rise to weak cardiac action, and in some cases sudden death occurs from paralysis of the heart. In diphtheritic neuritis sensory symptoms are not conspicuous, but there may be some numbness and slight hyperæsthesia or anæsthesia over the paralyzed muscles.

Diagnosis. The diagnosis usually depends upon the distribution of the paralysis to the throat muscles and the history of diphtheria. There are cases, however, in which the local symptoms have been so slight that they have not been observed, and the fact of an attack of diphtheria has been recognized through the paralysis.

Diphtheritic neuritis in children may be mistaken for poliomyelitis. It differs from the latter in the fact of the slower onset, the symmetry of paralysis, and the slighter degree of the loss of power and atrophy. In diphtheritic neuritis the electrical reactions undergo but slight quantitative changes. The presence of sensory symptoms also distinguishes the disease from poliomyelitis.

MALARIAL NEURITIS. Strachan[1] has described a form of neuritis which he met with in Jamaica, and which he ascribed to malarial poisoning. According to this observer, the muscles of the trunk and limbs and cranial nerves are equally involved. There is much pain in the extremities, and muscular wasting is extreme. Vesicular and other cutaneous eruptions are common, and the pigmentation of the skin is increased. The knee-jerks were absent in one-half of the cases reported by Strachan, but the cutaneous reflexes were variable. Sensation was but slightly impaired.

In a case of malarial multiple neuritis which came under my care, the patient, a young woman, about thirty years of age, had lived for the greater part of her life in a climate saturated with malaria, and had had frequent attacks of remittent and intermittent fever. When she came under my observation there was marked paresis of the extensors of the feet as well as the extensors of the wrists and arms. The paralysis was not complete, and the knee-jerks, although diminished, were not entirely absent. There was a considerable amount of paralysis of the muscles of deglutition, causing much trouble in swallowing food, and frequent regurgitations of liquids through the nose, and speech was imperfect from labial paresis. There was but little sensory disturbance. There was no anæsthesia or tenderness over the nerve-trunks, but a sense of numbness was complained of. The patient eventually died from bulbar paralysis.

TYPHOID NEURITIS. Neuritis not infrequently occurs in connection with typhoid fever. It may come on during the fever, or is met with as a sequela. It is probable that many of the cases of paralysis from typhoid fever which have been reported in years past have been cases of neuritis. In a case of neuritis under my care which occurred during an attack of typhoid fever, the atrophy of the legs was extreme, and the contractions of the hamstring muscles were so great that a prolonged and careful course of massage and other means failed to relax the contractures. Tenotomy of both outer and inner hamstring muscles was done, and the patient entirely recovered the use of her limbs.

Neuritis follows many other acute diseases, among which are smallpox, grippe, scarlet fever, pneumonia, pleurisy, and erysipelas. Gray[2] has put on record a case of multiple neuritis following varicella in a child two years and five months old. There was paralysis of motion and sensation in both legs, but the patient recovered completely.

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[1] Annual of the Universal Medical Sciences, 1888, p. 139.

[2] Brit. Med. Journ., March 31, 1894.

SYPHILITIC NEURITIS. Syphilitic neuritis usually occurs in a single nerve, and when syphilitic multiple neuritis is met with it is commoner in connection with locomotor ataxia, the so-called "peripheral neuro-tabes."

TUBERCULAR MULTIPLE NEURITIS. Tubercular multiple neuritis is of occasional occurrence in phthisis. I have seen cases of well-marked neuritis in tubercular patients in the Philadelphia Hospital. In one patient there was also a history of extreme alcoholism, so that it was a question as to whether the neuritis was not due to this cause rather than to the tuberculosis from which he was suffering.

The facts seem to indicate that the tubercle bacillus is capable of producing a poison which has a specific toxic action upon the nerves. Gowers suggests that neuritis of branches of the vagus nerve may give rise to a low form of pneumonia, which makes the bacilli more effective if they enter the lungs.

RHEUMATIC MULTIPLE NEURITIS. Neuritis is met with as a complication of acute rheumatism, and is probably the result of a toxic infection similar to the poison which produces rheumatism. In rheumatoid arthritis there is almost always more or less evidence of peripheral neuritis, as shown in the great degree of muscular wasting, glossy skin, and tapering fingers which are typical of neuritis; in fact, it seems probable that many cases which have been called rheumatoid arthritis are, in reality, a form of peripheral neuritis.

PERIPHERAL NEURITIS IN THE AGED, which has been recognized as a special form of the affection, is probably due to arterio-sclerosis. Oppenheim has recently written fully on the subject of senile multiple neuritis, and has pointed out some special features which characterize it. Among the indi-dividual characteristics of this form of neuritis are the incomplete development of motor and sensory symptoms, the integrity of the cranial nerves, and the tendency to recurrence.

SEPTICÆMIC MULTIPLE NEURITIS is the result of absorption into the system of septic material from wounds, abscesses, or other lesions. The knowledge of the occurrence of neuritis from these causes explains many of the conditions which are met with following suppurating wounds. In a case recently seen by me, the patient received a slight wound of the hand from the shell of a lobster which he was cleaning. The wound healed without trouble, but was followed by pain and paræsthesia, with swelling of the hand, stiff joints, and glossy skin.

DIABETIC NEURITIS. Multiple neuritis occurs during the course of diabetes. The attack of neuritis does not appear to depend upon the amount of sugar in the urine, as cases are met with in which the percentage of sugar is small. It is supposed to be due to the influence upon the peripheral nerves of toxic materials which are analogous to acetone.

ENDEMIC NEURITIS, BERI-BERI, KAK-KÉ. This disease is an infectious and contagious form of neuritis due probably to a micro-organism, and is met with in epidemics in Japan, China, parts of India, Ceylon, the Philippine Islands, the coast of Brazil, and other localities. Diet appears to have a considerable influence in the production of the disease, although this has been denied by many writers. Putnam has reported a number of cases among the fishermen on the banks of Newfoundland, and the disease seemed to have developed in consequence of unsuitable and insufficient food.

The disease has also occurred among sailors on vessels carrying cargoes of sugar, and in these cases the predisposing cause was supposed to be the carbonic acid gas and toxic matters arising from the fermentation of the large quantity of sugar in the hold of the ship.

Food which is insufficient or lacking in albumin is considered a cause of the disease, and the epidemics in China and Japan have been ascribed to the fact that rice is a staple article of food in those countries. Takaki,[1] a Japanese physician, states that he has succeeded in reducing the number of cases in the Japanese marine service to an enormous extent, simply by adopting a food regimen in which beef, pork, eggs, etc., are given in addition to rice. In 1884 there were 713 cases, and in 1889 there had not occurred a single case.

It has also been attributed to an intestinal parasite.

Dr. James H. Walker[2] has related a summary of the results of 927 cases of beri-beri treated during the last five years at Sandakan (British North Borneo). Out of this number, 887 patients were examined with reference to the presence of intestinal parasites in the dejecta. The results were as follows:

The ankylostomum duodenale was found in 756 cases, or 85.5 per cent.; the trichocephalus in 284, or 31.5 per cent; the ascaris lumbricoides in 155, or 17.4 per cent.; the oxyurus vermicularis in 123, or 13.8 per cent.; the distoma in 2, or $\frac{1}{5}$ per cent.; various parasites in 24, or 2.6 per cent.; an ascaris in 3, or $\frac{3}{10}$ per cent.

The ankylostomum, generally speaking, is not very frequent among the indigenous population. Its predominance in beri-beri must, it seems to me, be of some significance. Dr. Walker admits, for several reasons, that this parasite cannot be regarded as the specific pathogenic agent of beri-beri, but thinks it not impossible that it may be a predisposing cause of this affection; at any rate, it is an aggravating factor. In his opinion, the causes of both diseases—beri-beri and ankylostomiasis—are produced simultaneously outside the organism, the conditions favoring the development of the ankylostomum (uncleanliness and stagnant water) being also eminently favorable to the evolution of the germ of beri-beri.

Careful researches on the etiology of beri-beri leave no doubt of the propagation of this disease by water, a fact which makes the boiling and filtering of drinking-water a necessity in regions where beri-beri prevails. With regard to treatment, the first thing to do is to examine the stools of all patients suffering from beri-beri, in order to institute measures for the expulsion of the entozoa which may be present.

Symptoms. The symptoms are the same as those of other forms of multiple neuritis, with the addition of œdema and a tendency to effusion into the serous cavities. There is also greater liability to cardiac disturbance.

Cases of beri-beri are mild or severe in character. In the mild cases the onset is gradual, and the patient complains of weakness in the legs, so that he cannot walk as much as usual. Soon numbness and pain occur. There is palpitation, epigastric oppression, loss of appetite, and general ill feeling. Œdema of the extremities occurs, and the pulse is irregular and dicrotic. The anæsthesia is slight and irregularly distributed. In these cases, under appropriate treatment, recovery takes place in a few days, or occasionally the convalescence may be slower.

The severe cases have been divided into three types: The atrophic or dry type, in which the attack begins like a mild case, but the intensity of the symptoms increase with greater rapidity, paralysis becomes complete and extends to almost all of the muscles of the body, including the face. Muscular wasting is excessive, and the pain and paræsthesia are intolerable; in fact, the pain in the extremities is a prominent feature of the disease. The anæsthesia is not complete. There are some trophic changes, such as glossy skin, but in this form there are no gastric symptoms and no œdema. The attack may terminate fatally, but most cases recover after prolonged convalescence.

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[1] Brit. Med. Journ., Sept. 24, 1892.

[2] Medical Week, September 21, 1894.

The second form is the hydropic, or wet type. In this form œdema occurs early, and the entire body is swollen with the subcutaneous effusion. There is also effusion into the cavities of the body. The atrophy in the muscles is masked by the œdema, and is as great as in other forms.

There is also a pernicious type of the affection, in which the symptoms of the other forms are present, but occur in more rapid succession, and, in addition, gastric symptoms are prominent. There may be also suppression of urine. The heart is weak and irregular, and death usually results from paralysis of this organ.

LEPROUS NEURITIS. This is met with in cases of leprosy, the so-called anæsthetic leprosy, and resembles beri-beri in its infectious nature, although differing from it in the fact that the specific bacillus of leprous neuritis acts directly upon the nerve-tissue instead of being found in the blood. Leprous neuritis differs from other forms of neuritis in the irregular distribution of the anæsthesia and in the discoloration of the skin in dark maculæ. The disease is gradual in its invasion, but occasionally the anæsthesia is developed suddenly without previous evidences of leprosy. Muscular weakness and wasting are not conspicuous features.

The form of neuritis met with is perineuritis and interstitial neuritis. When the ultimate fibres themselves are diseased it is of secondary occurrence. This is another point of distinction from other forms of neuritis.

In recent cases the characteristic bacilli are found in the interstitial connective tissue of the nerves in large numbers, but with the increasing development of the fibrous tissue in the nerve-bundles the bacilli disappear.

Leprous anæsthesia may be mistaken for syringomyelia, but in the latter affection there is generally involvement of the legs, with exaggerated knee-jerks, and the dissociation of the thermal sense is a characteristic feature of syringomelia. Some cases of Morvan's disease have proved to be leprous neuritis.

Pathology. The pathological changes in multiple neuritis are much like those in simple neuritis. The peripheral branches of the nerves are involved throughout, and degenerative changes in them are much greater than in the nerve-trunks proper. The latter are usually affected in but a limited degree, and it frequently happens that the fibres in the nerve-trunks show no evidences of disease, even in cases in which there has been tenderness over the nerves. The neuritis is of the parenchymatous type, showing all the features of Wallerian degeneration similar to that met with after injury of a nerve. In recent cases the nerve may appear red and swollen to the naked eye, and in cases of long standing the nerve is soft and may be even pulpy. In some cases the connective tissue appears principally involved, and then the sheaths of Schwann are found under the microscope to be infiltrated with leucocytes. In the septa, between the fasciculi and in the secondary sheaths surrounding these, lymphoid cells are also found in large numbers. After a time regeneration begins in the nerve-fibres, and the process is the same as that which occurs after neuritis from injury. The period of improvement in a case begins with the regeneration.

In the muscles the same changes occur as in simple neuritis. The muscular fibres are reduced in bulk and may lose their striat[illegible] and become granular, and in bad cases fatty degeneration occurs.

in the ganglion cells of the cord are met with. In alcoholic neuritis changes in the cord are quite frequent. Chronic myelitis in irregular areas is the principal lesion, and sometimes chronic inflammation of the spinal meninges has been found. Various other organs are also found diseased, more especially the liver and kidneys. The liver is enlarged and cirrhotic or, it may be, fatty; the kidneys are large and white, or are contracted. Phthisical changes are frequently met with in the lungs, the heart is usually flabby, and the muscular fibres have undergone degeneration.

Diagnosis. The history of the case and the combination of motor symptoms which have been preceded by decided sensory disturbances make it almost necessary to arrive at a diagnosis of multiple neuritis. The tenderness of the muscles and nerve-trunks is also an important diagnostic feature in the disease. The diseases most likely to be confounded with multiple neuritis are anterior poliomyelitis and locomotor ataxia. The differential diagnosis of multiple neuritis from the latter has been considered under the head of Alcoholic Neuritis. The following summary from Starr[1] shows the prominent differences between anterior poliomyelitis and multiple neuritis:

Anterior Poliomyelitis.	*Multiple Neuritis.*
Sudden onset with fever and development of paralysis in all limbs, followed in from three to five days by subsidence of paralysis, which remains in a few muscles of one limb; or, if two are affected, the paralysis is very rarely symmetrical. If onset is subacute, four weeks is the duration of onset. Muscles not tender.	Fatigue for some weeks, then sudden onset and progress for two weeks with or without fever. Legs first affected, then arms, then body, and paralysis has no tendency to subside for a month; limbs are affected symmetrically, and the muscles affected are very tender.
Sensory symptoms are rare, and when present soon subside.	Sensory symptoms are constant and severe, and increase; anæsthesia becoming well developed.

Acute ascending myelitis resembles multiple neuritis in some respects, but cases of this kind are uncommon. In myelitis the loss of sensation is complete, and involves the trunk as well as the extremities, beginning at a well-defined line around the body at the level of the lesion in the cord. Girdle-pains are common, and bed-sores and incontinence of urine are also constant symptoms of myelitis. In myelitis there is no tenderness of the muscles and nerve-trunks. Occasionally hysterical cases are seen with symptoms which simulate those of multiple neuritis. The distribution of the symptoms, however, is irregular, and there is no loss of the reflexes. If there is any change at all in these, they are exaggerated.

Prognosis. This depends largely upon the general condition of the patient, whether he is broken down by dissipation or disease, or if he is of good constitution. The acuteness of the attack also increases the risk of death. If there is a progressive increase in all of the symptoms for two or three weeks, the case is unfavorable; but cases of alcoholic neuritis are seen in which the symptoms grow progressively worse for five or six weeks, and yet eventually recover. When the muscles of the trunk are enfeebled, in addition to paralysis of the extensors of the hands and feet, the case is more grave, because then respiratory difficulties are liable to arise. Paralysis of the diaphragm has been mentioned already as an unfavorable symptom, and one should be on the lookout for diaphragmatic paralysis, which may come on unexpectedly. When the disease has become stationary, that is, when there is no further increase in the degree and extension of paralysis, then the danger of life is lessened and recovery becomes probable. In cases in which there is evidence of involvement of the spinal cord, as shown by bed-sores, paralysis of the bladder, etc., the prognosis is more serious. The prognosis is most serious in cases which are dependent upon alcohol or some toxæmic state of the blood, and in acute cases resembling Landry's paralysis.

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[1] "Familiar Forms of Nervous Disease," page 216.

In cases which tend to recovery the full development of the symptoms is followed either by improvement or a stationary period which may last for one or two months. The course of convalescence is slow, and muscles which have been only partially affected take from three to four months to recover their power. The sensory symptoms improve more rapidly. There is an impression prevalent that in cases which do not terminate fatally there is in the course of time complete restoration of power, but this is not always the case. In a patient now at the Philadelphia Hospital there was extensive paralysis from alcoholic neuritis, followed by great atrophy and contractures of the flexors of the feet and hands. These contractures still remain, although the acute attack occurred five years ago.

In some cases, especially those of alcoholic origin, death may occur within a few days from heart failure or congestion of the lungs, due to involvement of the vagus. If the patient has vitality enough to live until regeneration of the nerves begins, he will probably recover. In young subjects recovery is the rule, and in cases of moderate intensity the restoration of power is generally complete.

Treatment. In the treatment of multiple neuritis the first consideration is to eliminate the cause of the disease, if possible. In alcoholic and toxæmic cases this is, of course, clearly the indication. It is frequently exceedingly difficult to prevent alcoholic patients from taking stimulants, and it requires the utmost watchfulness on the part of the nurse and the physician to prevent liquor being furnished them. It is important in alcoholic neuritis to stop stimulants entirely, but it is necessary to do this with caution, as many patients are in such a condition of weakness that the alcohol cannot be completely withdrawn without risk of heart failure and general collapse. In these cases strychnine, together with strophanthus or digitalis, is of great importance. When neuritis can be traced to lead, arsenic, or other metallic poisoning, means must be taken for the elimination of these—*i. e.*, the administration of iodides, baths, etc. (See p. 196 *et seq.*)

Absolute rest in bed is of the first and utmost importance in all forms of neuritis. The treatment can be more readily carried out, especially in regard to applications to the painful and sensitive limbs, and the patient's strength is economized. One of the earliest indications requiring treatment is the pains in the extremities. Hot fomentations and poultices applied over the tender nerves and muscles often afford relief, but care must be exercised that the applications are not made too hot, as the skin readily blisters and troublesome sores may remain. Warm baths often give great relief to these pains.

The salicylates have been recommended for the relief of the acute symptoms, but their depressing influences should be remembered and counteracted if necessary by the administration of digitalis or other cardiac stimulant. The coal-tar derivatives, such as acetanilid, antipyrin, and phenacetin, are also useful in allaying pain, but must always be used with caution on account of the feeble heart. The compound tincture of cinchona or the tincture of gentian may be used with advantage in cases of alcoholic neuritis, where it is not advisable to withdraw the stimulants altogether. In these cases cocaine in doses of one-eighth to one-quarter of a grain may be given. It acts as a stimulant and as an analgesic as well. It may be given hypodermatically for the nerve-pains. Strychnine is indicated in all forms of neuritis, and the nitrate of strychnine seems specially applicable in cases of alcoholic neuritis, on account of its alleged power of reducing the craving for stimulants. Cannabis indica also may be given continuously to relieve pain, but if this and the remedies mentioned above fail, morphine should be given hypodermatically. If the patient is wakeful, one of the bromides or chloralamid may be given, but if there is much excitement a hypodermatic injection of hyoscin hydrobromate, one-one-hundredth of a grain, is preferable.

Massage is of great importance, and may be employed from the beginning of the attack if a skilful masseur can be obtained. The movements should be made with the utmost gentleness, and effleurage is the form of massage to be selected. When the acute symptoms are subsiding deeper massage may be employed, and thorough kneading of the muscles may be resorted to.

Electricity should not be used early in the disease, except in the form of the galvanic current passed continuously down the limbs, with the view of allaying pain. When the active progress of the disease is arrested the faradic current may be used to stimulate muscular contractions and prevent wasting.

Care should be taken to prevent contractions of the muscles during the course of the disease. It will be found necessary to use frames to keep the bed-clothes from resting upon the limbs of the patient, as even the weight of a sheet often causes suffering, and sand-bags and other appliances should be used to keep the limbs in proper position. The tendency to foot-drop, which occurs almost universally, must be overcome by pads and other appliances, and particular care is necessary to prevent contraction of the hamstring muscles, as there is a natural tendency of the patient to lie with his knees flexed. The painful extremities may be wrapped in absorbent cotton with advantage, and sometimes a covering of oiled silk to retain heat and moisture adds to the patient's comfort.

Iodide of potassium may be given during the stage of convalescence, and arsenic is also useful as a nerve tonic. The latter is a remedy, however, which should be given with caution, as it is known to have produced neuritis even in small doses. Tonics are indicated, and cod-liver oil, when it can be borne by the stomach, is one of the most useful tonics which can be administered.

The diet of the patient demands especial attention. During the acute stages light and easily assimilated food, like milk, oysters, fish, meat broths, and eggs, should be given freely. The patient often has an abnormal appetite, and it becomes necessary to see that no indiscretions in diet are committed.

ERYTHROMELALGIA.

This affection was first accurately described by Dr. S. Weir Mitchell[1] in 1878, and since then many cases have been recorded by other writers. It is a disease which affects the feet principally, and is distinguished by intense burning pains and redness of the parts affected. The disease usually attacks men, but may be met with in women as well. It begins in the ball of the foot or heel, and the pain comes on when the patient attempts to walk, or when the foot is hanging down. The affected part becomes swollen and intensely red, of a bright scarlet hue. The disease is usually symmetrical, but one leg alone may be involved. The pain at first is of an aching kind, but afterward it assumes an intensely burning character, which is increased by warmth, and relieved by the application of cold or by the recumbent position. The flushing of the painful areas is the most characteristic feature. The feet, according to Dr. Mitchell's original description, "get redder and redder, the veins stand out in a few minutes as if a ligature had been tied about the limb, and the arteries throb violently for a time, until at length the extremities become of a dark purplish tint." The parts involved may perspire profusely. In the worst cases, when the patient is at rest, the limbs are cold and even pale. The disease increases until finally all of the branches of the plantar nerve are involved. The pain is generally worse at night, and is much increased by exercise of any kind. The feet become so tender that standing or walking is excessively painful, in some cases the patient is unable to stand upon his feet, and one of Dr. Mitchell's patients, whom I saw, was compelled to go on his knees when he wished to go across the room. The hands also may be affected.[1] Blisters and ulcerations are liable to arise from slight injuries. All of the symptoms become worse in warm weather.

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[1] American Journal of the Medical Sciences, July, 1878.

In some cases the disease is progressive and may be associated with some evidences of spinal disease, such as girdle pains, partial paralysis, and atrophy of some of the leg muscles. In the case described by Ross[2] there was a tender spot in the centre of the heel, and the whole course of the external plantar nerves was tender to pressure. The feet were bathed in a sour-smelling sweat, and the skin of the sole had a sodden appearance, becoming somewhat glazed during the paroxysm of pain and redness.

Pathology. The disease was considered by Mitchell in his original paper as a vasomotor neurosis, but it is, no doubt, a peripheral neuritis involving the branches of the plantar nerve.

Diagnosis. The diagnosis is to be made from plantar neuralgia or podalgia and alcoholic neuritis. In podalgia there is no redness or swelling, and in alcoholic neuritis the tendereess of the feet is not accompanied by redness, and there are also present other peculiar symptoms of the disease.

Treatment. Elevation of the feet gives temporary relief. Electricity and massage have been used, and in some cases with benefit. Prolonged rest in bed, with general tonic treatment, has relieved the disease in some cases. Dr. Weir Mitchell has recently suggested excision of the posterior tibial nerve as a means of relief, and in one case at least the operation has been entirely successful.

TUMORS OF NERVES.

Hypertrophy of nerve-trunks is rarely met with. In anæsthetic leprosy there is found an increase in the size of the nerve-trunks due to hyperplasia of the connective tissue. The enlargement of the nerves in this disease is a cirrhotic enlargement rather than a hypertrophy.

NEUROMA. This term is applied to a tumor growing in or upon a nerve-trunk, Neuromata have been divided into true and false. True neuromata are composed of nerve-tissue, and are met with almost exclusively upon spinal nerves. They consist of medullary nerve-fibres in some cases, and in others of fibres of the non-medullary variety, the axis-cylinders existing in increased numbers. A neuroma may also consist of true ganglion cells, with a surrounding network of fibres. In most neuromata the nerve-tubes are not continuous with those of the nerve-trunk, but are found in an irregular network mixed up with loose connective tissue.

Neuromata are met with either singly or in considerable numbers. They vary in size from 1 c.cm. to 6 c.cm. in diameter. The button-like growth at the end of a nerve in the stump of a limb which has been amputated is a good example of a neuroma.

Multiple neuromata are generally neuro-fibromatous in composition. They are frequently very numerous, many thousands having been found in a single case. They exist under the skin and form painful nodular bodies. Duhring[1] has described a case in which a great number of neuro-fibromata were found scattered over the entire surface of the body. Some of these were excised and found to consist principally of non-medullated nerve-fibres, with connective tissue interwoven among them. The pain which the patient suffered was excessive.

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[1] Gerhardt (Annual Med. Sciences, vol. ii. C. 58) reports a case in a needlewoman of forty-four years who suffered from erythromelalgia in the hands as well as the feet. There were swelling and redness in the affected parts.

[2] Diseases of the Nervous System, vol. i. p. 518.

False neuromata are tumors of nerves containing no nerve fibres or nerve cells, such as fibromata, gliomata, myxomata, carcinomata, sarcomata, and syphilomata. These growths are generally secondary. The most common form of false neuroma is carcinoma. The small neuromata, which are found situated just under the skin on the ends of the sensory nerves, are known as tubercula dolorosa.

Etiology. The causes of neuromata are frequently obscure, the principal being some hereditary predisposition which tends to the production of the multiple variety. Injuries and surgical operations are common causes of neuromata. This is more particularly the case in stumps, where there is almost invariably found a neuroma at the end of the divided nerve. The tubercular and cancerous diatheses have also an influence in the production of false neuromata. Multiple neuromata may be developed early in life or may be congenital.

Symptoms. In many cases of neuromata, no symptoms are present, especially in the case of multiple neuromata. The latter are sometimes not discovered until after the death of the patient. Frequently, however, there is excessive pain as the result of the nerve-growths. The character of the pain is generally acute and burning, and is usually referred to the distribution of the nerve. The neuromata themselves are tender on pressure. When the nerve-fibres suffer to any great extent there are paræsthesia and other disturbances of sensation. Occasionally paralysis of the muscles supplied by the nerve occurs, but this is only where the nerve-trunk is compressed by the tumor.

In the case of neuromata in stumps there is often excessive tenderness, which may prevent the use of an artificial limb. There is also occasionally some reflex disturbance as the result of these growths. I recall a case seen many years ago, in which the neuroma in a stump of an arm, which had been amputated many years before, gave rise to intense neuralgia, which was relieved as soon as the growth had been removed.

Neuromata grow with greater or less rapidity, but, as a rule, the symptoms come on gradually and continue for a length of time.

Diagnosis. The diagnosis of neuromata can only be made when they are in superficial positions. The symptoms to which they give rise may merely lead one to suspect the presence of these growths. In the case of subcutaneous multiple neuromata they are to be distinguished from fibromata and carcinomata by the difference in their size. Neuromata are always quite small, seldom exceeding an inch in diameter at the utmost. False neuromata are usually secondary, and depend on the existence of some other morbid growths.

Treatment. The treatment of nerve-tumors is almost purely surgical. Except in the syphilitic varieties, medical treatment is of no value. Extirpation is easily effected if the growth is superficial, and involves little or no risk. If the whole nerve-trunk is infiltrated by the growth, the affected part must be excised and the ends brought together. The risk of loss of function in the nerve after this operation is comparatively small with the antiseptic precautions which are now taken in surgery. There is a tendency to relapse, however, in the case of neuromata; and in multiple neuromata surgical treatment is almost hopeless on account of the continual recurrence of new growths.

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[1] American Journal of the Medical Sciences, vol. lxxiii. p. 413.

When there is a painful neuroma in a stump the nerve-tumor is easily removed by operation, and recurrence is not usual. In cases in which "amputation neuromata" cause great pain and reflex spasms, nerve-stretching has been found to give relief when excision has not been successful. Cocaine, hypodermatically, also gives temporary relief, although the danger of the cocaine habit is great. In many cases of amputation neuromata, however, the paroxysms of pain usually occur with the change of the weather and the approach of storms, so that palliatives, like cocaine and morphine, may be used with less risk of a drug-habit being formed.

MECHANICAL INJURIES OF NERVES.

Nerves may be injured in a variety of ways, either from blows or contusions, which do not divide the overlying skin, and they may also be severed by cuts or injuries which lay open the tissues above them. The results of contusions and compressions are more or less laceration and inflammation at the seat of the injury, together with neuritis extending upward from this point. The symptoms connected with this form of injury have been described under the head of neuritis.

Open wounds may be caused by cutting instruments or by bullets, giving rise to different symptoms dependent upon the extent of the lesion. The nerve-trunk may be either completely severed or only partially divided, or, possibly, injured by being merely grazed by a ball. The most common causes in everyday life of incised wounds of nerves are from glass, either through the accidental thrusting of the hand through a window-pane, or by bottles which break while being filled or cleaned. Injuries are not uncommon in bottling establishments, where a bottle often bursts from the pressure of carbonic-acid gas while being corked. Knife wounds from accident and from stabs also occur quite frequently.

Wounds of the sciatic nerve may be inflicted by tools or sharp instruments, on which a person may fall or sit. I have seen one case of division of the sciatic nerve in the case of a ship-carpenter, who fell upon an adz, and cases have been reported in which the sciatic was divided by a fall upon a scythe.

The nerves which are most frequently injured are those which lie superficially in the upper extremities. The musculo-spiral nerve, from the position in which it lies as it turns around the humerus just above the elbow, is quite frequently the seat of injuries. I have seen two men who received stabs in this position which completely divided the nerve. It is probable that the arm was thrown up to ward off the blow, and it was, therefore, struck at this point. In one case the wound must have been made by a pocket-knife with a narrow blade, as the scar at the wound of entrance was not more than three-eighths of an inch in length, but the nerve was completely divided.

The median nerve is more liable to injury than any other nerve from its exposed position at the wrist, and in this situation it is frequently severed by cuts inflicted by sharp weapons or by glass. I know of an instance of a patient who, during an attack of melancholia, attempted suicide by cutting herself across the wrist. The median nerve was divided by the incision. The ulnar nerve is also occasionally injured at the inner condyle of the humerus.

The extent to which a nerve has been injured can only be determined by careful examination of the parts to which the nerve is distributed, in order to determine the loss of motion and sensation.

Puncture wounds of nerves are rare, but those cases which have been reported, as, for example, when a nerve has been punctured by a hypodermatic needle, or when in the operation of phlebotomy the nerve has been accidentally struck, inflammation of the nerve is likely to follow, accompanied by the usual symptoms of pain, paræsthesia, etc., and possibly by reflex disturbances. Mitchell[1] mentions the case of a man who had driven an awl through the ulnar nerve. In this patient there was excruciating pain, followed by choreic twitchings and finally by spasm in the flexors of the fingers.

During the late war a large number of gunshot wounds of nerves were treated in the United States Army Hospital at Philadelphia for Injuries and Diseases of the Nervous System, and the classical treatise on the subject by Mitchell, Morehouse, and Keen, which was published in 1864, has been a source of information since that time.

A nerve-trunk may be completely severed by a bullet, or it may be partly divided, or only contused. The results and symptoms of gunshot wounds differ little from injuries inflicted in other ways. The immediate effect, however, of a gunshot wound through a nerve-trunk is rather different from injuries received from blows and cuts. According to Mitchell,[2] the first effect of a gunshot wound of a nerve varies in different individuals; in some cases, the man feels as if he had been struck with a stick or stone; in other cases, instant and intense pain is felt in the wound and down the nerve-trunk; and in some cases there is little or no pain or discomfort. In 91 cases analyzed by Dr. Mitchell, one-third had no pain, and many did not know that they were shot until weakness or the sight of their blood showed that they had received a wound. If the nerve is completely divided, of course, paralysis of motion in the parts supplied by it immediately occurs; but in some cases, even when the nerve is only grazed by the bullet, its function may be completely abolished at once. A case recorded by Mitchell, a soldier who was injured in the brachial plexus by a bullet, had sudden and violent muscular contraction of the muscles of his hand, so that he was obliged to ask a comrade to unclasp his fingers from their hold on the musket. Shock is a frequent consequence of grave nerve injuries. The patient becomes faint and cold, and passes into a condition of general depression.

The secondary effects of injuries of nerves are practically those of intense neuritis. All of the evidences of degenerative changes are present, whether the nerve be completely divided or only partially so. These symptoms have been fully described in the section on Neuritis. I will here refer only to the fact that trophic changes are much more common after wounds of nerves than as a consequence of simple neuritis. There is marked atrophy of the muscles supplied by the injured nerve, and also anæsthesia and hyperæsthesia, with greater or less pain in the distribution of the nerve. One of the characteristic forms of suffering is the violent, burning pain which has been called by Mitchell causalgia.

The condition of the skin and its appendages is greatly changed. Cutaneous disturbances, in the form of herpetic and vesicular eruptions and ulcerations as the result of bullæ, are often present. These heal slowly. The peculiar shining, glossy state of the skin described by Paget is a well-known result of nerve-wounds. Mitchell speaks of excessive growth of hair in the skin to which the injured nerve is distributed. Deformities of the nails are common. They are frequently clubbed, and have marked furrows and ridges upon them. In many cases they are thin, atrophied, and fragile. Loss of hair sometimes occurs as well as increased growth of hair. Sometimes there is an excess of perspiration, which may also be acid and of disagreeable odor.

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[1] Injuries of Nerves, p. 92.

[2] Loc. cit., p. 135.

CHANGES IN NERVES AFTER COMPLETE DIVISION. When the nerve has been severed there occurs within a short time degeneration in the divided ends. The degenerative changes differ materially in the upper and lower ends of the nerve. The alterations which take place in the lower end have been well understood for many years, but it has only been within the past few years that a more thorough knowledge of the changes which occur in the proximal end has been attained.

On examining a nerve which has been divided it is found that at the proximal end there is a bulbous enlargement, a neuroma, which varies in size according to the length of time after injury that the examination is made.

At the lower or peripheral end there is only slight enlargement with flattening, and sometimes, instead of enlargement, there is shrinkage of the end of the nerve. From the cases which I have seen I believe that there is generally a slight enlargement of the distal end up to a year after the injury. Many experiments have been made upon the lower animals as to the degenerative processes which take place in nerves after section, but most writers are agreed that the results of experiments upon animals cannot be altogether applied to man, as in the former changes of degeneration and repair take place much more rapidly than in human beings. However, most of the examinations which have been made in man after injuries have given practically the same results as those experimentally produced in animals. Of late years so many opportunities for examinations have occurred in man after accidents and injuries that sufficient data have been obtained for reliable conclusions.

The degeneration of the fibres in the peripheral end begins almost immediately after section, the degeneration taking place according to the well-known laws established by Waller in 1862. From the fourth to the sixth day, according to Mitchell,[1] there begins to be a change in the nerve-fibres. The white substance of Schwann undergoes irregular segmentation, then becomes granular, and finally disappears. The axis-cylinder also undergoes degeneration. The process extends through the whole of the peripheral end of the divided nerve, so that complete degeneration takes place through its entire extent.

Changes in the medullary sheath have been found to begin as early as the fifth day, and by the twelfth day the axis-cylinder has been found to have disappeared. Bowlby[2] mentions two cases which he had examined, one fifteen days and the other thirty days after nerve-section, when operations for secondary suture were undertaken. In the case of the nerve which had been divided fifteen days previously, there was found much segmentation of the myelin sheaths, and there was still some myelin present, but in a few tubules it was completely absent. The nuclei of the sheaths had increased in number. The axis-cylinders had disappeared in most of the sections which he examined. In the case of longer standing all of the changes were more pronounced. Only a few drops of myelin remained, the axis-cylinder had entirely disappeared, and the nuclei in the sheaths were slightly more increased. In a case which the same author examined three months after section no nerve-tubules could be discovered, and the spaces formerly occupied by nerve-bundles were filled with connective tissue.

CHANGES IN THE PROXIMAL END. The fibres of the central end undergo

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[1] Injuries of Nerves.

[2] Injuries and Diseases of the Nerves, p. 18.

but slight changes after nerve-section. There may be some changes extending to one, or, at the most, two of the internodal segments, according to Ranvier, who says that the myelin instead of becoming broken into large fragments which subsequently become smaller, as in the peripheral end, is rapidly reduced to fine granules, the nuclei multiply and increase in size, but the axis-cylinders remain intact.

The bulbous enlargement at the proximate end was formerly regarded as mainly consisting of fibrous tissue. Mitchell[1] speaks of neuromata of stumps being composed of fibrous tissue, with a layer of nerve-fibres spread over them. Microscopic examinations of these bulbs show very clearly that they consist almost exclusively of nerve-fibres. There is also an increase in the connective-tissue element, with some infiltration of leucocytes. Sections of the nerve above the bulb show none of the degenerative changes which are met with in the peripheral end. In cases which are examined years after the injury there are some atrophy and degeneration in the nerve-fibres. Bowlby says he has examined the nerves from limbs in which amputation had been done many years before, and he found that in them many of the nerve-fibres are diminished in size, with the myelin sheath greatly shrunken.

REGENERATION AND UNION. Regeneration of the proximal end of a divided nerve follows the process of degeneration, and begins frequently within a short time after the injury. There has been a difference of opinion among pathologists as to the process by which the new nerve-fibres are formed, some believing that they are developed from the nuclei of the sheath, and others, among them Neumann and Ranvier, holding that the new fibres are formed by a longitudinal division of the axis-cylinders, which split up like a brush, and which are subsequently covered by myelin. The generally accepted view at the present time appears to be that the new fibres develop in the nuclei of the sheath of Schwann. As soon as the new fibres begin to form they bud out from the proximal end of the nerve and extend toward the peripheral extremity. Regeneration also takes place from the peripheral end, beginning at a later period, and the process takes place more slowly. After a time the ends become united. If union does not take place the peripheral extremity again undergoes degeneration.

The length of time which is required for union to take place varies in different cases, and depends to a great degree upon the width of the space between the ends of the divided nerve. In a case recorded by Mitchell,[2] in which the musculo-spiral nerve was excised for an excessively painful neuritis, six months after the operation evidences of regeneration were observed in return of sensation in the area supplied by the nerve. Fifteen months after the operation muscular power had been restored to such a degree that the patient could extend the wrist, and a year later the functions of the whole extensor group had returned. A second operation was performed about two and a half years after the first, and one inch of regenerated nerve-tissue was found. In another case reported by the same writer, complete union was found after eighteen months in one of the digital nerves, of which an inch had been excised; and in a third case a radial nerve was united within ten months of excision of two inches of its trunk.

We have frequent evidences of degeneration and union of nerves in cases where excision has been performed for neuralgia, and even where an effort has been made to prevent reunion by turning back the proximal end of the nerve, recurrence of pain in the distribution of the nerve has followed after a year or two. In many cases, if the separation between the two ends of the nerve is only slight, union takes place within a few days, but restoration of the function does not occur until some weeks later. If the ends of a divided nerve are immediately brought into apposition and sutured, union generally takes place, and in some cases, if the suturing has been done within a short time of the accident, there is no apparent loss of function. In a case which I saw, in which the sciatic nerve was divided in the popliteal space in mistake for one of the hamstring muscles in an operation for division of those tendons, the divided ends of the nerve were immediately sutured, and, with the exception of some numbness and pain in the foot for a few days, there was no impairment of sensation or motion, and no ill effects followed.

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[1] Injuries of Nerves.

[2] American Journal of the Medical Sciences, April, 1876.

TREATMENT OF WOUNDS OF NERVES. When there is no evidence that the nerve has been completely divided the treatment of an injured or partially divided nerve should be on the same principles that would govern the treatment of neuritis from any other cause. The limb should be kept absolutely at rest, and the wound should be treated on the strictest antiseptic principles. If the external wound has healed and evidences of neuritis remain, it will be necessary to use counter-irritation and maintain the part at rest. Should severe pain be experienced in the nerve-trunk, application of ice-bags will frequently afford relief. Should we have reason to believe that the nerve has been completely divided, it is imperative to immediately bring the severed ends into apposition. The result of immediate operation in such cases is generally satisfactory, and occasionally, though in rare instances, there is union by first intention. Bowlby[1] records several cases in which immediate suturing of divided nerves was made with excellent results. In one case the patient was brought into the hospital a few minutes after a lacerated wound of the wrist by glass had divided the median nerve in two places, so that nearly an inch of the trunk lay loose in the wound. The ends of the nerve were brought into apposition and sutured with catgut. Ten days after the operation sensation began to return, and eventually there was complete restoration of motion and sensation.

The operation of suturing is considered a perfectly safe one, provided thorough cleanliness is observed. Bowlby[2] and Willard[3] say that aseptic catgut or sterilized silk sutures should be used in bringing the ends of the nerve together. Willard advises that two sutures should be used; one should pass directly through the body of the nerve, and a second at right angles to the first.

Willard records 117 cases of primary suture in which the nerves united were as follows: In 41 cases, the median nerve; 38, the ulnar; 30, the median and ulnar; 3, the median, ulnar, and radial; 4, the radial; 3, the musculo-spiral; 1, the sciatic; 1, the external popliteal; 1, the posterior tibial; 1, the anterior tibial. The degree of separation varied from 1 cm. to 5 cm.

The results of primary suture are thoroughly satisfactory. Bowlby[4] records the results of primary suture in 81 cases, as follows:

Successful	32	
Doubtfully successful	12	
Partially successful	22	
Failures	14	
Results not stated	1	
	—	81

The details of the operation will be found in Chapter XXXIII. Several methods have been suggested, but that of bringing the ends in apposition with sutures at right angles is apparently the most desirable.

SECONDARY SUTURE. If the nerve has been divided for a length of time, varying from several months to several years, and no restoration of function has occurred, the success of operation is uncertain; nevertheless, a number of cases have been recorded in which excellent results have been obtained after an interval of many months. Favell[1] records a case in which the sciatic nerve was cut across by the point of a scythe. Nine months afterward the nerve was exposed, and it was found that the ends were an inch and a half apart, the upper end was bulbous and the lower was flattened, no attempt at union having taken place. The two extremities were cut off, and by traction the nerve was stretched sufficiently to unite the two extremities. Steady improvement took place, and in time a good result was obtained.

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[1] Loc. cit.

[2] Loc. cit.

[3] Med. News, October 6, 1894.

[4] Loc. cit.

In the cases in which the separation of the ends of a divided nerve is so great that they cannot be brought into apposition, the operation of nerve-grafting has been employed with more or less success. In a case of Tillemann's a section 4½ cms. long from a nerve of a rabbit was inserted in the median and ulnar nerves. Sensation returned in four weeks, and motion in nine. In a case reported by Robson,[2] 2½ inches of a posterior tibial nerve, which was obtained from a fresh amputation, was implanted in the gap in a median nerve forty-eight hours after division by injury. Four months later sensation was almost entirely restored. A number of other cases have been reported in which nerve transplantation has been successfully performed.

Secondary suture of nerves has been made at intervals, varying from several months to many years after the original injury. In a case reported by Marsh, at St. Bartholomew's Hospital, he united the ulnar nerve twelve years after the injury, and decided improvement in motion and sensation took place. As a rule, however, secondary operations, after a year, are not promising as to the results.

The period at which improvement begins after operation varies, and many circumstances influence the rate of nerve-regeneration; usually improvement begins to show itself after a few days in partial return of sensation. Motor gain begins later. In some cases, however, there is no marked improvement seen until after several months have elapsed, and all hope of improvement should not be given up until after a year.

The results following secondary suture given by Willard[3] in 130 cases are as follows:

Total number of cases slightly improved	10
Greatly improved in motion and sensation, or absolutely cured .	102
Slightly improved or not improved at end of a year	15
Death resulting from hydatids	1

With such results as these, giving 80 per cent. of improvement by operation, we should feel encouraged to operate in every case, no matter how great an interval of time has elapsed after the injury.

The conditions which mainly influence recovery from secondary suture are the general state and habits of the patient and the season of the year. Restoration is more rapid and perfect in young subjects than in old, and is said to be more rapid in warm weather than in cold.[4] Certain nerves unite more readily than others; the musculo-spiral, for instance, seems especially prone to unite after section. In patients who are intemperate, or who insist upon using the member in which nerve-suture has been performed, recovery takes place more slowly, and there is greater risk of failure. The use of electricity and massage is of great importance after nerve-suture. As soon as the external wound is healed a mild galvanic current should be applied to the paralyzed muscles, and in three or four weeks it is well to begin massage to the affected limb. It is necessary to avoid active massage immediately over the wound, but, after an interval of six weeks, gentle massage may be applied directly over the sutured nerve. The faradic current should be substituted for the galvanic as soon as the muscles begin to respond to the former. The strength of the current should never be greater than just sufficient to cause decided muscular contractions, and the application should not be too long at a sitting, but it is important that there should be a treatment at least every alternate day for months.

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[1] Brit. Med. Journ., Aug. 5, 1876.

[2] Trans. Clinical Society, London, 1889.

[3] Loc. cit.

[4] Mitchell: "Injuries of Nerves," and Bowlby, loc. cit.

INJURIES OF SPECIAL NERVES. The nerves most frequently injured are the median, ulnar, radial, musculo-spiral, and sciatic, in the order given.

The *median nerve* supplies the pronators of the wrist, the flexor carpi radialis, the superficial and deep flexors of the fingers, except the ulnar half of the deep flexor, the flexor longus pollicis and abductor pollicis, and the two radial lumbricales. It also supplies the skin on the radial side of the palm, the anterior surface of the thumb, the first two fingers and half of the third. The posterior aspect of the distal phalanges of the fore and middle fingers are also supplied by this nerve. The cutaneous distribution varies greatly in individuals; in some it is much more limited than in others. In some cases of injury of the median nerve there is but little loss of sensation; but, as a rule, loss of sensation on the palmar surface of the thumb and forefinger is complete, and on the same surface of the middle-finger it is partial. The patient is unable to flex the last phalanges of the thumb, forefinger, and middle-finger. The third and fourth fingers can be flexed through the part of the flexor profundus which is supplied by the ulnar nerve, and flexion of the proximal phalanges of the fore- and middle-fingers is accomplished by the interossei. In some cases, however, the loss of power of flexion is confined to the thumb and forefinger. The patient is unable to abduct the thumb, and it remains in a position of extension and adduction, resembling, as Gowers remarks, the thumb of an ape.

FIG. 241.

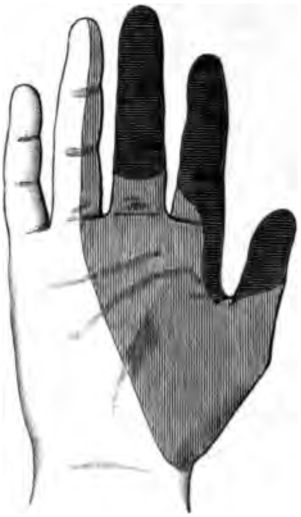
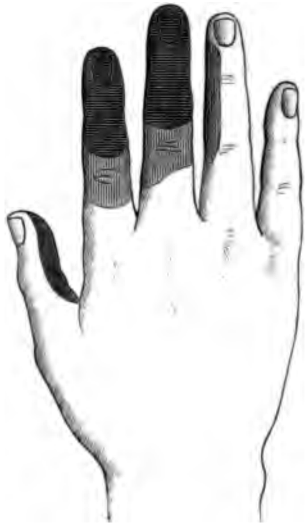


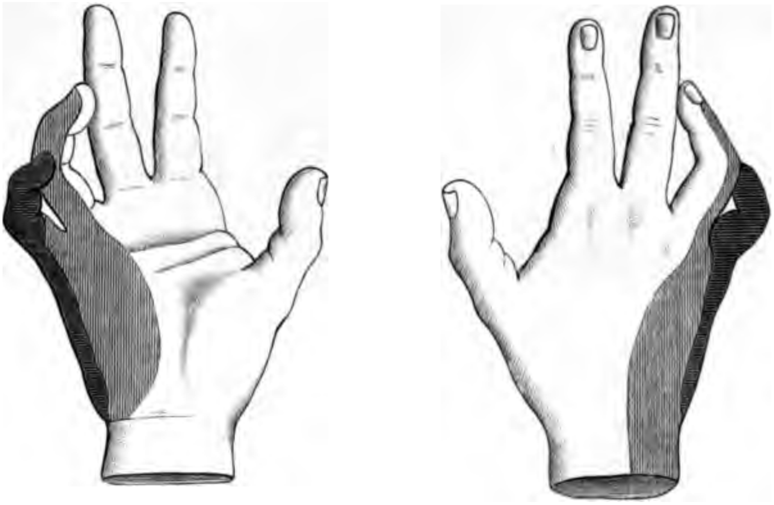
FIG. 242.



Showing areas of sensory loss in injuries of the median nerve. (BOWLBY.)

The *ulnar nerve* supplies the flexor carpi ulnaris and the inner half of the flexor profundus digitorum, the muscles of the little finger, the interossei, some of the lumbricales, and the adductor pollicis and inner head of the flexor brevis pollicis. The sensory distribution of the ulnar nerve is to the little finger and inner half of the third finger. After division of the ulnar nerve there is distinct anæsthesia in the little and third fingers, both on the palmar and dorsal surfaces, but more markedly upon the latter. Adduction of the thumb is lost, and so are all of the movements of the little finger. None of the fingers can be flexed in their proximal phalanges, but in the first two fingers the loss of power is not so great, because the median nerve sends branches to the first two lumbricales. Atrophy takes place in the muscles, giving rise to great wasting of the hypothenar eminence, the palm becomes hollow, and contraction of the little finger occurs. After several months have elapsed great contraction of the common extensors and long flexors of the fingers and thumbs takes place in consequence of the paralysis of the lumbricales and interossei, giving rise to the claw-like hand.

FIG. 243. FIG. 244.

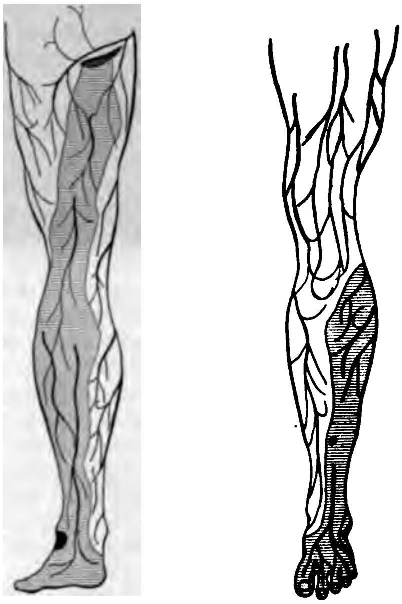


Showing sensory loss in injuries of the ulnar nerve. (BOWLBY.)

The *radial nerve* supplies the skin on the radial side and ball of the thumb by its external branch, and by its internal branch it supplies the skin on the ulnar side of the thumb, the radial side of the index finger and the adjoining sides of the index and middle fingers, and also the adjacent sides of the middle- and ring-fingers. In a case reported by Bowlby, in which a deep wound on the back of the radial side of the wrist divided the radial nerve, sensation was completely lost over a small area, including the back of the first and second metacarpal bones of the thumb. The radial nerve gives off no muscular branches.

Division of the *musculo-spiral nerve* gives rise to characteristic symptoms. This nerve supplies the extensor muscles of the fingers and wrist, and the supinators. The triceps also receive branches from the musculo-spiral; but, as the nerve is usually injured near the middle of the humerus, the branches supplying these muscles escape. There is little or no anæsthesia after division of this nerve, although there are subjective sensations of tingling and numbness in the hand and fingers. In a patient in whom the musculo-spiral nerve was divided by a stab there was an area of lessened sensation on the back of the thumb. The paralysis of the extensors causes the typical wrist-drop and loss of power of extending the distal phalanges of the fingers and thumb. There is ability to extend the proximal phalanges which are supplied by the interossei and lumbricales. The patient is unable to flex the fingers strongly on account of the loss of antagonism of the extensors. This is shown when an attempt is made to squeeze the dynamometer. If the instrument is placed in the hand and the patient makes an effort to grasp it, the wrist is flexed and the dynamometer registers only a few degrees. If the instrument is supported so that flexion of the wrist does not occur, the grasp of the hand shows almost normal strength. There is marked muscular wasting in the forearm. It is unusual to see trophic changes after division of this nerve; but, as a result of the overflexion of the wrist and the loss of support by the extensor tendons, there is frequently a prominence of the back of the hand, due to an enlargement of the synovial sacs, giving rise to a characteristic hump. Although the supinators are paralyzed, the patient is still able to supinate the forearm by the aid of the biceps muscle.

FIG. 245.



Area of anæsthesia from division of sciatic nerve.

The *sciatic nerve* supplies the flexors of the legs and all of the muscles below the knee. The cutaneous distribution of the sciatic nerve is to the integument of nearly the whole of the leg. When the nerve has been divided there is inability to flex the leg and to flex the foot. The patient, however, is able to walk, very much like a child suffering from poliomyelitis, by swinging the leg around so as to make the toes clear the ground, and by lifting the foot by the aid of the hip muscles. The amount of anæsthesia met with after division of the sciatic nerve is much less extensive than would be supposed, owing to the distribution of the small sciatic, the long saphenous, the external, middle, and internal cutaneous nerves. There seems to be

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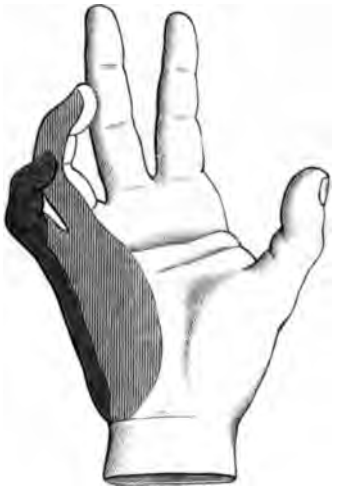

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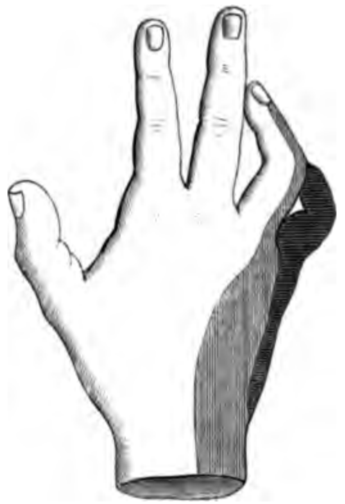

Showing sensory loss in injuries of the ulnar nerve. (BOWLBY.)

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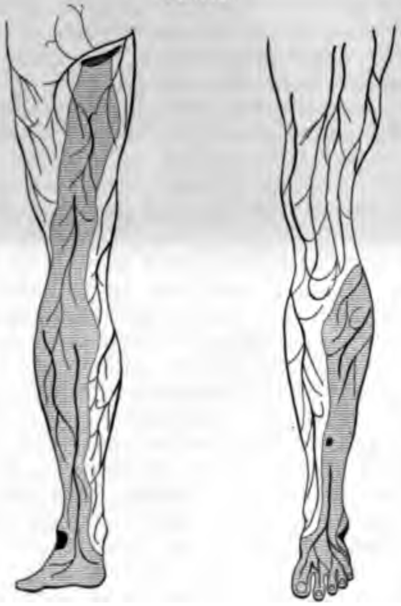

Area of anæsthesia from division of sciatic nerve.

The *sciatic nerve* supplies the flexors of the legs and all of the muscles below the knee. The cutaneous distribution of the sciatic nerve is to the integument of nearly the whole of the leg. When the nerve has been divided there is inability to flex the leg and to flex the foot. The patient, however, is able to walk, very much like a child suffering from poliomyelitis, by swinging the leg around so as to make the toes clear the ground, and by lifting the foot by the aid of the hip muscles. The amount of anæsthesia met with after division of the sciatic nerve is much less extensive than would be supposed, owing to the distribution of the small sciatic, the long saphenous, the external, middle, and internal cutaneous nerves. There seems to be great want of accord in the reports of different writers as to the areas of anæsthesia after division of the sciatic. The following case shows quite well the area of anæsthesia after complete division of the nerve.

John B. W., aged nineteen years, ship carpenter by occupation, was admitted to my wards at the Episcopal Hospital, Philadelphia, April 26, 1877. On December 25, 1876, while working at his trade of sparmaking, he slipped and fell upon an adz, receiving a severe wound in the upper posterior part of his left thigh. He bled profusely, and fainted. About a week after the accident he began to feel pain in the back of the feet and toes, which persisted for nearly two months and then disappeared. About a month after the injury small vesicles formed upon the dorsum of the toes, which gradually attained the size of a three-cent piece, and, after breaking, left ulcers, which healed slowly. The original wound did not heal for about four weeks. On admission to the hospital a scar three inches in length was found in the upper inner posterior surface of the left thigh just below the tuber ischii. There was complete loss of power of motion below the knee. The left calf measured 11 inches in circumference, and the right 12½ inches. A shallow ulcer 1½ inches in diameter existed over the external malleolus, and there was another small ulcer on the anterior portion of the leg. The toe-nails were marked by transverse grooves about the middle of each nail. The muscles responded to a very strong slowly interrupted faradic current, but did not respond to the rapidly interrupted current. The galvanic current caused much more active response in the muscles of the left leg than in those of the right. The accompanying diagram, made by Dr. Robert Meade Smith, the resident physician, shows very accurately the area of anæsthesia.

NEURALGIA.

Neuralgia is a functional disease of the sensory fibres of the nerve-trunks, and is characterized by pain, as indicated by the name. This definition is not as universally applicable as it would have been a few years ago, as it is now recognized that certain forms of neuralgia are due to neuritis; and it is a question as to whether we should consider any form of neuralgia as purely functional, as, no doubt, during the attack of pain there are distinct changes in the nerve. Neuralgias are either *idiopathic*, in which the disease is dependent purely upon functional disturbance, or *symptomatic*, due to some organic disease affecting the nerve, such as a tumor or toxic influences. In neuritis we have an example of a symptomatic neuralgia, but the ordinary forms of neuralgia in which the pain is transient, and where it flies from one part to another, the symptoms cannot be due to actual organic changes.

Neuralgias are classed according to their cause, and also according to their location. The commonest forms of neuralgia are: trigeminal, or neuralgia of fifth nerve, cervico-occipital, brachial, intercostal, sciatic, and visceral. Neuralgias may depend upon some toxic cause, as, for instance, gout, rheumatism, or diabetes, and other diseases causing dyscrasia. The most common form of neuralgia is that affecting the fifth nerve, and next in order come sciatic and intercostal neuralgias.

Neuralgia is essentially a disease of adults; it never affects young children, except in the form of migraine. In old age neuralgia is rare. Women suffer much more from neuralgia than men, in the proportion of five to three, according to Dana,[1] who made an analysis of 887 cases. More cases occur in winter and autumn than in warm weather, and neuralgia is more common in damp and cold climates than in those which are dry and temperate.

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[1] Diseases of the Nervous System, p. 81.

Heredity plays a decided part in the production of this disease. The gouty and rheumatic diatheses also predispose to it. Anæmia is one of the most common causes of neuralgia, the nerve-pain in these cases having been termed "the cry of the nerve for more blood." In some cases neuralgia seems to be dependent upon reflex irritation, as in the case of caries of the teeth, which causes neuralgia of the fifth nerve, or from irritation from disease of the eye, as glaucoma, in which the ophthalmic division of the fifth nerve is the seat of severe pain. Neuralgias are recorded as being produced by reflex irritation in various organs, as the ovaries, stomach, and brain.

The pain of neuralgia is usually sharp, lancinating, boring, or burning in character, or it may be merely dull and gnawing. In certain nerves the pain is intensely acute, as, for example, in the fifth nerve, where it seems to dart with lightning-like rapidity into every filament. The pain of neuralgia is not steady, but is intermittent and paroxysmal. Sometimes between the paroxysms a dull ache remains. The pain is increased by cold and sometimes by pressure on the affected part, although often this relieves it. In some cases the skin in the distribution of the affected nerve is excessively tender. The intervals between the paroxysms of pain are greater or less in length, sometimes being only a few minutes, and in other cases hours intervene between the attacks. Frequently the paroxysms recur at the same hour of the day, giving rise in many cases to the suspicion that the disease is malarial. In sciatic neuralgia the most common time of the day for the attack of pain is about 6 P. M. An attack of neuralgia may last only a few days or it may continue for years, with but brief periods of relief. The pain in neuralgia is sometimes of such an explosive character as to give rise to the name of epileptiform neuralgia, as seen in *tic douloureux*.

Neuralgias are sometimes met with in neurasthenic and hysterical patients, in whom a painful impression remains after the true paroxysm has passed away. In patients who are addicted to morphine, this form of "reminiscent" or "hallucinatory" neuralgia is frequently seen, and the pain seems to be due to the craving of the patient for the narcotic.

Pathology. In many cases of neuralgia there is neuritis to a greater or less extent. This is especially the case in sciatica and in neuralgias of the brachial plexus. In cases of neuralgia of the fifth nerve of long standing there is almost always found an inflammatory condition of the nerve-trunk; even in short attacks of neuralgia, as in the supra-orbital nerve, where the attack is of but a few hours' duration, the nerve can be felt swollen under the finger in the supra-orbital notch. In some cases neuralgia seems to be due to some irritant poison in the blood, as in gout, rheumatism, or diabetes, and in other cases to some of the metallic poisons, like lead, arsenic, and mercury. There are other cases in which no diathetic or local cause can be traced, and in these it is supposed that the pathogenic focus is in the spinal ganglia or in the sensory cells of the posterior horns of the cord. This, however, is not an explanation of the cause, as we do not know what is the condition of the cells in the spinal cord or the spinal ganglia which produces the attack of neuralgia.

Diagnosis. The diagnosis between neuralgia and neuritis depends on the fact that in the former the pains are paroxysmal and shift from one point to another, taking the course of the nerves in some cases, and in others flying from one nerve to another. In neuralgia there is no anæsthesia in the distribution of the nerve-trunks, and motor paralysis does not occur. (See Chapter XXVII. for detailed consideration of treatment.)

CHAPTER XXVI.

DISEASES OF THE CRANIAL NERVES.

By GEORGE E. DE SCHWEINITZ, M.D.

DISEASES OF THE OPTIC, OCULOMOTOR, PATHETIC AND ABDUCENS NERVES.

THE nerves designated in the title represent two of the four groups into which, according to their physiological actions, it is customary to divide the cranial nerves. Their intimate association with the complex problems of intra-cranial physiology, as well as their anatomical relations to the structures at the base of the brain, and, through their nuclei and centres of origin, with its deeper tissues, render changes at the intra-ocular end of the optic nerve, disturbances of the equilibrium of the external ocular muscles, and anomalies of the pupillary reflex of the utmost importance in the study of many problems of nervous disease.

DISEASES OF THE OPTIC NERVE.

NORMAL RELATIONS. The optic nerves, second in the list of nerves of special sense, arise from the anterior part of the commissure, pass through the optic foramina, traverse the orbits, and enter the sclerotic and choroid coats to expand in the retinas. Each nerve is invested with an *inner* (pial) and an *outer* (arachnoid-dural) sheath, between which is a space—the *inter-vaginal space*—which is derived from the subdural and subarachnoid spaces.

The optic nerve is usually regarded as exclusively concerned with the sense of sight, but it also contains the afferent fibres of the pupil-reflex, which, according to von Gudden, may be histologically differentiated from those which are designed for vision. The total number of fibres in the optic nerve has been estimated at 400,000.

The smallest branch of the ophthalmic artery, arising from the cavernous portion of the internal carotid, is the central artery of the retina, which pierces the optic nerve obliquely and reaches the retina, upon which its branches are distributed. It is accompanied by a vein which collects the blood from the retinal veins and empties into the ophthalmic vein, which, in its turn, pours its blood into the cavernous sinus. There is free anastomosis between the ophthalmic vein and the branches of the facial vein.

VISUAL PATH.[1] The deeper relations of the optic nerves are best appreciated by tracing the visual pathway. The peripheral percipient elements are the rods and cones of the retina, and the macular fibres, constituting about one-fourth of the nerve, enter the papilla at its infero-temporal side, forming the so-called *papillo-macular* bundle which gradually approaches the axis of the optic nerve, reaching it in the optical canal. In the front of the chiasm it occupies the upper and inner portion, being divided into crossing and direct fibres, but in the tractus it sinks to the central portion and passes along the tract on its way to the cortex in the cuneal region.

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[1] According to Noyes: N. Y. Med. Record, April 4, 1891, and Wilbrand: Die Hemianopischen Gesichtsfeld-Formen und das optische Wahrnehmungszentrum.

The remaining fibres (*intermediary and peripheral*) are so arranged that the remaining right temporal and all of the left nasal, on the one hand, and the remaining left temporal and all of the right nasal on the other, by commingling in the chiasm, join respectively at the beginning of each optic tract and proceed, the one set to the right and the other to the left cuneus.

The *chiasm* is the flattened body, resting upon the sphenoid, in which the fibres undergo a semi-decussation,[1] and which lies between the optic nerves and their continuations—the optic tracts. Each tract winds around the corresponding *crus cerebri* and terminates in two roots upon the *corpora geniculata externa* and *interna*, and upon the posterior part of the *optic thalamus*, called the *pulvinar*. Fibres also go to the anterior part of the *corpora quadrigemina;* but these organs are not regarded as concerned in vision, but in the activity of the pupil. The parts just referred to are called the *primary visual ganglia* or *primary optic centres.*

In them are found innumerable ganglion cells in which the fibres of the tractus lose themselves, and thereafter a new set of fibres proceeds backward through the posterior part of the *internal capsule* to the cortex, under the name of the *visual radiation*, or *fibres of Gratiolet or of Wernicke.* Passing through the internal capsule they cross the sensitive fibres coming down from the hemisphere, and then, spreading out like a fan, rise upward, wind outside the tip of the *lateral ventricle* to reach their destination at the lower part of the median surface of the *occipital lobe.* (See Fig. 136, p. 491.)

Hyperæmia of the Optic Nerve; Congestion.

Etiology. (*a*) Refractive error, especially hypermetropia and hypermetropic astigmatism.

(*b*) Occupations which expose their subjects to intense glare and heat.

(*c*) Toxic agents, for example, tobacco, alcohol, and lead.

(*d*) Diseases of the eye, *e. g.*, inflammation of the iris.

(*e*) Disorders of the brain and spinal cord, the former including various types of insanity and general paralysis of the insane, and the latter chronic lesions of the posterior columns of the spinal cord, particularly locomotor ataxia. In mania Albutt has found the disks hyperæmic and sometimes pale; Noyes has observed both anæmia and hyperæmia, but in only one case could Gowers distinctly determine a pathological congestion. Melancholia is usually unassociated with ophthalmoscopic changes, although hyperæmia has been recorded. In chronic dementia Allbutt noted changes twenty-three times out of thirty-eight—sometimes atrophy and sometimes hyperæmia. Lautenbach[3] examined 707 insane patients, including mania, dementia, melancholia, and other cases not exclusive of epilepsy, and records frequent retinal hyperæmia and congestion, amounting to 40 per cent. in the acute cases. There is, however, no fundus-lesion characteristic of insanity.

In general paralysis of the insane, hyperæmia has also been observed by Uhthoff and Gowers, while Albutt believes that the atrophy is ushered in by a stage of hyperæmia, and he has made a similar observation in locomotor ataxia. In cases examined by the author no true congestion of the disk has been observed, although not infrequently there is a dull red appearance, with marked grayness of the deeper layers, followed later by undue broadening of the scleral ring and ultimately by atrophy of the nerve. Focal brain lesion may be accompanied by hyperæmia, for example, cerebral embolism associated with secondary brain irritation.[1]

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[1] This semi-decussation is denied by some observers.

[2] Ophthalmic Studies of Acute Mania, Journal of Nervous and Mental Disease, 1886, vol. xiii. p. 337.

(*f*) Heart disease, violent cough, etc., or any cause which produces engorgement of the veins of the head and neck, for example, convulsive seizures, may occasion retinal hyperæmia. It is not uncommon to find congestion of the nerve-tip and undue fulness of the venous retinal circulation in general epilepsy, but nothing characteristic of the disease is visible in the fundus oculi, and even the hyperæmia which has been described may not always be due to the disease in which it occurs. In epilepsy of sufficient moment and standing, however, Oliver[2] considers low and chronic forms of retinitis associated with a dirty red-gray incipient degeneration of the optic disk to constitute the type of ophthalmoscopic findings.

The difficulty of deciding whether congestion of the nerve-tip or retina is caused by a cerebral condition depends upon the fact, as Gowers has expressed it, that increased vascularity of the papilla is not an index of hyperæmia of the cerebral vessels, and hyperæmia of the retina is inferred by the undue redness of the disk, rather than by an altered appearance of the membrane itself, although we may speak of a hyperæmia of the central vessels, meaning, if it is active, that the arteries are distended, tortuous or lengthened; if it is passive, that the veins are large, twisted, and filled with dark blood.

Symptomatology. (*a*) *General Considerations.* In order to appreciate morbid changes at the intra-ocular end of the optic nerve, familiarity with its natural condition is necessary.

NORMAL FUNDUS OCULI. When viewed with the ophthalmoscope the optic nerve appears as a nearly round or slightly oval disk, with distinct margins, especially on the temporal side, varying in color from a grayish pink to more decided red, the tint being most marked upon the nasal side. The centre is occupied by a whiter patch, marking the position of the emergence and entrance of the retinal bloodvessels, or by an actual excavation shelving toward the temporal border (*the physiological cup*). The disk is surrounded by two rings: the outer, often incomplete and frequently wanting, is dark-colored, and represents the margins of the choroidal opening through which the nerve passes (hence the *choroid ring*), the other a faintly marked whitish circle, indicates the rim of the sclerotic coat (hence the *scleral ring*).[3] From the central spot the principal retinal arteries emerge, and into it the chief venous trunks empty. The arteries divide dichotomously, and usually spring from one stem, which separates into two principal divisions, which spread over the retina and are accompanied by the veins which pass in the same general direction. The veins are larger than the arteries in the proportion of three to two, and dark red in color; the arteries assume the natural blood red tint. Normally the arteries do not pulsate, but spontaneous pulsation in the veins is frequent.[4] (Fig. 1, Plate V.)

(*b*) *Ophthalmoscopic Appearances of Congestion.* Mere redness of the intra-ocular end of the optic nerve is not congestion. This term is applicable only, according to Gowers, when the color of the disk is dull red or brick-dust, when it is difficult to differentiate the margin from the general red color of the eyeground, when one eye is more affected than the other, so that the second may be taken as a point of comparison, and especially when

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[1] Consult Gowers's Medical Ophthalmoscopy, second edition, p. 46.

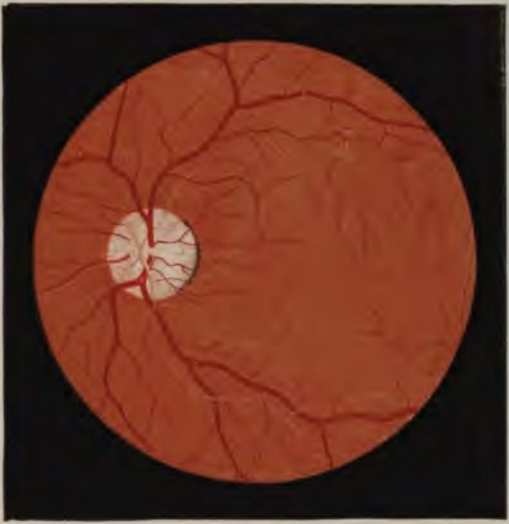
[2] University Medical Magazine, March, 1894.

[3] Called by Loring the "connective tissue ring."

[4] Lang and Barrett found venous pulse in 73.8 per cent. of the cases examined, and the author in 62.1 per cent., and Dr. Veasey, one of his assistants, in 58.3 per cent., making a general average of 64.7 per cent.

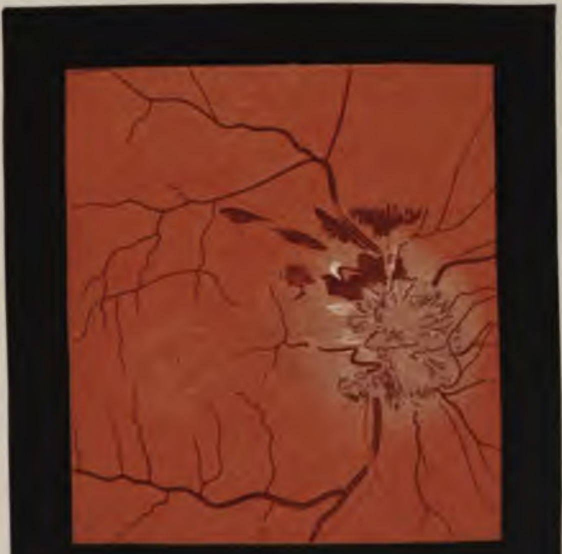
PLATE V.

Fig. I.



Normal eye-ground (average tint). (Norris & Oliver.)

Fig. 2.



Ophthalmoscopic appearances in early stage of papillitis.
(Norris & Oliver.)

previous ophthalmoscopic examination has demonstrated that the color is now an unnatural one. The disk is not swollen and the borders are not hidden, but obscured, and there are no hemorrhages. There may be a disproportion between the size of the veins, which are unduly dark and tortuous (the normal proportion being as 3 to 2), and the arteries, remembering that the diameter of the veins is much more frequently increased than that of the arteries, while it is more common to find small arteries than those which are distended. Normally, the veins are slightly tortuous in many eyegrounds; but in cases of true hyperæmia, or rather, exaggerated filling of the central vascular system, this becomes a marked feature. It is often difficult to interpret the significance of hyperæmia, and much undue prominence has been ascribed to hyperæmic nerve-heads in connection with cerebral symptoms when their existence was but a coincidence.

Prognosis. The prognosis depends entirely upon the cause. If it is removable, the intra-ocular health may be restored to normal. Lautenbach believes that congestion itself may be utilized as a prognostic point in acute manias as compared with more serious lesions. Thus, hyperæmia which tends to disappear occurs in cases which recover; hyperæmia which goes on to neuritis and subsequent atrophy is seen in those which do not recover.

Treatment. This, independently of those cases which belong strictly to ophthalmic work, is the same as that indicated for the case in which the symptom occurs. Rest for the eyes should be prescribed, and, although rarely accompanied with subjective symptoms, if there should be dread of light, tinted glasses would be indicated, and, internally, bromide of potassium and ergot.

OPTIC NEURITIS. This may be divided into those cases exhibiting distinct lesions at the intra-ocular end of the nerve (*intra-ocular optic neuritis*), and into those unassociated with such lesions, or at most apparent only in an indistinct manner (*orbital optic neuritis*).

Varieties of Intra-ocular Optic Neuritis. Various types of inflammation, either with or without the appearances of engorgement, are seen at the intra-ocular end of the optic nerve. Systematic writers in former days, and occasionally at the present time, describe two chief types, called respectivly *choked disk* (Stauung's papille), and *descending neuritis* (interstitial neuritis), to which a third is sometimes added, namely, *neuro-retinitis.* To avoid confusion in nomenclature, and at the same time to escape unproven theories in pathology, Leber proposed the general term *papillitis*, and this word will be used in the following pages to describe the various types of optic neuritis which chiefly manifest themselves to the ophthalmoscope at the nerve-tip.

Theories Concerning the Mechanism of Papillitis. As is well-known, Von Graefe at one time sharply distinguished between descending neuritis and so-called choked disk. The former, as its name implies, indicated a descent of inflammation from the seat of disease along the optic nerve, which then became manifest at the intra-ocular tip. The latter was reserved for a condition which was supposed to indicate, by its appearance, engorgement, œdema and mechanical obstruction, and to represent a mechanism and pathology quite different from that ascribed to the former. Inasmuch as ophthalmoscopically it is frequently impossible to tell one from the other, and as the conditions may be mixed, it is wise to coincide with the opinion of Hughlings Jackson, that there is one kind of optic neuritis from intra-cranial disease which may manifest itself under different appearances, sometimes with and sometimes without "swelling of the disk." The following are the most important theories of the mechanism of papillitis:

(*a*) *The Back Water Theory*, propounded by Von Graefe, taught that through increased intra-cranial pressure there was compression upon the cavernous sinus, which induced venous congestion in the central vessel of the optic nerve, because the passage of blood returning from the eye was hindered, and that this congestion was increased by the rigid scleral ring which acted as a multiplier. This theory ceased to be tenable when Sesemann demonstrated the anastamosis between the ophthalmic vein and the anterior facial vein. (See Fig. 79, p. 367.)

(*b*) *The Lymph-space Theory* arose after the demonstration by Schwalbe and Retzius of the lymphatic circulation of the eyeball and the communication between the subarachnoid space and the inter-vaginal sheath of the optic nerve. First Schmidt-Rimpler, and later Manz, ascribed to the dropsy of the inter-sheath space of the optic nerve, which is caused by the increased subarachnoid fluid being forced into this situation under the influence of elevated intra-cranial pressure, a mechanical or compressing action, or to the fluid which found its way into the lymphatic spaces of the optic disk, an action causing œdema, congestion and inflammatory symptoms.

(*c*) *The Vasomotor Theory* was first suggested by Schneller, and has been advocated by Benedict and Hughlings Jackson. To use Jackson's words,[1] "Optic neuritis may be a doubly indirect result of local gross organic disease; that first there are changes of instability about the tumor; that next these lead on to discharges, by intermediation of vasomotor nerves, to repeated contractions, with subsequent paralyses, of vessels of the optic nerves or centres, and thus, at length, to that trouble of nutrition which is optic neuritis." This hypothesis has few followers on account of the lack of anatomical demonstration of the possibility of such a mechanism.

(*d*) *The Inflammatory Theory*, with various modifications, assumes, as Leber suggested, and Deutschmann afterward experimentally showed, that papillitis was not a product of œdema, but an inflammatory affection, the fluid which distends the sheath of the nerve possessing an irritative quality; or, in other words, that the subarachnoid fluid is infected by products from the intra-cranial disease or lesion which is the prime cause of the trouble. Microscopic investigation has shown that not only in basilar meningitis may an inflammation be traced from the source of the disease to the intra-ocular end of the nerve, but also in many cases of tumor, the focus of inflammation being, perhaps, a localized meningitis or a surrounding cerebritis. Dr. Gowers points out the frequency of such direct inflammatory connection, while he does not deny the well-known ophthalmoscopic appearances of mechanical congestion, or swelling of the disk, which have given rise to so much confusion in nomenclature. He further states, papillitis may continue slight and without such appearances throughout the course of its existence, or they may arise for reasons not now definitely known, but not on account of a compression by the scleral ring, but from the deposition of inflammamatory products within the inflamed nerve-head. The intensity of the process may also be added to by the distention of the nerve-sheath, and perhaps, although not yet proven, by the admission of the cerebro-spinal fluid, possessing an irritative quality, within the lymph spaces of the papilla. This, in the author's opinion, best describes, so far as our knowledge goes, the mechanism of optic neuritis, and corresponds with numerous microscopical investigations which he has had the privilege of making.

Elsching,[2] in a research based upon the post-mortem results of fifty-five cases of intra-cranial disease, shows that the ophthalmoscopic picture to which the term "choked disk" is applicable is indicative of an inflammation of the optic papilla characterized by a high degree of swelling of all tissues of the papilla. In no case are well-marked evidences of inflammation wanting, and similar changes are found in the trunks of the nerves.

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[1] Transactions of the Ophthalmological Society of the United Kingdom, vol. i. p. 89.

[2] Wien. klin. Wochenschrift, December 20, 1894.

Etiology. The most usual intra-cranial cause of papillitis is tumor of the brain. According to the statistics of Annuske[1] and Reich[2], 88 cases of intra-cranial and, for the most part, brain tumors were accompanied in 95.4 per cent. with optic neuritis or neuritic atrophy, in 93.2 per cent. with double optic neuritis, and in 2.2 per cent. with monocular optic neuritis. In 4.5 per cent. ophthalmoscopic changes were absent. Oppenheim[3] found that in 82 per cent. of his 23 cases there was neuritis of one or both sides, and accepts Annuske's views as to the frequency of papillitis. Gowers believes that optic neuritis is present in $\frac{4}{5}$ or 80 per cent. of all cases of cerebral tumor.

A very important analysis by Edmunds and Lawford,[4] of cases of intra-cranial tumor, throws light, not only upon the frequency of optic neuritis, but upon its relative frequency, according to the situations of the neoplasms. (See table.)

Locality of Tumor.	Optic neuritis.	No optic neuritis.	Total.	Per cent. of optic neuritis.	Per cent. without optic neuritis.
1. Anterior frontal convolutions	8	2	10	80	20
2. Motor convolutions	...	12	12	...	100
3. Occipital lobes	5	4	9	55.5	44.5
4. In hemispheres	6	4	10	60	40
5. Ganglia at base	17	3	20	85	15
6. Tempero-sphenoidal lobes	1	2	3	33.3	66.6
7. Cerebellum	20	3	23	86.9	13.1
8. Medulla and pons	3	6	9	33.3	66.6
9. Meningeal growths at motor convolutions .	4	1	65	80	20
10. Meningeal growths elsewhere . . .	4	2	6	66.6	33.3
Total	68	39	107		

From this analysis these authors point to the comparative immunity from optic neuritis of the cases of tumor in the cortical motor area, and the frequency and severity of papillitis in cerebellar tumors. If all cases toward the convexity of the brain (groups 1, 2, 3, 4, and 9) be added together, there is a yield of 50 per cent. of optic neuritis, while those toward the base (groups 5, 6, 7, 8, and 10) constitute 61 cases, with a percentage of 74 of optic neuritis. Hence it may be safely stated, on the basis of these statistics, as Gowers declares that fully 80 per cent. of cases of intra-cranial tumor at one time or another of their existence develop optic neuritis.[5] Perhaps this percentage would rise even higher if investigations were more thorough and frequently repeated, because a tumor may exist for a long time without causing papillitis, but later this symptom appears. Growths in two regions of the brain seldom produce papillitis, namely, the medulla, and, according to Rath,[6] the hypophysis. True, tumors involving the medulla and pons may have this association, but when the growth is strictly localized in the medulla, optic neuritis, if it occurs at all, must be extremely rare.

Of the four varieties of meningitis—simple, tubercular, traumatic, and cerebro-spinal—tubercular disease of the base is most frequently the cause of optic neuritis, the percentage varying from 76 per cent. (Allbutt) to 81 per cent. (Heinzel) of the cases. In simple meningitis of the convexity, ophthalmoscopic changes usually fail, unless it has lasted for a long time, but neuritis by propagation may be the result of this disease at the base of the brain. Optic neuritis has been described in hemorrhagic pachymeningitis, and may be present in high degree in purulent meningitis; for example, in septic conditions.

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[1] Archiv. f. Ophthalmologie, 1873, Bd. xix. Abth. iii. p. 165.

[2] Monatsbl. f. klin. Augenheilk., Jahrgang xii. p 274.

[3] Quoted by Philip Coombs Knapp: "Pathology, Diagnosis, and Treatment of Intra-cranial Growths."

[4] Trans. of the Ophthal. Soc. of the United Kingdom, vol. iv. p. 172.

[5] Examinations by the author in the Philadelphia Hospital and Infirmary for Nervous Diseases yield a percentage fully equal to this.

[6] Quoted by Vossius, Lehrbuch der Augenheilkunde, p. 592. This is not according to the author's experience in two cases; there was no autopsy.

With cerebral abscess papillitis occurs, differing in no wise from that provoked by cerebral tumor, but it is less frequently observed with cerebral hemorrhage, softening from vascular disease of the brain, meningeal hemorrhage, and chronic cerebritis. In the cases of cerebral hemorrhage it is difficult to decide whether the hemorrhage or the disease—for example, gout, syphilis, or nephritis, which caused the intra-cranial extravasation—is the etiological factor. Hemorrhages into the substance of soft intra-cranial neoplasms would be accompanied by neuritis, which then should be ascribed to the tumor, and not to the hemorrhage. Other intra-cranial causes, also uncommon, are thrombosis of the cavernous sinus, chronic hydrocephalus (unassociated with tumor), and aneurism of the internal carotid.

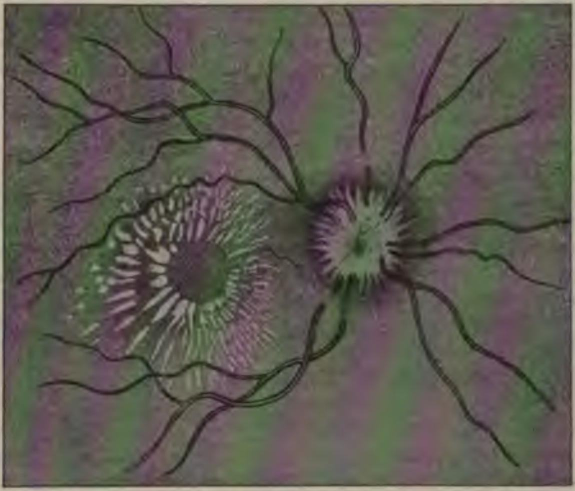
In rare instances myelitis is associated with optic neuritis, and slight forms are seen in general paresis (Uhthoft), and in some cases of chronic insanity, epilepsy, and disseminated sclerosis.

The non-intra-cranial causes of papillitis are: Acute febrile affections, syphilis, toxic agents (lead, alcohol), anæmia, menstrual disorders, exposure to cold, rheumatism, injuries, diseases of the orbital region, and perhaps intranasal lesions. Occasionally papillitis is a congenital affection, and may occur as an idiopathic disease without evident cause. The slight grades of neuritis which are so frequent with refractive error have been mentioned.

SIGNIFICANCE OF OPTIC NEURITIS. Double papillitis is highly significant of intra-cranial disease, especially tumor or basilar meningitis, but is not a pathognomonic sign. Other causes which have been mentioned, prominently Bright's disease, gout, and certain poisons, which, indeed, may or may not be associated with the intra-cranial lesion, must be excluded. Conversely, brain tumor (usually cerebellar) may cause "choked disk," with a star-shaped figure of whitish color in the macular region, exactly simulating the appearances usually considered characteristic of a type of albuminuric retinitis. The author has seen this in a number of cases at the Philadelphia Hospital and at the Infirmary for Nervous Diseases. (Fig. 246.)

Optic neuritis gives no information of the nature of the tumor which may cause it, although sometimes it is the only positive sign of its presence, because it occurs with all forms of neoplasm found in the brain—fibroma, sarcoma, glioma, carcinoma, solitary tubercle, gummata, cysts (entozoic and otherwise), and hæmatoma of the dura mater. Neither does neuritis afford evidence as to the size of the growth, as it may be intense with a small lesion and practically absent with a large one; but unusual swelling of the nerve-head is more significant of tumor than slight papillitis (interstitial neuritis or descending neuritis), which is rather indicative of meningitis. The subsidence of a neuritis which has not greatly developed points to subsidence of its intra-cranial cause; but it must be remembered that neuritis under all circumstances is a transient phenomenon, although it may be long-continued. Again, sudden increase in papillitis, particularly associated with the symptoms of mechanical obstruction, is not without indication of increase, if not of the size, of the irritative quality of the intra-cranial lesion. Finally, it should be remembered, in searching with the ophthalmoscope for evidences of brain disease, that the neuritis may have entirely passed away, except traces in the form of spots of degeneration in the retina, filling in of the centre of the disk, or white tissue surrounding the vessels.

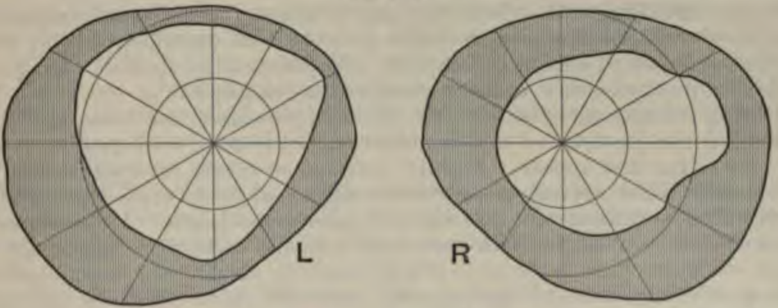
FIG. 246.



Right fundus oculi of a woman aged twenty-five years; optic neuritis and star-shaped macular figure simulating appearances seen in albuminuric retinitis. Kidneys normal.

Symptomatology. There is increased redness of the nerve-head and obscuration of the borders, followed by a swelling, which gradually increases and assumes a mound-shape of mixed grayish color, so that the form of the disk is lost, and can be inferred only by the convergence of the vessels. The arteries, often smaller than normal, are partly concealed, while the veins are dark, tortuous, distended, and dip into the infiltrated tissue. In many cases hemorrhages are found upon the swollen papilla or in its immediate neighborhood; they may be narrow and flame-shaped, or large, elongated patches of blood. This swelling of the disk, according to the variety of papillitis

FIG. 247.



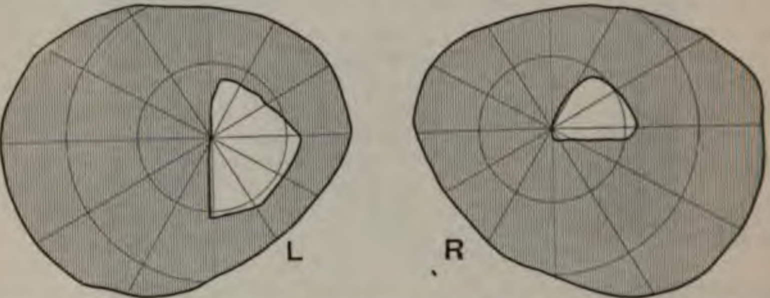
Concentric contraction of the fields of vision. The outer boundary marks the limits of the normal fields; the shading indicates where the vision was lost. (From a case of optic neuritis, caused by tumor of the temporal lobe, studied by the author while under the care of Dr. H. C. Wood.)

which is present, varies from 2 to 9 dioptres, or in other words, an elevation of 0.5 to 3 mm. (Fig. 2, Plate V.).

Vision may be entirely unaffected—a most important point, and, moreover, may remain unaffected until late in the disease. Occasionally it is lost with suddenness. Early blindness is more common in tumors of the cerebellum than with other intra-cranial causes of optic neuritis.[1]

The field of vision may be normal in its periphery, concentrically contracted, or there may be hemianopsia, if the cause of the papillitis is so situated as to produce this phenomenon. (Fig. 247 and 248.)

FIG. 248.



Left hemianopsia, with marked reduction of the remaining half fields, greatest upon the right side, caused by hemorrhagic softening the right cuneus from the same case as Fig. 241—a later condition of the visual fields.

Color perception is often defective, and that of red and green is usually lost before the others. There are no external appearances indicative of papillitis, and no pupillary phenomena suggestive of its presence, unless there is blindness, when the light reaction of the iris is lost.

The Diagnosis depends upon ophthalmoscopic examination. Neuritis may be the result of hypermetropic astigmatism, and often amounts to a swelling of 1½ dioptres; if the prominence is 2 dioptres or over, it betokens intra-orbital or intra-cranial disease (Norris).

Course and Prognosis. Papillitis may begin as an ordinary congestion associated with œdema, and gradually go on to a fully developed condition, or subside before this stage is reached; sometimes it originates with great rapidity; it may be slow in its progress and development.

During the stage of subsidence the elevation decreases, the veins grow less distended, the grayish-red tint becomes uniformly gray, and gradually the margins of the disk appear. Finally, both sets of vessels contract, and often much white tissue appears along their margins, while spots of degeneration in the fundus indicate the position of former hemorhages. An uncommon phenomenon is a second attack of optic neuritis, implanted upon a nerve-head which has been in the state of post-papillitic atrophy for many months. The author has observed and reported, with Dr. A. G. Thomson, one such case.[2]

The prognosis depends entirely upon the cause. It may be good when the neuritis has arisen under the influence of a removable intra-cranial deposit, for example, a gumma; or perfectly hopeless, the nerve passing into atrophy, for instance, in a sarcoma or glioma of the cerebellum.

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[1] For the cause of this, consult Dercum, Journ. of Nervous and Mental Diseases, Oct. 1893, p. 675.

[2] Archives of Ophthalmology, 1895, vol. xxiv. No. 2.

James Taylor,[1] after alluding to the observations of Horsley, Bruns, and Erb, that there may be subsidence of optic neuritis after operation undertaken with a view to the removal of cerebral tumor, even when the tumor was not removed, describes additional cases of decrease and disappearance of papillitis. Under exactly similar circumstances, in two instances no operation except the trephining had been performed. W. W. Keen has seen optic neuritis subside after removal of brain tumors, and the author has made the same observation. He has also, however, noted marked increase of neuritis subsequent to trephining.

Pathology and Pathological Anatomy. The swelling of the disk is often visible when the backs of the eyes which have been removed are examined. So, also, a pear-shaped distention of the optic nerve-sheath is not infrequently present.

Microscopic changes in the nerve-head vary according to the stage at which the examination is made, from œdematous nerve-fibres to marked degeneration of these tissues, with aggregations of fatty change, granular corpuscles, and colloid changes. The nerve and its head are crowded with inflammatory corpuscles which are deeply stained with carmine, and, as has been pointed out, these changes, both within the nerve and its sheath, may often be traced through the entire length of the nerve. The bloodvessels, large and small, in the nerve-head itself are distended, but do not show signs of compression at the lamina. (Gowers.)

Treatment. This should consist of remedies suited to the disease which has caused the neuritis, especially mercury, iodide and bromide of potassium, salicylic acid, pilocarpine, and, during the stages of atrophy, strychnine. Incision of the swollen sheath has been practised.

ORBITAL OPTIC NEURITIS. Although not strictly pertinent to the neurological aspects of the diseases of the optic nerve, it seems proper that a few words should be said in regard to that form of optic neuritis in which the inflammation occurs in the orbital part of the optic nerve, and which is therefore known as orbital optic neuritis, or retro-bulbar neuritis.

Varieties of Orbital Optic Neuritis. Two varieties are described by systematic writers:

1. *Acute Retro-bulbar Neuritis*, in which the appearances are not unlike those described with hyperæmia of the nerve-head. Direct vision is greatly impaired owing to the presence of a large central scotoma, which may be either positive or negative, that is, a defect appreciated by the patient or one of which he is unconscious, the latter usually being a color scotoma in which red and green are not recognized as such. There may be complete restoration of vision after this affection, which is either monocular or binocular, a long interval sometimes occurring between the involvement of the first and the second eye.

Etiology. The disease is usually attributed to certain toxic agents, for example, lead, alcohol, nicotine, etc., to menstrual disturbances, rheumatism, the exanthemata, diphtheria, and other acute infectious diseases, and to overwork.

2. *Chronic Retro-bulbar Neuritis.* This variety is generally known under the name of *tobacco amblyopia* or *toxic amblyopia.* The ophthalmoscopic lesions may be absent or the disk is discolored, and in the latter stages a triangular-shaped patch of atrophy appears in its lower and outer part. Occasionally general atrophy supervenes.

There is diminution of direct vision, particularly in bright light. The peripheral boundaries of the field of vision are normal, but there is a central scotoma, usually oval in shape, situated between the fixation point and the blind spot, in which area the perception of red and green is lost. Sometimes the recognition of other colors also fails, and in bad cases the scotoma may become absolute.[1]

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[1] Ophthalmic Review, June, 1894.

Etiology. Most of the cases of chronic retro-bulbar neuritis are the result of the abuse of tobacco. Generally the patients have also consumed alcohol in one form or another, but there is sufficient evidence to show that pure tobacco amblyopia exists. The disease is nearly always bilateral and uncommon before the thirty-fifth year of life. The same character of scotoma may be caused by poisoning with lead, carbon bisulphide, iodoform, cannabis indica, quinine, opium, and has been attributed to syphilis, diabetes, rheumatism, gout, and influenza. Analogous scotomas are occasionally seen in patients with locomotor ataxia (progressive atrophy, with scotoma), but it is difficult in all of these cases to separate the influence of tobacco and alcohol,[2] and also in disseminated sclerosis. (Uhthoff.) (See Fig. 252.)

Pathology. Retro-bulbar neuritis is a form of peripheral neuritis, and is caused by an interstitial inflammation of the papillo-macular fibres of the optic nerve (see page 752). Therefore, the quadrant-shaped patch of atrophy which is visible in the latter stages represents the area of degeneration which has attacked this bundle, which in the nerve-head consists of a triangle with its base in the lower and outer part and its apex in the central vessels.

Treatment. Both acute and chronic retro-bulbar neuritis must be treated by removing the cause—toxic agent or general disease—and this alone will suffice to cure many of the cases. Other remedies that suggest themselves are free diaphoresis, inhalation of nitrite of amyl, iodide of potassium, and particularly the free exhibition of strychnine, suitably administered by hypodermatic injections.

ATROPHY OF THE OPTIC NERVE. The general term *atrophy of the optic nerve*, usually abbreviated into *optic atrophy*, includes various types and grades of degeneration of the fibres of the optic nerve.

VARIETIES OF ATROPHY OF THF OPTIC NERVE. (*a*) *Primary atrophy*, usually designated gray, progressive, spinal, or tabetic atrophy; (*b*) *secondary atrophy*, or that form which results from pressure more or less directly applied to the chiasm or optic nerve itself; and (*c*) *consecutive atrophy*, also called neuritic or post-papillitic atrophy, because it follows an inflammation of the nerve. *Retinal* and *choroiditic atrophy* are forms of consecutive optic nerve degeneration, caused, as their names imply, by pre-existing disease of the retina and choroid.

Etiology of Primary Atrophy of the Optic Nerve. The causes of primary atrophy may be divided as follows:

(1.) SPINAL LESIONS. (*a*) *Locomotor Ataxia.* It is difficult exactly to state the frequency of essential optic nerve atrophy in locomotor ataxia. The average of a number of observations gathered by Berger[3] gives 33.7 per cent. of atrophies. Among his own 106 carefully studied cases there was marked atrophy in 44, and 7 cases of amblyopia without ophthalmoscopic changes, or, in other words, 46.7 per cent. of disturbance of the optic nerve.[4]

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[1] For the detection of scotomata or blind spots in the field of vision, the patient is placed before the perimeter, or a blackboard, precisely as if the visual field was to be mapped out. Small test-objects, white or colored, ¼ of a centimetre square, are employed, which are moved in different directions from the spot which the eye under observation attentively fixes, and the point marked where the object begins to disappear or change its color.

[2] There is also stationary atrophy with scotoma seen in men after thirty years of age, and of hereditary tendency.

[3] Archives of Ophthalmology, vol. xix. p. 429.

[4] The following figures, showing the diversity of opinion as to this point, may be quoted, and are taken partly from Berger's paper: Erb, 3 cases of optic atrophy in 60 patients; Topinard, 49 to 102;

The differences depend largely upon the point of view. Careful examinations—such, for example, as William F. Norris has made—would probably show that Berger's average is by no means too high. In a certain number of cases one eye is affected before and more than its fellow.

(*b*) *Friedreich's Ataxia.* According to Griffith,[1] atrophy of the optic nerve is very rare in this disease. In 38 cases in which ophthalmoscopic examinations were recorded, white disks were noted in one (Power) and partial atrophy in another (Seguin). Sinkler and Oliver, however, who have made really careful investigations of the optic nerves under these circumstances, have found similar changes to those which occur in the earlier stages of tabes, and this has been the experience of the author in one or two examinations, although the degeneration, if present, was incipient.

(*c*) *Lateral Sclerosis.* Atrophy of the optic nerve is uncommon, Gowers having seen it only once in an uncomplicated case. This corresponds with the experience of the author in the Infirmary for Nervous Diseases and the Philadelphia Hospital, but it is desirable that more accurate ocular examinations in this disease should be made than those which are recorded.

In amyotrophic lateral sclerosis some changes in the optic nerve have occasionally been observed. Petelsohn,[2] for example, reports a case of optic nerve atrophy.

Optic atrophy has also been noted in chronic myelitis, paralysis agitans, spastic spinal palsy and bulbar palsy; but Gowers[3] has not observed it in progressive muscular atrophy.

(*d*) *Insular Sclerosis.* The most complete investigation of the ocular disturbances which accompany multiple sclerosis has been made by Uhthoff.[4] The analysis includes 67 males and 33 females, between the ages of ten and seventy years. Pronounced atrophy was present in 3 per cent., incomplete atrophy of the whole disk in 19 per cent., and partial atrophy of the temporal half of the disk, the inner half being normal, or, in other words, appearances similar to those seen in toxic amblyopia, was present in 18 per cent. Forty-eight of the 100 cases had normal ophthalmoscopic appearances, but in 5 of these there were disturbances of vision. Central scotomas were found in 15 cases. Therefore, the amblyopia of multiple sclerosis, as compared with other diseases of the optic nerve, most resembles non-toxic retro-bulbar neuritis. The difference, however, consists in the fact that the pallor of the disk in multiple sclerosis does not necessarily imply the presence of a central scotoma, while in ordinary retro-bulbar neuritis the quadrant-shaped patch of atrophy practically always means a scotoma, usually between the fixing point and the blind spot. The difference in the pathology of the two affections accounts for this state of affairs; the degeneration is probably irregularly scattered through the nerve in sclerosis, while there is a regular degeneration of the axial fibres in retro-bulbar neuritis. Buzzard[5] agrees with Uhthoff, that, excepting tumors and tubercular meningitis, there is no disease of the nervous system so frequently accompanied by ophthalmoscopic changes as disseminated sclerosis, and finds pallor of the disks in 43 per cent. of his cases.

Cyon, 40 to 50; Leber's estimate is 26 per cent.; Michel, 12 to 13 per cent.; Althaus, one-sixth of the cases; Moeli, 13.5 per cent.; and Gowers, 20 per cent.

Buzzard finds optic atrophy in 15.3 per cent. of his cases, and Nettleship (Trans. Oph. Soc. United Kingdom, vol. iii. p. 251), among 76 cases of progressive atrophy, found 38 the subjects of locomotor ataxia, whilst 20 others suffered from mixed symptoms of spinal and cerebral disease and from other forms of chronic spinal-cord disease, not ataxia.

[1] "A Contribution to the Study of Friedreich's Ataxia." Transactions of the College of Physicians of Philadelphia, February 1, 1888.

[2] Centralblatt f. praktische Augenheilkunde, 1886, p. 108.

[3] Trans. Oph. Soc. United Kingdom, vol. iii. p. 197.

[4] For a valuable abstract of this elaborate work, consult the Ophthalmic Review, vol. ix., 1890.

[5] Brit. Med. Journ., Oct. 7, 1893.

(*e*) *Injuries of the Spinal Cord.* In ordinary injuries of the spine there are no lesions in the optic disks, and in injuries below the level of the third dorsal nerve-roots the ophthalmosopic changes do not arise (Thorburn). As is well known, Clifford Allbutt has noted changes in chronic cases, an observation that has been challenged, and properly, by Thorburn,[1] who, in 7 cases among 21 severe injuries above the second dorsal nerve, found that 3 of them showed want of definition of the disk and slight distention of the retinal veins, while in 6 cases, which survived the accident for long periods, no subjective symptoms arose, so that atrophy of the optic nerve probably never took place.

(2.) PARETIC DEMENTIA. Changes analogous to those seen in locomotor ataxia are common, not only in the form of essential atrophy of the disks, but as a discoloration of the papilla and surrounding fibre layer of the retina. The latter phenomenon has been observed by Uhthoff[2] 32 times among 150 cases, *i. e.*, in 28 per cent. of the cases. The same observer noted atrophy of the disk in 8.04 per cent. of the cases, and abnormal color of its surface in 14 per cent. In the third stage, Oliver reports decided and unequal semi-atrophy, especially on the temporal side, and in the earlier stages dirty, gray-red degeneration of the disks. The percentage of atrophy among 167 cases recorded by various observers (Galezowski, Boy, Yehn) is 7.2, while Klein found the same condition in 5 per cent., and Siemerling in 6 per cent. of his cases. Atrophy, preceded by scotoma, has been recorded by Hirschberg.

(3.) MISCELLANEOUS CAUSES. Among the miscellaneous causes of essential atrophy of the optic nerve, the following are usually enumerated: Cold, depraved nutrition, syphilis, menstrual disturbances, venereal excesses, malaria, diabetes, the abuse of the narcotics, (preceded by inflammation of the axial fibres of the optic nerve), toxic doses of quinine, and probably of other drugs, *e. g.*, iodoform.

Etiology of Secondary Atrophy of the Optic Nerve. As Gowers points out, secondary atrophy of the optic nerve may result from a lesion of the optic centres or of the fibres of the nerve itself. Quoting Ferrier, he shows that a cortical lesion in the brain about the supra-marginal gyrus may entail loss of sight of the opposite eye.

Atrophy without precedent neuritis, however, usually results from direct pressure, for example, on the chiasm, the tracts, or the nerve itself. In the first two situations this may be produced by tumors, aneurisms, exostoses, or by the distention of the third ventricle, as in chronic hydrocephalus. It is said that a meningitis occasionally produces a secondary atrophy; that is, one without pre-existing papillitis.

A not uncommon cause is injury to the optic foramen, or pressure due to periostosis, syphilitic deposits, caries, and necrosis. A blow on the head, especially in the neighborhood of the supra-orbital foramen, may, it is said, occasion atrophy, but there is good reason to believe that the degeneration is the result of periostitis, rather than of an uncomplicated injury.

Etiology of Consecutive Atrophy of the Optic Nerve is chiefly of ophthalmological interest, except in so far as it is of post-neuritic origin. The relation of papillitis to the production of atrophy has been referred to in the section on Prognosis.

SIGNIFICANCE OF ATROPHY OF THE OPTIC NERVE. Probably one-half of the cases of *primary* atrophy of the optic nerve are associated with spinal lesions, and certainly a very large proportion of the cases are forerunners of locomotor ataxia. Buzzard, however, finds atrophy most frequently associated with insular or disseminated sclerosis; next frequently with fasciculated sclerosis of the posterior columns of the spinal cord (tabes and general paralysis of the insane), and least often with fasciculated sclerosis of the lateral columns.

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[1] "A Contribution to the Study of the Spinal Cord," 1889.

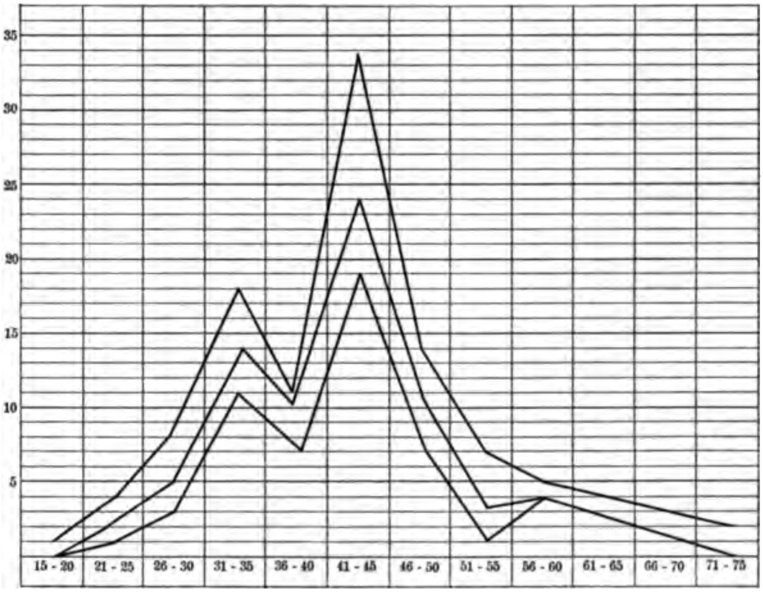
[2] Bericht der Ophthalmologischen Gesellschaft. Heidelberg, 1883, p. 189.

Practically all authors are in accord that optic nerve atrophy usually develops in the beginning of tabes, often in the pre-ataxic stage, although it may also arise late in the course of the disease. Berger points out that cases with palsy of the ocular muscles are more disposed to atrophy than those without such association.

Sometimes the interval between the development of the ocular and spinal symptoms is a long one. Thus, Förster has seen optic atrophy precede the development of other symptoms by a period of three years, Charcot by ten years, and Gowers fifteen and twenty years. Buzzard also has observed blindness and lightning pains fifteen years before the ataxic symptoms developed. Norris[1] calls particular attention to early gray degeneration of the nerve, while there is full acuity of central vision and only slight contraction of the field for form and color, as a forerunner of spinal cord degeneration. These cases, moreover, had both diminished, lost and increased knee-jerks.

The following chart from Berger gives a good idea of the frequency of tabes at the various periods of life, of severe ocular symptoms and of atrophy of the optic nerve:

FIG. 249.



Upper curve, frequency of tabes; middle curve, frequency of severe ocular symptoms; lower curve, frequency of atrophy of the optic nerve.

As a localizing symptom, optic nerve atrophy is often of little value in the absence of other phenomena, particularly changes in the visual field, but, taken into consideration with these, with pupillary changes, and with the general symptoms, it assumes great importance, while its presence, even in incipient degree, may often decide between organic and so-called functional disorders; for example, hysteria and disseminated sclerosis. The gray degeneration of paretic dementia, frequent late in the disease, may also precede the mental symptoms by a long period of time.

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[1] "On the Association of Gray Degeneration of the Optic Nerves with Abnormal Patellar Tendon Reflexes." Transactions of the American Ophthalmological Society, July, 1885.

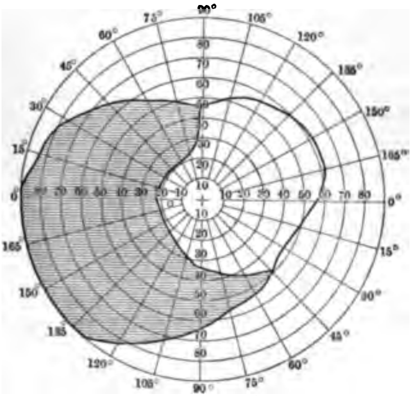
The significence of secondary and post-papillitic atrophy has been referred to on page 762.

Symptomatology. The clinical types of optic nerve atrophy present different ophthalmoscopic appearances. In general the color of the nerve-head varies from a slight gray to an entirely white hue, with intermediate pure gray and greenish-gray tints. Often the disk is grayish-red on its surface, but decidedly gray in its deeper layers. The clinical types of optic nerve atrophy present a variety of ophthalmoscopic appearances. (See section on Diagnosis, p. 768.)

The acuity of central vision varies from a slight depreciation to complete blindness.

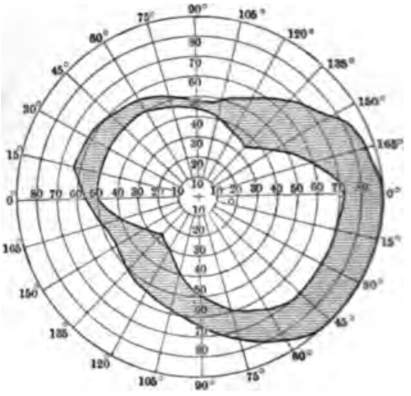
The changes in the field of vision consist of contraction, irregular limitations in the form of re-entering angles (Fig. 251), quadrant-shaped defects, loss of one-half of the field (hemianopsia) (Fig. 250), and abnormal blind spots, or scotomas (Fig. 252). In disseminated sclerosis with ocular disturbances, Uhthoff describes four varieties of the visual field—central scotoma, with uncontracted field, central scotoma with contraction; irregular peripheral contraction, with relatively good central vision; and concentric contraction analogous to hysteric cases. Normally, in the visual field, between the outer limit of white perception and the outer limit of color perception, colors appear as light gray, white, or black. The same condition obtains in the contracted fields of tabes and disseminated sclerosis. Head has found the same phenomenon in the hysterical type of disseminated sclerosis, but it is not present in the true hysteric field (Fig. 254).

FIG. 250.



Visual field of a case of ataxia with atrophy of the disks, showing loss of temporal half. Perception of red and green was lost.

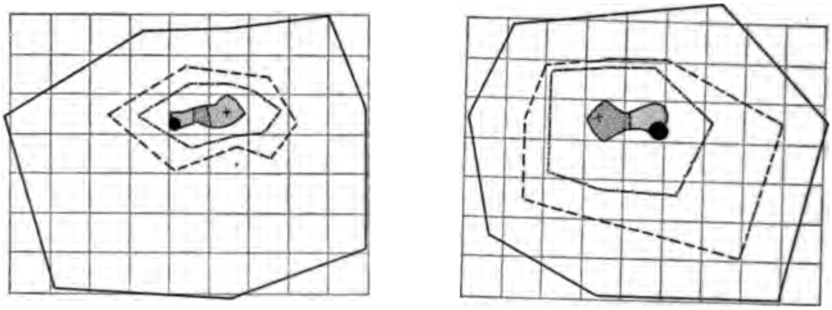
FIG. 251.



Right visual field from a case of ataxia with atrophy of disk, showing contraction and re-entering angles.

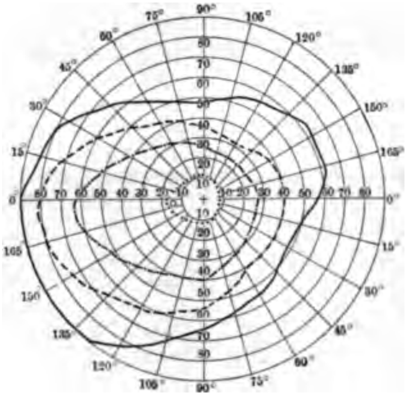
In tabes there may be ophthalmoscopic atrophy of the disk, with good color perception and with normal fields, or with normal central vision and color perception, but with contracted color-fields, especially green (Fig. 253), or with both form and color-fields restricted. Usually vision, color perception and fields of vision are affected simultaneously. A variety of forms of contraction

FIG. 252.



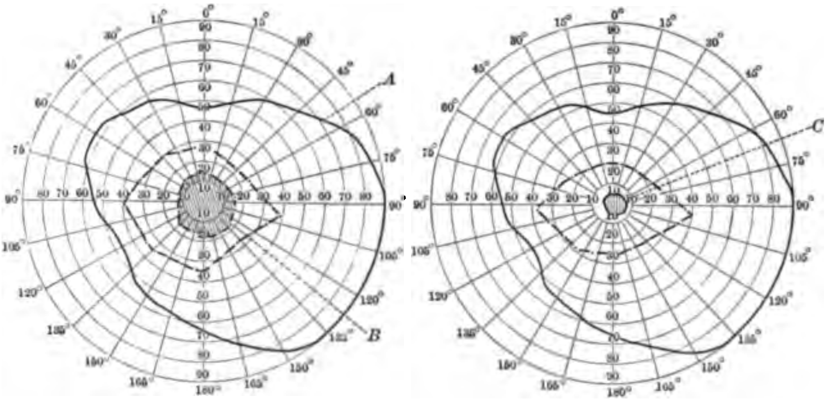
Visual fields of a case of ataxia (mapped on board), showing central scotomas. Patient also smoked.

FIG. 253.



Tabes, central vision normal, optic disk slightly discolored, form and blue field normal, red slightly contracted, green markedly restricted. White ——. Blue – – – – Red —·—·—·— Green . . . . .

FIG. 254.



From a case of disseminated sclerosis with optic atrophy after hysterical symptoms for many years. A, the white field. B, the shaded patch, represents the area over which red is seen as such; the outer line represents the limit, within which red on a white ground appears as black. C, the shaded patch, represents the area, over which green is seen as such; the outer line represents the limit within which green on a back ground appears as white. (After BUZZARD.)

of the field are seen. Berger concludes that, although contraction of the outer part of the visual field is most frequent, it does not occur in this position in more than half of the cases. Sector-formed defects are common. Defect in color vision may be marked before the form-field is at all affected. The appreciation of green is first diminished, then that of red, and later blue and yellow; finally there may be complete achromatopsia. The color fields are affected in the same order. Sometimes the red field is the first to contract.

Pathology and Pathological Anatomy. The anatomical changes in the optic nerve which has undergone atrophy depend upon the stage and the cause. If it follows an interstitial inflammation or pressure, the fibrous tissue of the septa is thickened, the fibres reduced in number, atrophied, and destroyed. This atrophy may be so great that the whole nerve is reduced to a fibrous cord.

When there is gray degeneration the nerve is rather gelatinous in appearance and shrunken. The fibres first lose their medullary sheath, later become granular, and often in the later stages fatty particles, compound granular corpuscles, and amyloid bodies are present.

In post-papillitic atrophy, in addition to the atrophic changes, nuclei and nucleated connective-tissue fibres may be found, with traces of the nerve fasciculi. In certain forms of atrophy—as, for example, that found with multiple sclerosis—according to Uhthoff, the changes are midway between tabetic atrophy and that which follows interrupted nerve conduction on the one hand and post-neuritic atrophy on the other, the changes sometimes resembling those of simple atrophy and sometimes those of interstitial neuritis. (See abstract, *loc. cit.*)

Diagnosis. It is essential not to mistake an ordinary pallor of the optic disk for atrophy; therefore, the importance of associating with ophthalmoscopic examination accurate investigation of the field of vision, particularly the field for colors, and the direct color-sense.

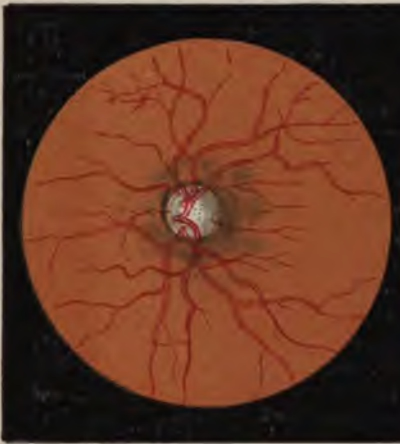
In primary atrophy the outline of the disk is sharp, its color gray-red or gray, the lamina usually visible and well mottled, and the vessels in the earlier stages uncontracted and free from white lines along their margins. (See Plate VI., Fig. 1.)

In consecutive atrophy, on the other hand, the margin of the disk is hazy, the color white, the white spot usually filled in, the arteries contracted, the veins often large and tortuous, with white lines bordering their margins. (See Plate VI., Fig. 2.) In the retinitic and choroiditic atrophies the disk is often yellowish and waxy in appearance, and both sets of vessels markedly narrowed. (See Plate VI., Fig. 3.)

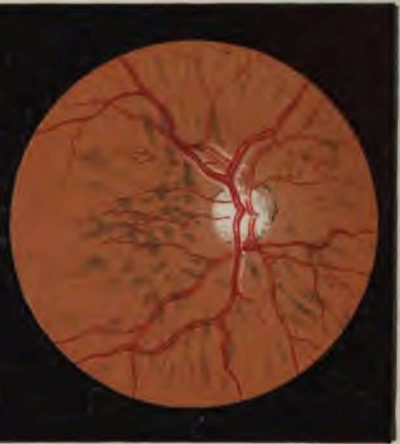
In secondary atrophy the color of the disk resembles that of the spinal form of the disease, but more often is distinctly white; both veins and arteries are contracted, the veins usually proportionately more so than the arteries.

Prognosis. Optic nerve atrophy is always slow in its course, and may last for months or years before total blindness is reached. The prognosis of a post-papillitic atrophy depends upon the amount of inflammation and the consequent shrinking of the tissues. Tabetic, or, as it is often denominated, progressive atrophy, affords an unfavorable prognosis, visual acuity and the field of vision gradually deteriorating; but much difference of opinion has been expressed in regard to its course. Berger states that Leber believed, as a rule, one or two years were required for the atrophy to result in total blindness, and quotes Gowers, that when atrophy develops early in the course of the tabes blindness follows more quickly than when it is a late phenomenon. In his own experience the quickest development of blindness was two months, and the slowest fifteen years in a syphilitic patient, and seventeen in a non-syphilitic patient. The average development of blindness in cases of optic

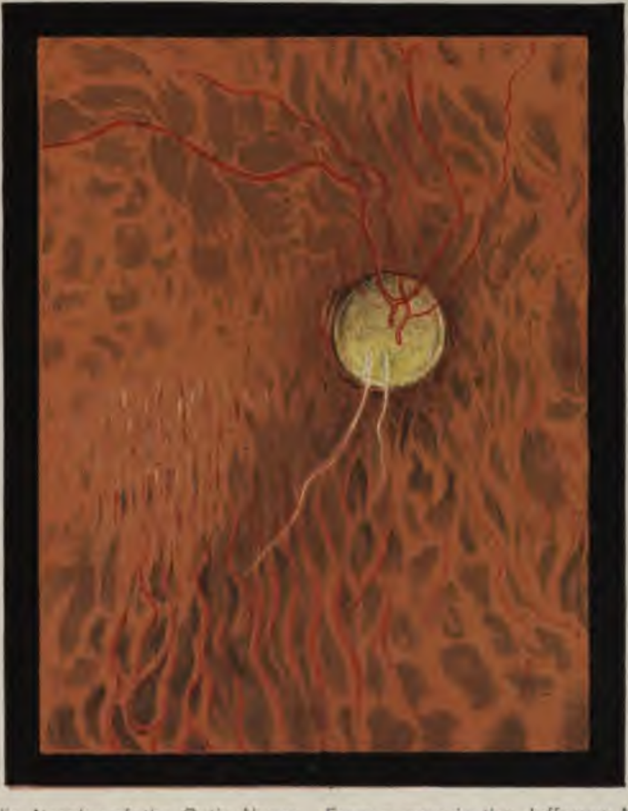
PLATE VI.



Primary Atrophy of Optic Nerve (Spinal Atrophy). Modified from Haab.



Post-papillitic or Consecutive Atrophy of the Optic Nerve. Modified from Juler.



Embolic Atrophy of the Optic Nerve. From a case in the Jefferson Medical College Hospital.

atrophy complicated with ocular palsies was 3.4 years; without ocular palsies, 3 years. As most of the optic atrophies develop in the pre-ataxic stage, the danger of this complication is lessened if this stage is passed. Optic atrophy is usually somewhat slower in syphilitic than in non-syphilitic patients; in a case under the care of J. K. Mitchell and the author the vision sank during twenty-eight months from $\frac{2}{3}$ to $\frac{1}{80}$ of normal.

Treatment. If there is reason to suspect syphilis the usual remedies are indicated, especially a prolonged course of bichloride of mercury; but it is worse than useless in advanced atrophy, even if the patient is syphilitic, provided there are no other indications, to depress his nutrition with a long course of mercury which can have no possible influence on nerve-fibres long since hopelessly degenerated. Nitrate of silver has sometimes been used with advantage, and the visual field of tabetic atrophy is said to have been enlarged by suspension.[1] Other remedies, according to the cause, are iron, arsenic, phosphorus, and, if a recent report of Valude's may be credited, antipyrine, although what its physiological action can be under such circumstances it is difficult to conceive. The most generally useful remedy, for the introduction of which we are indebted to Nagel, is strychnine. This sometimes has a very remarkable influence. It should be pushed to its full physiological effect, and it acts usually more promptly and more efficiently by the hypodermic method. It is well to associate with it nitroglycerin, which may be alternated with digitalis, both of them improving the nutrition of the nerve by their influence upon the peripheral capillaries. Santonin has been used, but, there is no good reason to believe, with any value. The same is true of injections of animal extracts after the manner of Brown-Séquard, and also, probably of suspension.[2] Competent observers have reported good results after the use of galvanism, and particularly after the method of voltaic alternatives. The author's results have been unsatisfactory.[3]

HEMIANOPSIA.[4] A lesion situated at the optic chiasm, in the visual tract, or at its ultimate destination in the cuneus, produces, so far as the eyes are concerned, a defect of vision characterized by an obscuration, usually in each eye, of one-half or the visual field. To this defect the name hemianopsia is applied.

Varieties of Hemianopsia. Hemianopsia is divided into horizontal, in which the dividing line between the darkened and preserved fields is horizontal; and vertical, in which the dividing line is vertical.

1. *Horizontal, or Altitudinal Hemianopsia* may be inferior or superior, both lower or both upper half fields being wanting. In addition to diseases of the eye, such a condition could arise under the influence of a lesion so situated as to press upon the upper or lower part of the chiasm, or downward upon one optic tract, or upon the lower or upper part of both optic nerves. A double lesion in front of the chiasm may produce loss of the upper half of the field in one eye and of the lower half of the field in the other.

2. *Vertical Hemianopsia.* This is subdivided into several varieties:

(*a*) *Bitemporal Hemianopsia* (peripheral), in which both temporal fields are wanting. This can only be caused by a lesion (tumor, aneurism or fracture, etc.) involving the crossing fibres of both optic tracts in the middle of the chiasm, and is a symptom perfectly characteristic of disease in this region (Fig. 255).

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[1] University Med. Magazine, vol. ii., p. 39.

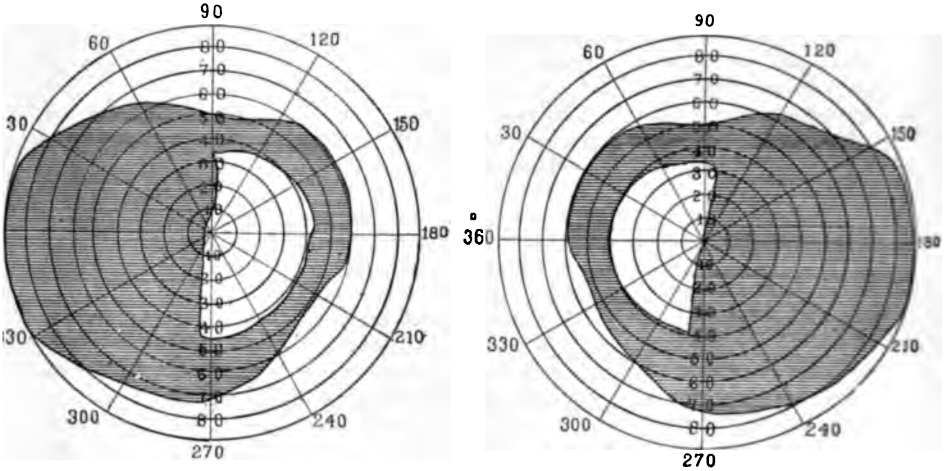
[2] See an article by the author on "Medicinal Ocular Therapeutics," Therapeutic Gazette, August, 1894.

[3] For a review of the literature of this subject and the results obtained, the reader is referred to an editorial by the author in the Therapeutic Gazette, April, 1893.

[4] The terms hemiopia and hemianopsia are sometimes used synonymously. Hemiopia, however, as Seguin points out, signifies loss in the perceptive power of one-half of the retina, while hemianopsia means obscuration of one-half of the visual field. Other names which are used are hemianopia and hemiablepsia.

(*b*) *Binasal Hemianopsia,* in which both nasal fields are wanting, is extremely rare. It necessitates a lesion on both sides of the chiasm, or one on the outer side of each optic nerve, which disables the direct fibres.

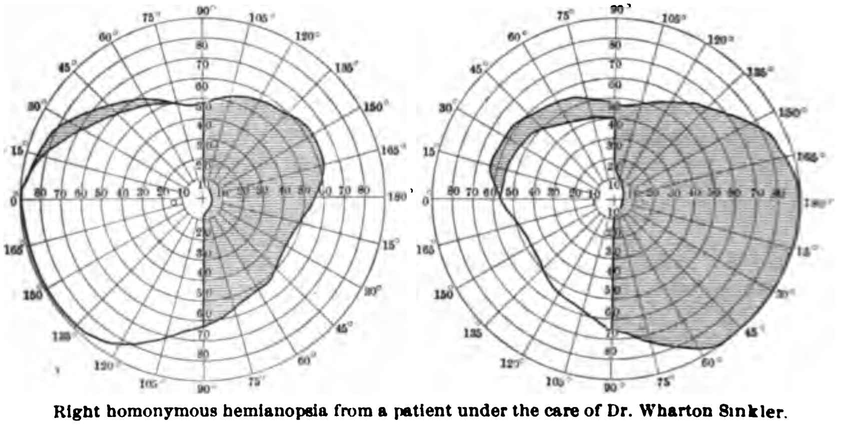
FIG. 255.



Bitemporal hemianopsia from a case of akromegaly originally under the care of Dr. H. C. Wood and later studied by Dr. F. A. Packard. Eyes examined in 1885 by the author, and above fields found.

(*c*) *Homonymous Hemianopsia* (central), in which the corresponding half of the visual field in each eye is wanting. Thus, both right or both left fields are darkened, in the former case indicating loss of the function of the left half of each retina, and designated *right homonymous lateral hemianopsia* (Fig. 256), and in the latter case indicating loss of function of the right half of each retina, and designated *left homonymous lateral hemianopsia* (Fig. 257).

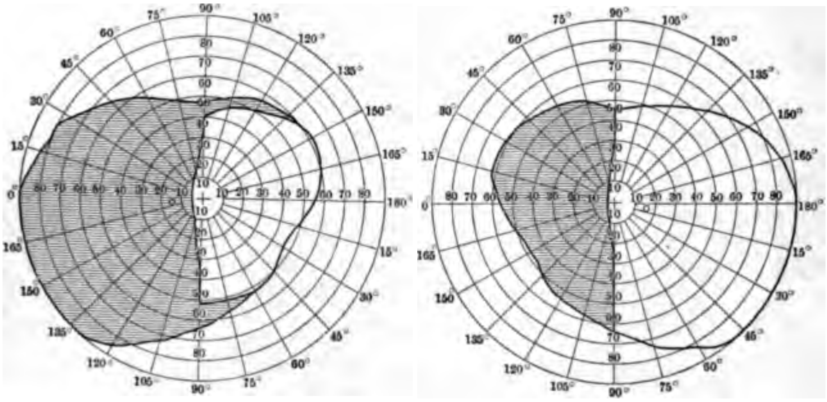
FIG. 256.



Right homonymous hemianopsia from a patient under the care of Dr. Wharton Sinkler.

This form of hemianopsia is caused by a lesion situated in the occipital lobe, the evidence being that the absolute optical centre chiefly occupies the cortex of the cuneus and of the superior occipital convolution, and also, at least so far as color-sense is concerned, the posterior part of the superior and inferior occipito-temporal convolutions. It is further produced by a lesion situated in the optic radiations, the internal capsule, primary optic centres, or the optic tract; in other words, by any lesion which breaks the continuity of the visual tract posterior to the optic chiasm.

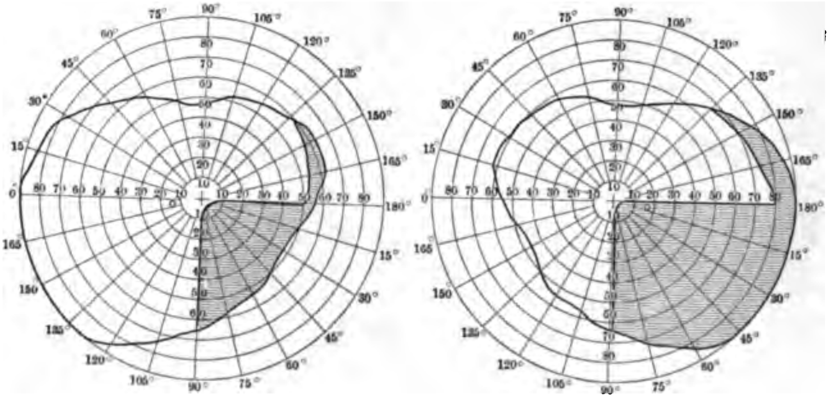
FIG. 257.



Left homonymous hemianopsia from a case of gunshot wound, with suspected lesion of the right cuneus, from a case under the care of S. Weir Mitchell, at the Infirmary for Nervous Diseases.

The lesion is in the optic tracts, or in the primary optic centres, and interferes with the sensory-motor arc of the pupil if Wernicke's symptom (page 802) is present; it is further on in the visual pathway if Wernicke's symptom is absent.

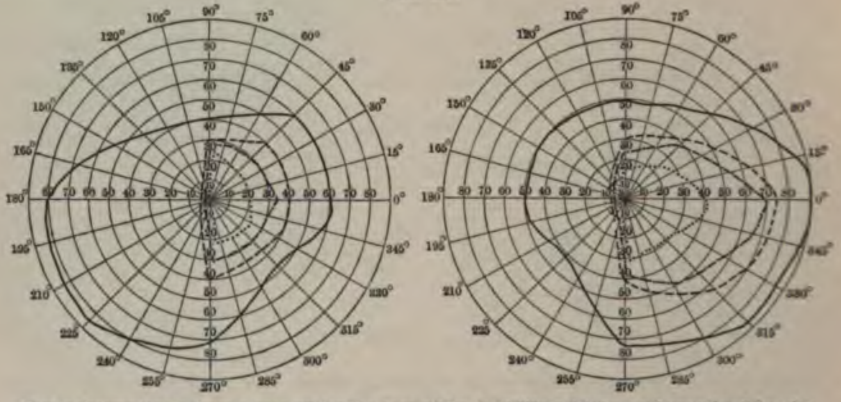
FIG. 258.



Quadrant anopsia, from a case in the Philadelphia Hospital. Probably softening in cuneus.

Homonymous lateral hemianopsia is the most common variety of hemianopsia. It may be *complete*, that is, the entire half of each field is wanting, or *incomplete*, that is, a portion of each half field is wanting, the defect usually being in the form of a quadrant (Fig. 258). The hemianopsia may be *absolute*, that is all the three functions of sight, perception of light, of form and of color, are wanting, or it may be *relative*, that is, perception of color, or perception of color and form is wanting in the deficient area of the field, but light-sense is preserved. In rare cases the half-defect is present for colors alone, and is known under the name of *homonymous hemiachromatopsia*. Under these circumstances the defect is situated in the cortex of the occipital lobe (Fig. 259).

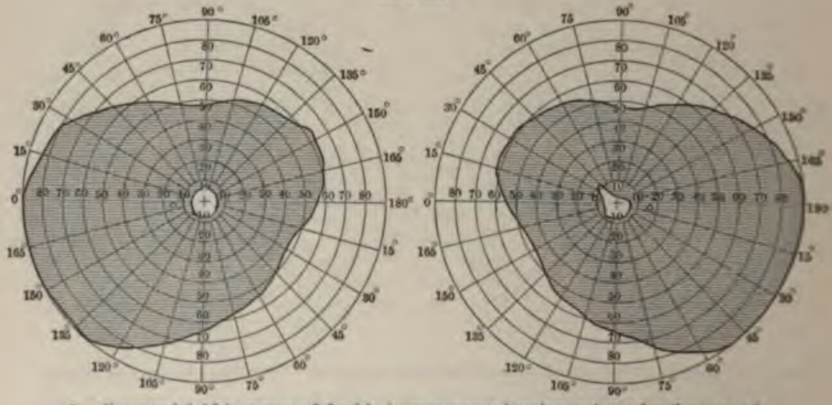
FIG. 259.



Hemiachromatopsia from a case under the care of Dr. J. William White, and examined by the author. White, ——. Blue, - - - -. Red, —. —. —. —. Green, ...

Given a case of homonymous hemianopsia, the lesion is on the opposite side of the darkened fields. If the preserved fields are accompanied by a concentric contraction, the smaller half field will be in the eye opposite the lesion. Contraction of the preserved half field is most common with lesions of the cortex, but it may also occur in lesions of the tract. If the hemianopsia is relative, the lesion must be in the cortex; elsewhere it produces absolute hemianopsia. However, cortical lesions are not excluded by absolute hemianopsia.

FIG. 260.



Small central field in a case of double homonymous hemianopsia under the care of Dr. T. D. Dunn, of West Chester.

The dividing line in hemianopsia may exactly cut the fixing point, but it usually passes around this point, leaving it in the area of preserved vision.

Again, it may fail to correspond with the perpendicular for some distance, or may assume an oblique or irregular direction. These peculiarities are explained (Schmidt-Rimpler, Gowers), by "individual variations in the expansion of the retinal fibres of the tractus opticus."

That the macula lutea is specially represented in the visual centre has been proven by cases of double homonymous hemianopsia, in which a small central field is preserved in each eye, indicating that the region in the cortical visual centre which supplies the macula lutea has not been destroyed. (See Fig. 260.) It is therefore also evident that destruction of this centre alone would be manifested in the visual field by a central scotoma.

The evidence at this time is constantly accumulating to show that there is a correlation between the parts of the retina and the occipital lobe; for example in Dr. Hun's case of left quadrant hemianopsia, the lesion was strictly limited to the lower half of the cuneus, indicating that the upper right quadrant of each retina terminates in the lower half of the right cuneus.

For the more particular relations of hemianopsia to the localization of cerebral disease and to other symptoms, both direct and distant, the reader is referred to the paragraphs specially devoted to these topics.

DISEASES OF THE ABDUCENS, PATHETIC, AND OCULO-MOTOR NERVES.

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS. The oculo-motor, pathetic, and abducens, or third, fourth, and sixth in the list of cranial nerves, constitute the nerve-supply of the external ocular muscles, while the third division of the oculo-motor sends a branch to the ophthalmic ganglion, forming its motor root.[1]

The deep origin of the *third nerve* has been traced to a nucleus about 10 mm. in length in front of the anterior corpora quadrigemina, extending from the level of the posterior commissure to within a short distance of the nucleus of the fourth nerve. According to Alexander Bruce,[2] the following groups of cells can be distinguished: 1. An anterior or ventral group. 2. A posterior external group (dorsal nucleus of Edinger and Siemerling). 3. A median or central nucleus. 4. A posterior median or pale nucleus (Edinger-Westphal nucleus). 5. Superior nucleus (antero-lateral or small-celled nucleus of Darkschewitsch). All the segments of the oculo-motor nucleus are connected with the posterior longitudinal fasciculus. Bruce has not demonstrated a decussation of the root fibres, but believes that this is present, just as there is free commissural connection between the anterior nuclei of both sides.[3]

Some authors have attempted to bring the various segments of the nucleus in connection with the ocular muscles, for example, Hensen and Voelkers place the nuclei in the following order from above downward (before backward): (1) ciliary muscle; (2) sphincter iridis; (3) rectus internus; (4) rectus superior; (5) levator palpebræ superioris; (6) rectus inferior; (7) obliquus inferior. The evidence upon which this arrangement is based was obtained by experiments in animals. An analysis of twenty cases of partial third-nerve palsy has caused Allen Starr[4] to locate the relative situation of the

[1] For its relation to the iris and ciliary muscle, see p. 794.

[2] Illustrations of the Nerve Tracts of the Mid and Hind Brain, p. 17.

[3] It is impossible to review the contradictory evidence of examination of the oculo-motor nuclei. Perlia's scheme is considered more useful by many than the one given. A recent review of the subject is by Bernheimer in a monograph entitled, "Das Wurzelgebiet des Oculo-motorius beim Menschen," Wiesbaden, 1894.

[4] Familiar Forms of Nervous Disease, p. 108; also Journal of Nervous and Mental Disease, vol. xv. 1888, p. 301.

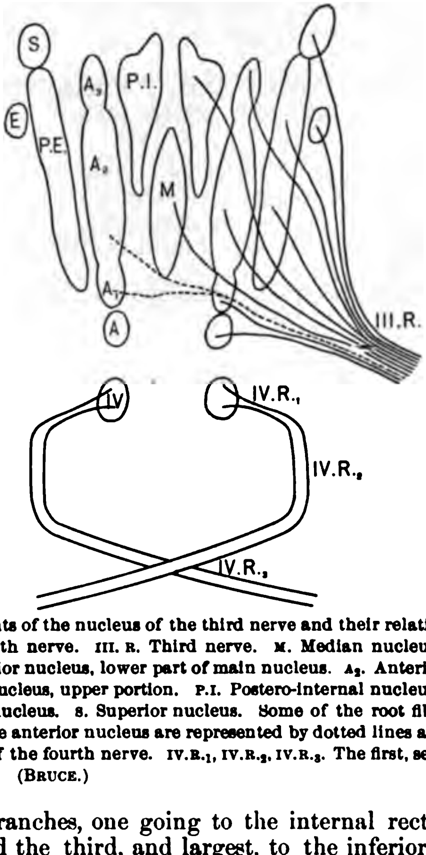
groups of cells governing the ocular muscles, and the following table constructed by Starr gives the situation of these groups. It practically agrees with the scheme of Kahler and Pick. In this he has named each group for the muscle which it controls.

ARRANGEMENT OF THE GROUP OF CELLS GOVERNING THE OCULAR MUSCLES.

Right.		*Left.*	
2. Sphincter iridis.	1. Ciliary muscle.	1. Ciliary muscle.	2. Sphincter iridis.
5. Levator palp. sup.	3. Rectus internus.	3. Rectus internus.	5. Levator palp. sup.
6. Rectus superior.			6. Rectus superior.
7. Obliquus infer.	4. Rectus inferior.	4. Rectus inferior.	7. Obliquus infer.
8. Obliquus super.			8. Obliquus super.
	9. Rectus externus.	9. Rectus externus.	

The nerve pierces the dura mater below the posterior clinoid process, passes along the outer wall of the cavernous sinus, and divides into two branches, which enter the orbit through the sphenoidal fissure. The superior division supplies the superior rectus and levator palpebræ; the inferior division separates into three branches, one going to the internal rectus, a second to the inferior rectus, and the third, and largest, to the inferior oblique. It also receives filaments from the cavernous plexus of the sympathetic, and its superior division is not infrequently connected with the ganglionic branch of the nasal nerve or third division of the fifth ophthalmic.

FIG. 261.



Scheme of the segments of the nucleus of the third nerve and their relations to each other and to the nucleus of the fourth nerve. III. R. Third nerve. M. Median nucleus A. Anterior nucleus, inferior part. A_1. Anterior nucleus, lower part of main nucleus. A_2. Anterior nucleus, intermediate portion. A_3. Anterior nucleus, upper portion. P.I. Postero-internal nucleus. P.E. Postero-external nucleus. E. External nucleus. S. Superior nucleus. Some of the root fibres from the lower and intermediate parts of the anterior nucleus are represented by dotted lines as crossing to the opposite side. IV. The nucleus of the fourth nerve. IV.R.$_1$, IV.R.$_2$, IV.R.$_3$. The first, second, and third portions of the root respectively. (BRUCE.)

The *fourth, pathetic* or *trochlear* nerve supplies the superior oblique. It apparently arises on the outer side of the crus just in front of the pons, and can be traced behind the corpora quadrigemina to the valve of Vieussens.

Its deep origin can be traced to the nucleus situated at the level of the depression midway between the anterior and the posterior corpora quadrigemina in front of the gray matter which surrounds the aqueduct of Sylvius. (Bruce.) Its connection with the nucleus of the sixth nerve, which has been described, is doubted by Bruce, who regards it as more probable that fibres may go to that division of the opposite oculo-motor nucleus which supplies the inferior rectus.

It pierces the dura near the posterior clinoid process, passes along the outer wall of the cavernous sinus, and enters the orbit through the sphenoidal fissure, being the highest of the nerves passing through the orbit. It also receives filaments from the cavernous plexus of the sympathetic and transmits a twig to the lachrymal, the first branch of the ophthalmic, or primary branch of the tri-facial.

FIG. 262.

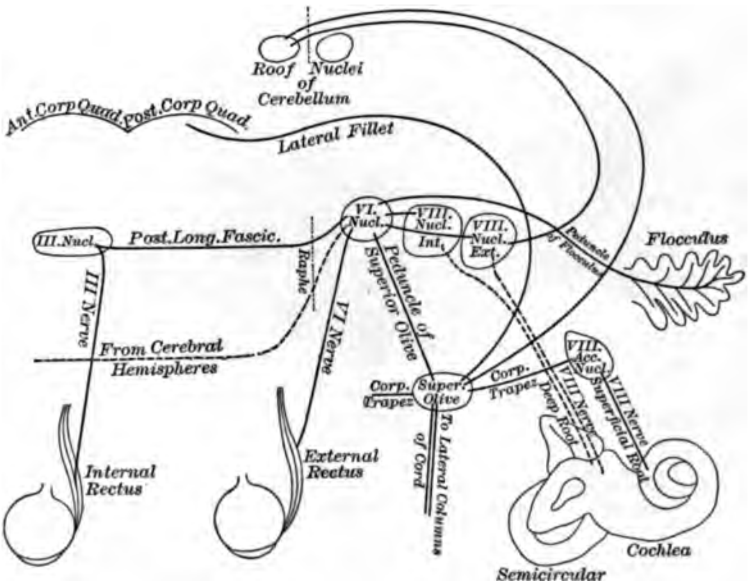


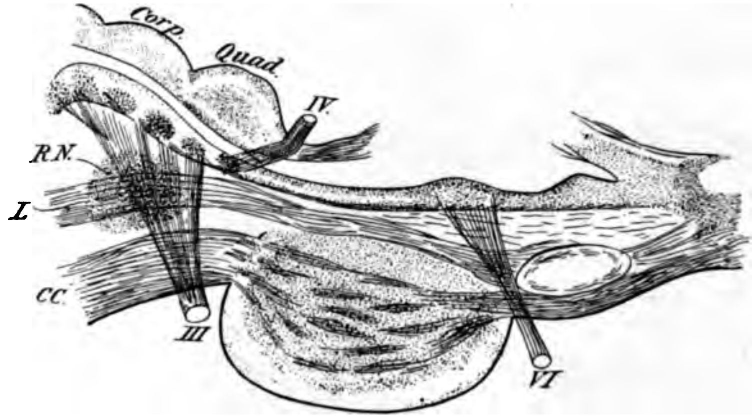
Diagram of the connections of the nucleus of the sixth nerve. (BRUCE.)

The *sixth*, or *abducens*, nerve supplies the external rectus. It apparently arises from the corpus pyramidale close to the pons. Its deep origin is from the nucleus situated under the floor of the fourth ventricle, on which its position is marked by a slight elevation, the eminentia teres, immediately above the acoustic striæ. The nucleus is covered by the ependyma of the fourth ventricle. It is bordered on its inferior, inner, and upper sides by the first, second, and third portions of the facial nerve. The root fibres leave the nucleus and become united at the outer side of the posterior longitudinal fasciculus into a number of bundles (Bruce). The connections of the sixth nucleus, according to Bruce, are somewhat as follows: With the second part of the root of the facial nerve and the segment of the third nerve nucleus supplying the internal rectus, with the superior olive, with the auditory nucleus and with the cortex of the opposite cerebral hemisphere, and probably also with that of the same side. Clinical investigations seem to show that the sixth nerve nucleus is the centre of conjugate lateral deviation. (See page 787.) The olive has also been supposed to contain this centre.

The nerve pierces the dura mater on the basilar surface of the sphenoid bone, passes through the posterior, or clinoid process, enters the cavernous sinus, and finally reaches the orbit through the sphenoidal fissure between the two heads of the external rectus. It receives filaments from the carotid and cavernous plexus of the sympathetic, from Meckel's ganglion, and from the ophthalmic nerve.[1]

Clinical, experimental, and anatomical data seem to show that, although the frontalis and orbicularis palpebrarum receive their peripheral nerve supply from the facial nerve, they are really eye-muscles. They are designated the *oculo-facial group*, and their central innervation, according to Mendel, is the oculo-motor nucleus, connected with the facial through the posterior longitudinal fasciculus.

FIG. 263.



Sagittal section through the cerebral axis, to show the nuclei of the ocular nerves in the floor of the aqueduct of Sylvius and the fourth ventricle, and the course of the nerves to their exit. The various groups of cells from which the third nerve arises are seen. *R N*, red nucleus of tegmentum; *L*, lemniscus (sensory tract); *C C*, motor tract in the crus cerebri seen to traverse the pons and enter the anterior pyramid of the medulla. (STARR.)

It will thus be seen that, in general terms, the cell groups constituting the nuclei of the nerves governing the ocular muscles lie in the gray matter of the floor of the aqueduct of Sylvius and of the fourth ventricle, the major gathering being beneath the corpora quadrigemina. (Fig. 263.)

PHYSIOLOGICAL ACTION OF THE OCULAR MUSCLES. The actions of the ocular muscles are reckoned from what is known as the *primary position* of the globe. The eyes occupy this position when they are directed straight forward, the head being held erect, and a distant object situated in the median line of the visual plane is observed with practically parallel visual lines. Positions of the eyes other than these are called *secondary positions.*

It is customary to separate the six muscles which control the movements of the eyeballs into three pairs, namely, the internus and externus, which control the horizontal movements or rotations; the superior rectus and inferior oblique, which control the upward movements, and the inferior rectus and superior oblique, which control the downward movements. Movement of the eyeball upward and inward is performed by the superior and internal rectus acting with the inferior oblique; downward and inward by the inferior and internal rectus acting with the superior oblique; upward and outward by the superior rectus and external rectus acting with the inferior oblique; and downward and outward by the inferior and external rectus acting with the superior oblique. The first series of actions, comprising the horizontal and vertical movements, are the ones of chief interest in the ordinary diagnosis of the ocular palsies, and it has not been considered necessary in these pages to describe the obliquity of the double images, which is also present in diagonal movements when there is paralysis of one or more muscles.

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[1] The course, connections, and distributions of the cranial nerves are according to Gray's Anatomy, American edition, edited by Keen. The deep origin of the nerves and the relation of the nuclei are taken from Alexander Bruce, "Illustrations of the Nerve Tracts in the Mid and Hind Brain."

Under normal conditions there is co-ordination in the movements of the eyes, one eyeball being associated in its movements with those of its fellow. If these *associated movements* regulated by equal impulses from the co-ordinating centre did not exist, single vision would not be possible, for the two images of an object could not fall upon corresponding parts of the retinæ.

In order to test the extent of the movements of the eyes in various directions, or the *field of fixation*, the limit of the excursion of the eye in four chief directions may be measured. This amounts in the vertical and horizontal directions to 90°.

PARALYSIS OF THE EXTERNAL OCULAR MUSCLES. Certain general symptoms are common to paralysis, or paresis, of the external ocular muscles.

General Symptoms of Paralysis of the External Ocular Muscles. These may be summed up as follows:

1. *Diplopia*, owing to the failure of images of an object to fall upon corresponding points of the retinæ, and which increases as the object is moved to the side of the paralyzed muscle.
2. *Strabismus*, which may be manifest only in complete cases, or only appear when attempt is made to move the eye in the direction of the action of the palsied muscle. Sometimes it results from secondary contractures.
3. *Primary deviation*, or limitation of movement of the eye in the direction of the action of the affected muscle. Therefore this is always in a direction opposite to the action of the muscle.
4. *Secondary deviation*, or the deviation of the sound eye, while the affected eye attempts to regard an object. This secondary deviation is always greater than the primary deviation, because the same degree of nervous impulse passes from the centre to the muscles of the affected eye and to those of the unaffected associate; the former requires an abnormally great impulse to stimulate its movement, and hence the latter is over-excited, causing an excessive movement.
5. *False projection of the field of vision*, or, in other words, an inaccurate estimation of the position of an object which is situated in such a portion of the visual field that it requires an effort on the part of the affected muscle to turn the eye toward it.
6. *Vertigo.* When both eyes are open this depends upon the diplopia. If the unaffected eye is closed, it depends upon an erroneous localization of objects in the field of vision.
7. *Altered carriage of the head*, which depends upon the impulse of the patient to turn his head in that direction in which he is least troubled by the double images.

Varieties of Diplopia. According to the relation which the double images bear to the eyes, there are two varieties of diplopia, *simple* or *homonymous*, and *crossed* or *heteronymous* diplopia. The former is present if the right, image pertains to the right eye, and the left image to the left eye, and the latter, if the reverse is true. The image seen by the unaffected eye is termed the *true image;* that seen by the affected eye, the *false image.* If the two images are on a horizontal line, there is *lateral diplopia;* if there is vertical displacement of the double images, *vertical diplopia.* Certain rules in regard to diplopia in cases of recent external ocular muscle palsy should be remembered, namely:

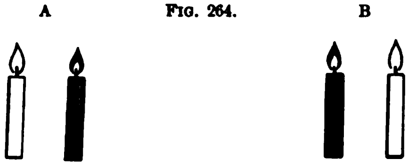
1. Diplopia is most marked and sometimes only noticeable when the patient turns his eyes in a direction which requires an action of the affected muscle, while if the eye moves in such direction that the paralyzed muscle is not concerned, diplopia is absent.

2. The image which belongs to the affected eye is projected in the direction toward which the paralyzed muscle normally rotates the eye.

3. The relative distance of the double images increases when the eyes are turned in the direction of the action of the paralyzed muscle, or, in other words, that image is false and belongs to the affected eye which in the region of diplopia moves faster than a moving test-object, for example, a candle flame.

Special Symptoms. The most important symptoms peculiar to paralysis of the ocular muscles are summarized in the following paragraphs. It is supposed that the *right eye is affected* and that the signs of complete or nearly complete palsy are present.

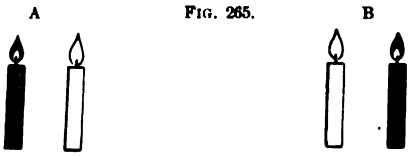
FIG. 264.



A, position of images in paralysis of the left external rectus, and B, in paralysis of the right external rectus. The false image is drawn in outline; the true image shaded. (Modified from FUCHS.)

1. *External Rectus.* Homonymous diplopia, the images being side by side and parallel when the eyes are directed on a horizontal level, the distance between them widening as the test-object is moved to the right; convergent strabismus, increasing as the object is moved to the right, and limitation of movement in this direction; inward secondary deviation of the sound eye; false projection of the field of vision to the right side, and turning of the face toward the right or affected muscle. (See Fig. 264 B.)

FIG. 265.

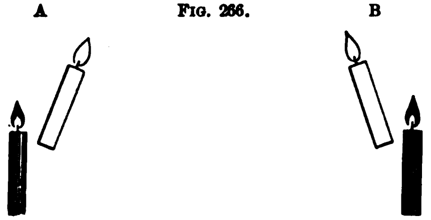


A, position of the images in paralysis of the left internal rectus, and B, in paralysis of the right internal rectus.

2. *Internal Rectus.* Crossed diplopia, the images being side by side and parallel if the eyes are directed on a horizontal level, the distance between them widening as the test-object is moved to the left; divergent strabismus, which increases as the eye attempts to follow an object moved to the left, and limitation of movement in this direction; outward secondary deviation of the sound eye; false projection of the visual field to the left, and rotation of the face toward the left or affected muscle. (Fig. 265 B.)

3. *Superior Rectus.* Crossed diplopia in the upper field, the images being one above the other, the image of the affected eye higher than its fellow and inclined to the left, and the distance between them widening as the test-object is moved upward and to the right; downward strabismus, which increases when the eye attempts to follow an object moved upward, and limitation of movement in this direction; upward secondary deviation of the sound eye; false projection of the visual field too high, and upward direction of the face. (Fig. 266 B.)

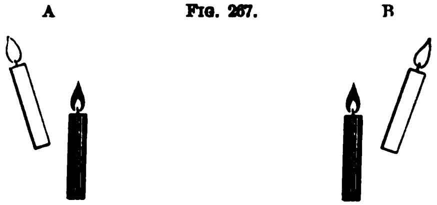
FIG. 266.



A, position of images in paralysis of left superior rectus, and B, in paralysis of right superior rectus.

4. *Inferior Oblique.* Homonymous diplopia in the upper field, the images being one above the other, the image of the affected eye being higher than its fellow and inclined to the right, the vertical distance between them widening as the test-object is moved upward and to the left; downward and inward direction of the affected eye, most marked when the eyes attempt to follow an object moved upward and outward, with limitation of movement in this direction; upward and inward secondary deviation of the sound eye; false projection of the visual field too far upward, and turning of the face upward and toward the left. (Fig. 267 B.)

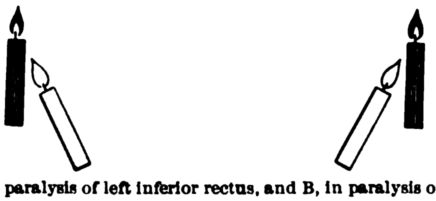
FIG. 267.



A, position of images in paralysis of left inferior oblique, and B, in paralysis of right inferior oblique.

5. *Inferior Rectus.* Crossed diplopia in the lower field, the images being one above the other, the image of the affected eye being lower than its fellow, and inclined to the right, and the distance between them widening as the test-object is moved downward and to the right; upward strabismus, which increases when the eye attempts to follow an object moved downward, with limitation of movement in this direction; downward and outward secondary deviation of the sound eye; false projection of the visual field too far downward, and turning of the face downward and to the right. (Fig. 268 B.)

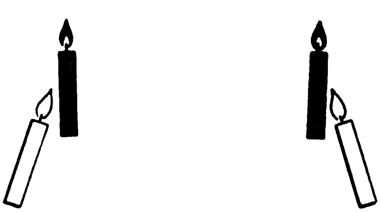
FIG. 268.



A, position of images in paralysis of left inferior rectus, and B, in paralysis of right inferior rectus.

6. *Superior Oblique.* Homonymous diplopia in the lower field, the images being one above the other, the image of the affected eye being lower than its fellow and inclined to the left, the vertical distance between them widening as the test-object is moved downward and to the left; upward and inward direction of the affected eye, especially when the eye attempts to follow an object moved downward and outward, and limitation of movement in this direction; downward and inward secondary deviation of the sound eye; false projection of the visual field too far downward, and inclination of the face downward and to the left. (Fig. 269 B.)

FIG. 269.



A, position of images in paralysis of left superior oblique, and B, in paralysis of right superior oblique.

7. *Oculo-motor* (*third nerve*) *Paralysis.* Crossed diplopia; divergent strabismus and limitation of movement in all directions except outward and slightly downward; outward secondary deviation of the sound eye; false projection of the visual field to the inner side, and inclination of the face to the right, the chin being tipped upward; in addition, ptosis, medium dilatation of the pupil, and paralysis of accommodation.

Method of Examination. In complete paralysis the strabismus and the limitation of movement of the paralyzed muscle are sufficient to identify the affected organ. In paresis, however, a diagnosis must be based upon a study of the double images. The patient, therefore, is seated four or five metres from the test-object, which is usually a candle-flame, and one eye is covered with a piece of red glass. This distinguishes the flames.

The lighted candle is then moved from the median line horizontally to the right and to the left, and the relative positions of the images noted. In like manner it is moved upward and downward and the same observation recorded. According to the rules already given under diplopia (see page 778) the false image, and consequently the affected eye, can usually be determined, and, by a process of exclusion, the paretic muscle. Many tables have been constructed to aid the memory in this respect. The following is very useful and has been prepared by Dr. F. C. Hotz:[1]

I. Lateral diplopia indicates paralysis of an internal or an external rectus.

1. Homonymous diplopia indicates paralysis of an external rectus.

a. Images separating to the right indicate paralysis of the externus of the right eye.

b. Images separating to the left indicate paralysis of the externus of the left eye.

2. Crossed images indicate paralysis of an internus.

a. Images separating to the right indicate paralysis of the internus of the left eye.

b. Images separating to the left indicate paralysis of the internus of the right eye.

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[1] The Diagnosis of Paralysis of the Ocular Muscles by the Double-image Test. International Clinics, vol. iii. fourth series.

II. Vertical diplopia in the upper field indicates paralysis of the superior rectus or inferior oblique.

1. Homonymous images indicate paralysis of the inferior oblique.

a. Image of right eye higher means paralysis of the inferior oblique of the right eye.

b. Image of right eye lower means paralysis of the inferior oblique of the left eye.

2. Crossed images indicate paralysis of the superior rectus.

a. Image of right eye higher means paralysis of the superior rectus of the right eye.

b. Image of right eye lower means paralysis of the superior rectus of the left eye.

III. Vertical diplopia in the lower field indicates paralysis of the inferior rectus or superior oblique.

1. Homonymous images indicate paralysis of the superior oblique.

a. Image of the right eye lower means paralysis of the superior oblique of the right eye.

b. Image of the right eye higher means paralysis of the superior oblique of the left eye.

2. Crossed images indicate paralysis of the inferior rectus.

a. Image of the right eye lower means paralysis of the inferior rectus of the right eye.

b. Image of the right eye higher means paralysis of the inferior rectus of the left eye.

It is exceedingly difficult, however, always to exactly localize the affected muscle, a difficulty which is much increased when more than one is paretic, the paresis being of different degrees.

VARIETIES OF PARALYSES OF THE OCULAR MUSCLES. Some authors reserve the name *paralysis* for those cases in which the affection is unilateral, or for the palsy of muscles supplied by single nerves, and *ophthalmoplegia* for all other cases; but as Knies points out, this distinction is not always maintained with accuracy, and hence the term ophthalmoplegia may be applied to the entire group of eye-muscle palsies. According to Mauthner[1] the ophthalmoplegias, as well as the single-muscle palsies, may be arranged in the following manner:

Ophthalmoplegia (Myo-paralysis.)
I. Intra-cranial.
    a. Cerebral.
        1. Cortical.
        2. Nuclear.
        3. Fascicular.
    b. Basal.
II. Orbital.
III. Peripheral.[2]

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[1] Die Lehre von den Augenmuskellähmungen, 1889, p. 310.

[2] Collins and Wilde (Amer. Journ. Med. Sci., N. S., vol. cii. p. 512) have prepared a scheme of division which is more elaborate and has many points to commend it, viz.:

I. Cerebral: (*a*) Cortical.
    (*b*) Cortico-peduncular.
    (*c*) Nuclear.
        iii. 1. Cycloplegia } "Ophthalmoplegia interna."
        iii. 2. Iridoplegia. } "Ophthalmoplegia interna."
        iii. 3. Palsy of extra-ocular muscles. Ptosis.
        iv. 4. Palsy of superior oblique.
        vi. 5. Palsy of external rectus.
    (*d*) Radicular (and commissural.)
II. Basal: (*a*) Region of pons.
    (*b*) Region of peduncles.
    (*c*) Region of cavernous sinus.
    (*d*) Region of sphenoidal fissure.
III. Orbital (including peripheral).

ORBITAL AND PERIPHERAL PALSIES OF THE EXTRA-OCULAR MUSCLES. The causes residing within the orbit which produce paralysis of the muscles (orbital palsies), are chiefly of ophthalmological interest, namely, cellulitis, tenonitis, tumors, hemorrhages, fracture, and affections of the frontal sinus and adjacent cavities.

It is very difficult to make an accurate separate classification of *peripheral palsies.* In fact many writers are content with the separation of the ocular palsies into the central and the peripheral, meaning by the latter those caused by an interruption in the course of the nerve-trunks themselves, and, therefore, including orbital and basal palsies.

It is more accurate, however, as Mauthner and other writers have pointed out, to call those cases peripheral which, so far as diagnosis is possible, are caused by influences not acting in the orbit or at the base, but on the nerve within the muscle.

Thus rheumatism probably always causes a peripheral palsy, and the external rectus is the muscle most usually affected. It occurs in rheumatic subjects after exposure, but rarely during attacks of acute articular rheuma-rheutism.[1] Mauthner calls attention to the fact that many cases of so-called matic palsy have been followed years afterward by tabes, disseminated cerebral sclerosis, progressive paralysis, etc., and may be looked upon as forerunners of such affections under certain circumstances.

Certain palsies of the external rectus are probably occasioned by simple neurasthenia, and high grades of insufficiency of the same muscle, if not an actual palsy, result from uric-acid diathesis, gout, and similar general conditions.

Diphtheria may also occasion a peripheral (as well as nuclear) palsy, andother causes are to be found in diabetes, influenza, and particularly certain toxic agents, lead, alcohol (these two causing a neuritis), gelsemium, conium, chloral, carbonic acid, and spoiled fish, meats, etc. (ptomaine poisoning).

BASAL PALSIES OF THE EXTRA-OCULAR MUSCLES. The lesions causing paralysis of the ocular nerves by affecting them in their course at the base of the brain may be briefly summarized thus: Hemorrhage, pachymeningitis, meningitis, both simple and tubercular, but particularly the latter, which sometimes in children first manifests itself by sudden convergent strabismus and diplopia;[2] purulent meningitis and localized abscess, for example, in connection with middle ear disease; aneurism, thrombosis, and other diseases of the cavernous sinus; arteritis obliterans, especially syphilitic disease of the basilar arteries; tumors, and, finally, those conditions which Mauthner (whose grouping has been quoted) has called essential diseases of the basal branches of the motor ocular nerves: neuritis, gummatous disease, tuberculosis, and gray degeneration.

Reverting to syphilis, we may say that it is the most frequent of all causes of ocular palsies, constituting, according to Alexander, 59.4 per cent. of the cases. It may cause a neuritis, perineuritis, inflammation or gummatous change of the nerve-trunks themselves, or originate deposits of gumma, or excite a periostitis at the sphenoidal fissure.

According to the region affected, the sixth, third, or fourth nerves may be included, the oculo-motor being most frequently selected by syphilitic disease. Generally a late symptom, and occurring usually at the gummatous stage of the disease, syphilitic oculo-motor palsy has been noted as early as the sixth month after primary infection, particularly in the form of ptosis. It should be remembered that it may also be a symptom of inherited syphilis, instances of this character having been recorded by Graefe, Nettleship, and Lawford.

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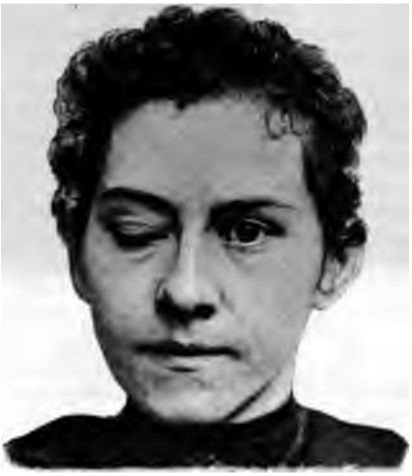
[1] The author has seen one such case in the Philadelphia Hospital.

[2] Mauthner states that he has seen under these circumstances a pure type of trochlear paralysis.

Recurrent Oculo-motor Paralysis. (*Intermitting paralysis of the oculomotor nerve,* sometimes called *ophthalmoplegic migraine*—Charcot.) This is comparatively a rare affection, which may be classed among basal palsies, about forty cases having been recorded in medical literature.[1] According to Darquier's collection, the disease attacks the female sex more frequently than the male, nineteen times in twenty-seven observations; but Knapp finds the two sexes affected about equally, the left nerve being more commonly involved than the right. It generally begins at an early period of life, from the second month to the fifth year, although it may begin later, and in rare instances, even in old age.

The symptoms are violent unilateral headache, lassitude, nausea, vomiting slight fever, and usually complete paralysis of the third nerve on the same side as the pain. Unusual symptoms are purulent discharge from the eye, swelling of the lids, and anæsthesia in the distribution of the trigeminus. A concomitant symptom may be facial paralysis, and in some cases there were recurring attacks affecting several cranial nerves. A similar condition of the abducens has been described (Nieden, Charcot), and the fifth has been involved in a few instances.

FIG. 270.



Recurrent ptosis of right eye, permanent palsy of all other branches. A case studied by Dr. Dercum and the author in the Jefferson Medical College Hospital, Philadelphia.

The attacks may come in periodic crises, and the disease may last from several days to long periods of time. It is difficult to make a strict classification, but in general terms it may be stated that in one variety there is complete recovery of the paralyzed muscles during the interval, while in the other some trace of the affection remains, for example, a paretic muscle. In several cases there was complete recovery in the interval between the earlier attacks, but increasing paralysis, amounting in two instances to complete paralysis, in the interval between the later attacks.[1]

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[1] See article by Darquier entitled "De Certaines Paralysies Récidivantes de la troisième Paire." Annales d'Oculistique, Tome cx. Octobre, 1893, p. 257, and a thorough analysis of the cases by Philip Coombs Knapp, in the Boston Medical and Surgical Journal, September 27, 1894, whose article has been freely quoted in the following paragraph.

The pathology is uncertain, but the evidence goes to show that it is a basal disease, for example, a meningitis, although it has been ascribed to hysteria, and also to nuclear lesion, but to the latter apparently without good cause. Knapp says "the most tenable hypothesis is that recurrent oculo-motor paralysis is due to some vascular change, inflammatory or œdematous, in a focal lesion involving the root of the third nerve." In three autopsies thus far reported they were lesions of the nerve at its exit in front of the pons.

Cerebral Extra-ocular Palsies. Paralyses of the extra-ocular muscles having a cerebral origin result from lesions degenerative, inflammatory or hemorrhagic, affecting the cortex of the brain, the cortico-peduncular region, the nuclei of the nerves, or the nuclear fibres.

Certain experiments indicate that there exist centres in the cortex (*cortical centres*) controlling the movements of the ocular muscles, and theoretically a lesion in such situation would be followed by paralysis of the ocular movements under its control. Risien Russell[2] believes, as the result of elaborate experimentation, that all the movements of the eyeball are represented in the cerebral cortex and not only the lateral movements. From pathological investigations it has been assumed that certain isolated paralyses of the levator producing the symptom of ptosis are of cortical origin.[3]

A lesion not affecting the cortex, the nuclei proper, the nerve-trunk at the base of the brain, or its distribution in the orbit and the muscles themselves, followed by paralysis of ocular movements, could be situated only in the region which connects the cortical centre with the nuclear origin. These palsies, which might be inferred from concomitant symptoms, may be called, according to Mauthner, *fascicular paralyses*, just as those which have been described in the previous paragraph may be denominated *cortical palsies*.

Ophthalmoplegia. Systematic writers have particularly reserved for that class of paralysis of the orbital muscles due to disease of the nuclei of the third, fourth, and sixth nerves the name *ophthalmoplegia* or *nuclear paralysis*.

As has already been stated, the term ophthalmoplegia may with perfect propriety be used to describe all of the ocular-muscle palsies, although it is frequently restricted by writers to the class just referred to. It was first employed by Brunner, and later by Von Graefe, who recorded a characteristic example of the disease under the term progressive ophthalmoplegia. Jonathan Hutchinson introduced the names *ophthalmoplegia externa* and *ophthalmoplegia interna*, meaning by the former a symmetrical, progressive paralysis of the external ocular muscles, in contrast to non-symmetrical, single muscle palsies, and by the latter paralysis of the intra-ocular muscles (iris and ciliary body), which he believed to be due to disease of the lenticular ganglion. This distinction, however, is not now maintained, because the two sets of muscles may be affected in the same case, the symmetrical character of the palsy does not always exist, and Mr. Hutchinson's idea of the pathology of the internal palsies is not correct. Collins and Wilde (*loc. cit.*) have found when palsy of either the iris or ciliary muscle coexisted with extra-ocular paralysis it was more frequently the former—31 cases among 34.

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[1] The author has studied one remarkable case, also seen by Dr. Dercum, which began at one and one-half years, with complete right oculo-motor palsy. The branch supplying the levator recovered; the rest of the branches remained paralyzed. From the time of the original attack until the twenty-ninth year there were numerous crises, with the typical symptoms which have been described, always resulting in recurrence of the ptosis. Finally, a violent attack occurred, the ptosis recurred, but has never disappeared; in other words, there is now permanent oculo-motor palsy. (See Fig. 270.)

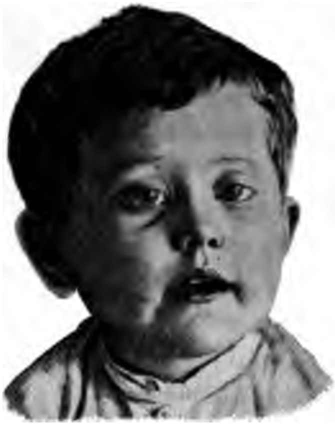
[2] Journal of Physiology, 1894, vol. 1, xvii.

[3] This subject is referred to again on p. 789.

Ophthalmoplegia, as ordinarily defined, may be divided into *acute ophthalmoplegia*, or *acute nuclear palsy*, and into *chronic ophthalmoplegia*, or *chronic nuclear palsy*. When it so happens that the intra-ocular muscles alone are affected, there is no objection to the term *ophthalmoplegia interna* (interior ophthalmoplegia, either complete or incomplete, unilateral or bilateral), if it is understood that it does not necessarily refer to a special disease. When the external muscles are alone affected, in like manner, *external ophthalmoplegia* (ophthalmoplegia exterior, complete or incomplete, unilateral or bilateral) is utilized. When both sets are affected *ophthalmoplegia universa*, or *total ophthalmoplegia*, is the term employed, synonymous with complete bilateral inward ophthalmoplegia of Dufour.[1]

Acute ophthalmoplegia, or *acute nuclear palsy*, is characterized by rapid paralysis of all the ocular muscles, often associated with fever, vomiting, and convulsions. Dufour classifies 27 of these cases among 220 examples of ophthalmoplegia. In the severest type they usually have been quickly fatal, and have occured with hemorrhage into the region of the nuclei (apoplexy), or as an acute hemorrhagic polioencephalitis on the floor of the fourth ventricle and aqueduct, the primary cause being tuberculosis, syphilis (?) ptomaine toxæmia, or poisoning from alcohol or sulphuric acid. A less fatal, or even benign, type (67 among 220—Dufour) may occur under the influence of injuries, and certain poisons—for example, nicotine, lead, and carbonic acid (Knapp), or result from constitutional diseases (diabetes, syphilis, epidemic influenza).

FIG. 271.



Bilateral paralysis of the external recti after diphtheria. From a case studied by the author in the service of Dr. Weir Mitchell at the Infirmary for Nervous Diseases.

It is probable that some of the cases of diphtheritic paralysis of the external ocular muscles are nuclear in type. Independently of the common cycloplegia of this disease (see p. 801), there may be paralysis of a single external ocular muscle, most frequently the external rectus, but also the superior oblique and the internal recti, or paralysis of both external recti, or, finally, paralysis of all the ocular muscles. In Mendel's case of complete ophthalmoplegia externa he believed the condition to be due to capillary hemorrhages (central), associated with a primary peripheral neuritis.

Acute ophthalmoplegia may be associated with acute poliomyelitis, with

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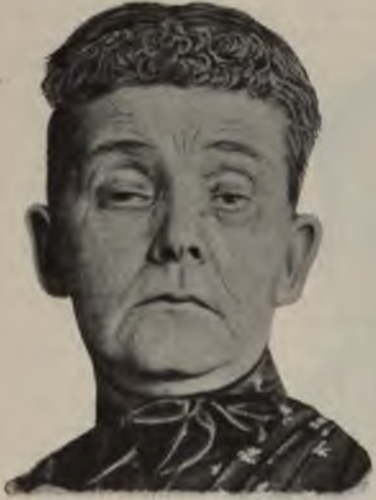
bulbar palsy, or with facial paralysis. It may be confounded with an acute peripheral neuritis of the orbital nerves, but, as Swanzy points out, there would then be an absence of the concomitant symptoms referable to intracranial origin.

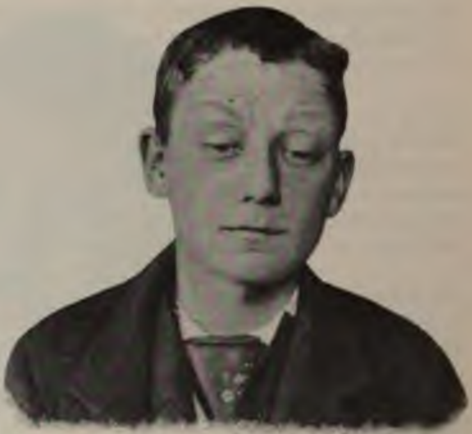
Chronic ophthalmoplegia, or chronic nuclear palsy, is characterized by loss of power in one or more eye muscles, which, at first slight, gradually increases and spreads until every muscle is paralyzed, although the levator may escape partially, and, indeed, ptosis may be absent. Two chief forms are distinguishable, a *stationary* and a *progressive,* the difference in their clinical characters depending on the fact that in the former, after a certain development of palsy, usually symmetrical, the process comes to a standstill, while in the latter it is truly progressive, as before stated, until all muscles are involved. The disease is not always symmetrical (it may be unilateral), and the diplopia, early present, usually disappears in the later stages. The disease depends upon atrophy or degeneration of the nuclei of the third and fourth nerves, and also the correlated nucleus of the sixth, and occurs (1) as a sequel of an acute ophthalmoplegia, the lesions of which have failed to clear up, or have produced chronic degenerative changes; (2) as a congenital (occasionally hereditary) affection, usually in the form of bilateral ptosis, with deficient power in the superior recti; (3) as an acquired affection in children (with whom it is more serious), or in adults, preference being given to the male sex; (4) in association with locomotor ataxia, paretic dementia, progressive muscular atrophy, chronic bulbar paralysis, disseminated sclerosis, and atrophy of the optic nerve, and in connection with palsy of the oculo-facial group.[1] (See page 776.)

FIG. 272.



Chronic ophthalmoplegia. (Infirmary for Nervous Diseases.)

FIG. 273.



Chronic ophthalmoplegia in a boy under the author's care in the Jefferson Medical College Hospital, showing effort of occipitalis to supplant the action of the levators.

It is often difficult to ascribe the condition to an exact cause, though in many instances constitutional syphilis can be demonstrated, and in other cases a tubercular process. It is essentially chronic, and may last for many years.

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[1] See cases by Hughlings Jackson, Lancet, July 15, 1893.

If the anterior cell group (anterior-lateral nucleus of Darkschewitsch) of the third-nerve nucleus escape (which is not necessarily the case), the intraocular muscles (iris and ciliary muscle) are unaffected, and there is strong presumptive evidence that a chronic paralysis of the external ocular muscles is nuclear in origin, but it is not a characteristic sign. Dammron (quoted by Siemerling) has recorded a case due to peripheral neuritis, the nuclei being unaffected, with preservation of accommodative power and the pupillary reflexes, and Goldzieher[1] has described a child with complete ophthalmoplegia externa, without changes in the power of accommodation or iris-movements, due to tumor of the corpora quadrigemina. There were no obvious changes in the nuclear area (the microscope was not utilized). Siemerling concludes that nuclear disease may be inferred from ophthalmoplegia externa, if it is not maintained that nuclear palsy *must* manifest itself as an external ophthalmoplegia.

Nuclear ophthalmoplegia may be closely simulated by the ocular-muscle-symptoms of pontine tumors, by patches of softening or sclerosis, with over-distention of the Sylvian aqueduct, but, as Swanzy remarks, these are not true nuclear palsies, and must be differentiated by concomitant symptoms.

According to Siemerling,[2] the pathological states underlying progressive paralysis of the ocular muscles may reside in:

1. Nuclear disease (disappearance of the ganglion cells), with participation of the nerves to their termination in the muscle.
2. Degeneration of the muscles and of the nerve-trunks, with intact nuclei.
3. Interruption of the conducting power of the intra-medullary roots on account of sclerotic foci, with intact muscles, nerve-trunks, and nuclei.

CONJUGATE LATERAL PARALYSIS. If the eyes are turned from a mid-position, with practically parallel visual axes, to fix an object on either side, the lateral movement is accomplished in obedience to an impulse coming from the cortex, and causing synchronous action of the external rectus of one eye and of the internal rectus of the other. (See page 775.) The pathway of this impulse is from the cerebral cortex to the sixth-nerve nucleus, thence, via the posterior longitudinal fasciculus, it crosses to the opposite side and passes to the nucleus of the third nerve. Lesions interfering with this movement beget a symptom to which the name *conjugate lateral paralysis* is given, and may be situated in the cortex, corona radiata, or internal capsule, *i. e.*, above the nuclei; or in the pons, *i. e.*, in, and in the neighborhood of, the nuclei.

Although we know that stimulation of the middle portion of the frontal lobe causes turning of the eyes to the opposite side and that stimulation in the visual area in the occipital lobe causes the same movement, but in an opposite direction,[3] we also know that conjugate movement is possible from stimulation almost anywhere in the cortex, or, in other words, that the cortical centre for conjugate deviation is unknown. Moreover, wherever it exists, it is readily affected, and consequently conjugate deviation may often be a distant symptom, and is likely to occur with cortical lesions, diversely placed.

Conjugate deviation is a common symptom in gross lesions of the cerebrum (apoplexy), and often is transitory (occasionally permanent), the eyes being turned to the side opposite to the palsy (Prevost's symptom). For example, in a left hemiplegia there is left conjugate lateral palsy (*i. e.*, paralysis of the lateral moving power of the left external rectus and right internal rectus), the eyes, by the action of the unaffected right external rectus and left internal rectus being turned to the right. If the lesion, similarly placed, is an irritative one, the deviation is in exactly the opposite direction. Although the eyes have lost the power to make a movement to one or the other side, the directing power of the muscles may be unimpaired when they exercise their function in a different association, *e. g.*, in the act of convergence; hence, there is palsy of movement and not of the muscles supplied by a given nerve.

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[1] Centralbl. f. prakt. Augenheilk., February, 1893.
[2] Archiv f. Psychiatrie und Nervenkrankheiten, 1891, Bd. xxii. Suppl. Heft.
[3] Consult Eye Paralysis by John A. Jeffries, Bost. Med. and Surg. Journ., Oct. 20 and 27, 1892. This article has been utilized in the preparation of this section.

If the sixth-nerve nucleus in the pons is destroyed by a lesion there is conjugate lateral paralysis of the same side and conjugate deviation to the opposite side, because the abducens supplies the external rectus of its own side and partly the internal rectus of the opposite side. Should the lesion be an irritative one, the deviation is in an exactly contrary direction. Thus it is evident that the conjugate lateral paralysis caused by cerebral lesions and by pontine lesions produces reverse forms of conjugate deviation. Swanzy[1] records this in the following tabular manner:

Cerebral lesions:	Destructive. Eyes turned away from palsied side. Irritative. Eyes turned toward convulsed side.
Pontine lesions:	Destructive. Eyes turned toward palsied side. Irritative. Eyes turned from convulsed side.

Certain other varieties of conjugate palsy have been observed: Complete conjugate paralysis (*i. e.*, one external rectus and the opposite internal rectus), so that neither muscle is capable of motion; the eyes turn to the opposite side, move up or down, but cannot pass the middle line toward the lesion. Lesions occupying an area in the pons above the sixth nucleus or immediately below and in front of it have been followed by such phenomena. The same condition, except that the internal rectus of the one eye was normal in convergence, has been recorded with a lesion just below the eminentia teres and in the upper quarter of the pons (Mills). The facial nerve may be affected at the same time. Jeffries[2] describes two other conditions, viz.: (*a*) When both eyes are open, the eye with the paralyzed internal rectus will not turn in for objects on the other side of the nose, but will if the other eye is covered; and (*b*) the internal rectus will not act in conjugate, but will act in near vision, the external rectus being normal. Autopsies do not explain these differences satisfactorily.

In conjugate palsy of upward or downward, but not of lateral movement, autopsies have revealed a lesion at the posterior part of the third nerve nuclei. The same condition has been recorded by Wernicke in disease of the corpus striatum and of the optic thalamus. Palsy of the upward movement of both eyes, without involvement of the levators, may be caused, as in Gowers's case, by a lesion (tumor) on the middle line behind the corpora quadrigemina, damaging these slightly, the velum, and the inferior vermiform process of the cerebellum.

Finally, we may have loss of the power of association of the internal recti in near vision (*i. e.*, convergence), although the ability to make lateral movements is unimpaired. Sometimes the power of accommodation is preserved, but convergence is lost; usually the two, normally associated together, are equally affected. Autopsies do not explain these conditions, and we may assume, in the absence of hysterical(?) states, a gross lesion affecting the centres for convergence, which are probably separate from but close to those for accommodation.

The Ocular Palsies; Significance of Palsies of the External Ocular Muscles. The significance of the palsies depending upon orbital and peripheral disease, often of pure ophthalmological interest, has been sufficiently detailed in the classifications which have preceded (page 782). It is well to insist with John Amory Jeffries, however, "that an eye paralysis, however simple it may seem, demands prompt and thorough examination of the patient."

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[1] Diseases of the Eye, 4th ed.

[2] Loc cit.

(*a*) *Sixth Nerve, or Abducens Palsy.* Abducens paralysis is not uncommonly a distant symptom; in fact, as has been pointed out by several authors, perhaps owing to the long course of this sixth nerve at the base of the brain, this phenomenon is more common with it than with other cranial nerves, abducens palsy, according to Wernicke, being a frequent symptom of tumor of the cerebellum. One or both nerves may be affected.

Paralysis of the sixth nerve occurring at the same time with a hemiplegia of the opposite side indicates a lesion in the pons on the same side as the ocular paralysis. This condition, as Mauthner points out, is rather rare, at least pure cases of it are uncommon; but when it occurs it points distinctly to a lesion in that portion of the pons through which the abducens fibres pass, therefore a fascicular paralysis. The sixth and facial nerves may be associated in a palsy, their nuclei being in close proximity, or, indeed, according to some authorities, identical. Abducens palsy occurring as a distant symptom with cortical hemiplegia may be differentiated from one which is a direct symptom of pontine lesion, because under the latter condition the paralysis of the extremities is contra-lateral. Complete unilateral paralysis of the sixth nerve is nuclear in origin when the associated action of the internus is also destroyed, but may be basilar from the pressure of the products of syphilitic or tubercular disease when there is no loss of the conjugate movements of the eyes toward the side of the lesion. Complete non-rheumatic palsy, focal lesions being excluded, indicates syphilis in adults, but tubercular disease in children (Wood.)

(*b*) *Fourth Nerve, or Trochlearis Palsy.* Isolated paralysis of the fourth nerve is not frequent. It is recorded twice by Collins and Wilde among 116 cases of ocular palsy, and yet, as Mauthner has pointed out, the purest types of trochlearis paresis may occur in basilar meningitis, especially in children, and not only from basal lesions, but in the form of a fascicular paralysis caused by the pressure of an intra-cerebral lesion.

As a focal symptom the paralysis is still more uncommon. Nieden (quoted by Swanzy) found this paralysis with tumor of the pineal gland. In a case presenting symptoms of cerebellar disease, and at the same time fourth-nerve palsy, it is fair to assume, according to Starr, that the anterior portion of the cerebellar hemisphere on the side of the paralysis is the part of the cerebellum affected.

It is more common to find the third and fourth nerves affected together; for example, in the Collins and Wilde analysis this occurred eight times. It might be basal in origin, or might occur with lesion of the cerebral peduncle.

(*c*) *Third Nerve, or Oculo-motor Palsy.* Third nerve palsy may be a distant symptom of lesion in the cerebral hemisphere. Indeed, Swanzy believes that ptosis as the result of cortical lesion is of this character in many of the cases. We have seen (page 784) that ptosis may be caused by a lesion of the cortex[1]—the co-called cerebral ptosis—on the opposite side of the lesion, for example, in the angular gyrus just below the inter-parietal fissure, as in a case of Herter.[2]

Disease of the corpora quadrigemina, as in a case of tubercular degeneration in this region observed by Steffen, may cause double ptosis, and, indeed, to quote Mauthner, that ptosis which accompanies cerebral and cerebro-spinal disease, as well as that ptosis which is associated with abducens paralysis of the same side, is usually nuclear in origin.

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[1] For an analysis of the cortical centres of the ocular muscles, see Mauthner's Die Lehre von den Augenmuskellähmungen, Wiesbaden, 1889, p. 385–389. Also Russell's recent researches (loc. cit.).

[2] Journal of Nervous and Mental Disease, January, 1895.

Contraction of the palpebral fissure producing apparent ptosis—called by Swanzy sympathetic or pseudo-ptosis—myosis, slight enophthalmos and lachrymation on the paralyzed side, has been described by Nothnagel with lesions of the corpus striatum.

Ptosis with crossed hemiplegia may be explained by an intra-peduncular lesion. If there is partial oculo-motor paralysis, (the functions of the iris and ciliary muscles being preserved), associated with contra-lateral hemiplegia, with or without facial or hypoglossus paralysis, according to Mauthner the lesion should be looked upon as affecting the fascicular fibres. If, on the other hand, with the same symptoms, the paralysis is a total one, it is almost certainly basilar, and when unassociated with other paralysis and total this diagnosis becomes practically certain. The common symptom of crossed paralysis, that is to say, oculo-motor palsy on the side of the lesion and hemiplegia upon the opposite side, is not certainly significant of crus disease unless the two paralyses come on simultaneously (Hughlings Jackson).

Double oculo-motor paralysis may be significant of nuclear disease, or compression at the base of the brain, for example, by a deposit from meningitis. Occasionally it develops somewhat rapidly and disappears without giving any signs of intra-cranial disease.[1] Syphilis may be suspected in many third-nerve palsies, the most noted exceptions being those due to diphtheria and those occurring in non-syphilitic tabetic patients.

The regions thus far discussed refer particularly, so far as the base is concerned, to the pons and the peduncles. If, now, a lesion affects:

(*d*) *The Region of the Cavernous Sinus*, the sixth, third, and fourth nerves are all likely to be involved. Thus, thrombosis of this venous channel produces ophthalmoplegia on the same side as the disease, œdema of the eyelids, proptosis, at first contracted and afterward dilated pupil, anæsthesia in the region of the distribution of the first division of the trigeminus, and sometimes neuro-paralytic keratitis. There may or may not be optic neuritis and distention of the retinal veins. These phenomena are markedly visible in the so-called pulsating exophthalmos, which is caused by an arterio-venous aneurism of the cavernous sinus. Meningitis, injury, or any form of pressure or lesion in the same region is likely to be followed by similar symptoms.

Lesions in the region of the sphenoidal fissure, frequently periostitis and syphilitic deposits, are fruitful causes of ocular palsies, and their significance has already been referred to on page 782 *et seq.*[2]

The *significance of nuclear disease*, in the form of acute and chronic ophthalmoplegia, and all those varieties of palsy originating in the cortical or cortico-peduncular areas which are conjugate, need not again be reviewed.

The *significance of certain causal varieties of paralyses* of the external ocular muscles, chief among which are those seen in connection with tabes dorsalis, paretic dementia and disseminated sclerosis remains to be considered.

The frequency of ocular paralysis among tabetic patients varies with different authorities. Berger (loc. cit.) notes it in 38 per cent., and quotes Erb, who found it in 27 per cent., and Moeli in 39.6 per cent. The paralysis is most apt to occur in the earlier and pre-ataxic stage, by some authorities, for example, Gowers, being considered to be more frequent in syphilitic subjects than in others, a conclusion not reached by Berger, who, however, believes that the paralyses are more apt to be permanent in the syphilitic tabetics.

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[1] For a description of these palsies, see Græfe's Archiv., Bd. xii. 2, 1866, and Mauthner, loc. cit. p. 415.

[2] The preceding paragraphs on the significance of the ocular palsies are to be understood as a brief and necessarily incomplete résumé of the subject, which is more fully treated in the chapters devoted to cerebral localization, in connection with which the study of cases of ocular palsies becomes of paramount importance. The author desires to note special indebtedness to, and frequent quotation from, Mr. Swanzy's admirable Bowman lecture on "The Value of Eye Symptoms in the Localization of Cerebral Disease." Transactions of the Ophthalmological Society of the United Kingdom, vol. ix. 1889, and Mauthner's Monograph, loc. cit.

There is some difference of opinion as to which nerve is most frequently affected, Erb and Charcot stating that the oculo-motor is more apt to be involved than others, while Woinow believes it is the abducens, especially the left. A very common partial oculo-motor palsy affects the branch supplying the levator. The internal recti may also be involved in a paralysis, or there may be merely—and this is an important point especially dwelt upon by Landolt—insufficiency of their converging power, an observation which the author has also made in a number of cases.

The cases of complete ophthalmoplegia in connection with tabes have already been referred to on page 786.

We are uncertain as to the exact cause of these palsies, some being explained as nuclear, others as the result of a descending neuritis, and still others as due to a chronic ependymitis. One of the characteristics of tabetic paralysis is its transitory nature in many instances, and by this peculiarity it may often be differentiated from the lesions of the nerve-trunks themselves. Fournier has developed the following table:

Symptoms of Tabes.	*Symptoms of Lesions of the Nerve-trunks.*
1. Paralysis, often partial, sometimes attacking only the iris.	1. Paralysis, total.
2. Paralysis, associated either with preservation of the accommodative reflexes or with myosis.	2. No such association.
3. Paralysis, quickly passing away, lasting perhaps a day, or even a few minutes.	3. Paralysis, lasting.
4. Paralyses prone to relapses.	4. Paralyses not prone to relapses.
5 Spontaneous cure common	5. Cure only after proper medication.

The important point is that a transient external ocular palsy may be a significant and early symptom of locomotor ataxia, and should never be disregarded.

In Friedreich's ataxia symptoms referable to the ocular muscles are not usual with the exception of nystagmus, although strabismus, diplopia, blepharospasm, and ptosis have been observed.[1] Nystagmus is exceedingly common, although late in appearing, occurring in fully 50 per cent. of the cases. Nystagmus, although less frequent, may occur in advanced tabes.

According to Uhthoff (*loc. cit.*), paralysis of the ocular movements was present in 17 of his 100 cases of disseminated sclerosis, namely, of the sixth nerve, 6 times; the third nerve, 3 times; the associated movements, 3 times; convergence, 3 times; and complete ophthalmoplegia in 2 cases. The lesion was probably nuclear, and in no case was there complete paralysis of an individual nerve. Coexistent nystagmus was frequent, and nystagmus or nystagmic jerkings were met with in 50 per cent. of the cases.

In paretic dementia, as in tabes, single or combined oculo-motor palsies may be early symptoms, probably, as in the other disease, of nuclear origin. The abducens seems to be most often affected. The palsy may be either temporary, as in tabes, or become complete, all muscles being affected and form one of the four types of ophthalmoplegia.

The significance of certain ocular palsies as the result of diphtheria, rheumatism, diabetes, influenza, poisons, and injuries, must not be forgotten in the investigation of each case.

Prognosis. The prognosis of ocular palsies depends upon the cause, many examples of peripheral and even of basal palsies due to rheumatism or syphilis being readily amenable to treatment. Mauthner's warning, that an apparent rheumatic palsy may be the forerunner of spinal-cord disease, should not be forgotten. In general terms it may be stated that if the primary cause can be influenced by treatment, the prognosis is good; but there are exceptions to this rule. For example, the original cause may beget a structural lesion, which exists so long that by pressure it destroys a definite region, for instance, the nuclei. Under these circumstances power does not return to the paralyzed nerves, even though the cause of pressure is removed.

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[1] Griffith, loc. cit.

The prognosis of nuclear ophthalmoplegia is unfavorable in many instances, fatal in a few, while in a small percentage there is amelioration and even recovery.

Much dispute has arisen in regard to the *relative frequency* of the paralysis of the muscles, most authors agreeing, however, that paralysis of the abducens is met with most frequently, next in order being unilateral paralysis of the oculo-motor. After these come paralysis of the superior oblique, inferior rectus, superior rectus, internal rectus, and inferior oblique. According to Duane,[1] isolated paresis of the superior rectus stands next in order to that of the external rectus, surpassing even paralysis of the superior oblique in frequency.

Treatment. In syphilis the usual remedies are applicable, and in many instances the best results follow large doses of iodide of potassium. These, however, should not be given to the neglect of mercury, a good plan being to follow the inunctions with massive doses of the drug named. In recent times there has been a revival of the treatment of syphilis by intra-muscular injections of mercury, various preparations finding favor with different surgeons. Thus far the author's experience with these injections has been an unsatisfactory one, but is not yet sufficiently large to form a definite opinion.

The annoyance of double images may be prevented by covering the affected eye with a patch or a piece of ground-glass mounted in a frame. Sometimes prisms may be utilized to fuse the images. In order to stimulate the weakened muscles, mechanical treatment has been suggested, especially by Michel, the conjunctiva being seized near the insertion of the affected muscle with forceps, and the eyeball drawn forcibly beyond the ordinary limit of contraction, and then back again. Electricity may be of occasional service. Ordinarily, one pole, the cathode, is placed upon the closed lid, while the other is put upon the temple. Usually a current of more than three milliampères is unbearable, especially if the pole is placed directly over the insertion of the muscle in the sclera. Of the drugs used for the purpose of stimulating the nerves, strychnine deserves the first rank. It should be given in full doses, if necessary, by the hypodermic method. The suspension treatment of ataxia is said to have been followed by relief of the ocular disabilities of this disease, not only in the optic nerve, but also in the muscles. The surgical measures for the relief of the diplopia of paralyzed muscles belong to ophthalmic treatises, to which the reader is referred. Necessarily all medication must be determined by the cause; for example, salicylic acid in rheumatic palsy.

Spasmodic Strabismus and Spasm of the Ocular Muscles. The different types of conjugate deviation which arise under the influence of a paralytic and convulsive lesion have been referred to on page 788. True spasmodic convergent strabismus occurs in certain types of cerebral disease, particularly inflammation at the base of the brain or in the meninges, and should not be confused with the periodic concomitant squint of hypermetropia.

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[1] Archives of Ophthalmology, 1894, vol. xxiii. p. 61.

Spasms of individual ocular muscles (with or without painful sensations) are rare, but they have been described with several conditions, for example, by Gowers in chorea, by Samelsohn (of the superior recti) in tubercular disease at the base of the brain, and by a number of writers in hystero-epilepsy, in one instance, at least, associated with periostitis of the jaw.[1]

Hysteria is responsible for several well-marked anomalies in the movements of the ocular muscles, and those which belong to the oculo-facial group, the most usual being blepharospasm, convergent strabismus, ptosis, and the so-called abducens palsy, although it is doubtful, as Mauthner suggests, whether the term "palsy" is at all applicable to these cases which, in the main, partake of a spasmodic nature. The convergence in hysterical strabismus is extreme, the cornea being almost buried beneath the inner commissural angle. Divergence is said not to occur, and in three marked cases studied by the author, although the spasmodic movements were both up and in and usually associated with intermitting blepharospasm, there was never outward deviation; in one there was marked diplopia.

Partial convulsions of the ocular muscles have been described by Gowers,[2] one case, in which the left eye moved outward near to the external canthus, the right remaining still, the attack being associated with blinking of both eyes, and when it had ceased, slight drooping of the left eyelid, being attributed to disease in the centres for the movements of the eyes in the pons, or beneath the corpora quadrigemina.

Spasm of the levator, from irritation of the fifth nerve, is a phenomenon occasionally observed. Synchronous movements of the upper lid and the maxilla have been reported in a number of instances since Helfreich and O. Bull described examples of unilateral paresis of the levator in which opening of the mouth produced an involuntary raising of the lid. This condition has been explained by Gowers by assuming that the levator fibres of the third nerve in these cases arise from the motor nucleus of the fifth nerve. Bull,[3] however, dissents from this explanation, and prefers to regard the contraction of the levator as an associated or reflex movement.

NYSTAGMUS. This phenomenon is characterized by a rapid involuntary movement of the eyeballs, usually from side to side, but sometimes in a vertical or rotary direction; it may be congenital or acquired, and is bilateral in the vast majority of cases. In the few instances of unilateral nystagmus which have been reported the movements were usually in the vertical direction. The extent of the movement varies from one to ten millimetres, and its frequency from sixty to two hundred oscillations per minute (Gowers). Sometimes the movements are so slight that they are detectable only by watching the fundus of the eye with the ophthalmoscope.

Congenital nystagmus and that type which occurs from diseases of the eye or the pursuance of certain occupations, for example, mining, is not pertinent to the present subject.

From the standpoint of neurology, however, inasmuch as nystagmus is exceedingly common in diseases of the nervous system, the symptom may be of importance. It occurs in fully 50 per cent. of the cases of disseminated sclerosis and Friedreich's ataxia, and sometimes in advanced tabes (see also p. 791). As a differential diagnostic point between disseminated sclerosis and hysteria, it is a very important phenomenon, as it does not occur in functional diseases; indeed, as Dr. Gowers states, it may be the only marked symptom of the early stage of degenerative disease when other signs are still in abeyance.

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[1] Consult also Mauthner: Diagnostik und Therapie der Augenmuskellähmungen, 1889, p. 628

[2] Transactions of the Ophthalmological Society of the United Kingdom, vol. iv. p. 307.

[3] Archives of Ophthalmology, vol. xxi. p. 354.

As a localizing sign, however, in the present state of our knowledge, it is not of much value, because it may be seen with almost any of the many cerebral diseases. It has been noted in focal disease and in diffuse degenerations, with hemorrhages, tumors, inflammations of the meninges and in lesions in many regions of the cerebro-spinal axis. There is no doubt that it is especially common in tumors of the cerebellum, and Gowers states it is more frequent in organic brain disease when there is impairment of sight from optic neuritis than when this is not present. It has been reported in conjugate deviation with disease of the pons, and may assume a convulsive type.

Sometimes nystagmus is associated with other movements, although, curiously enough, it is rare in diseases attended with tremor other than those which have been mentioned. Vertical nystagmus, for example, may be associated with a movement of the levator, or with a hippic oscillation of the pupil (Jessop). Occasionally it is developed only when the eyes are moved in the line of direction of a weakened muscle, the movements then being rather jerky—the so-called nystagmic jerks.

Nystagmus has been ascribed to chronic fatigue of the muscles, oscillation of the globe consequent upon the muscular atony, and also to a central origin. In some cases it is probable that both explanations are correct. Risien Russell's experiments[1] with nystagmus caused by removal of portion of the cerebellum indicate that there are two varieties, one an irritation phenomenon and the other a paralytic condition due to weakness of the muscles producing rotation in any direction, or of their opponents. Its analogy to vertigo, especially in those cases in which external objects appear to move in accord with the ocular movements, has been pointed out by Gowers.

AFFECTIONS OF THE INTRA-OCULAR MUSCLES
(Iris and Ciliary Muscle).

Anatomico-physiological Considerations. Slightly toward the nasal side of the iris, the stroma of which is composed essentially of bloodvessels and a cellular mesh-work, is the *pupil*. Close to its margin is a ring, about one millimetre broad, of smooth, muscular fibres—the *sphincter iridis*, which lies near to the posterior surface of the iris. Beyond the sphincter, lying between posterior pigmented epithelium and the vascular layer, resting upon the former, is a thin layer composed of fibres having a radial direction from the ciliary to the pupillary margin. This is regarded by some authors as muscular in structure, and having the properties of a dilating mechanism, hence the *dilator iridis*; but by others it is considered as a specially developed limiting membrane without such physiological properties.

The vascular supply of the iris is rich, the arteries being derived from the long and anterior ciliary arteries and the vessels of the ciliary processes.

The nerve supply of the iris is derived from the ophthalmic division of the fifth, the third nerve, and the sympathetic, as follows: The ciliary ganglion is connected by its short, or motor root, with the third nerve, by its sympathetic root with the cavernous sympathetic plexus and cervical sympathetic nerve, and by its long, or sensory root, with the nasal branch of the ophthalmic division of the fifth. The short ciliary nerves supply the iris and the ciliary muscle, and the long ciliary nerves are also distributed to the iris. The filaments from the motor (short) root go to the circular fibres (sphincter), and those from the sympathetic to the radiating (so-called dilator) fibres. The motor sympathetic fibres come from the superior cervical ganglion and run in the carotid plexus, and are influenced by a spinal centre corresponding to the lowest part of the cervical cord (about the seventh cervical and first dorsal vertebra)—the so-called *cilio-spinal centre of Budge*.

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[1] Loc. cit.

The ciliary muscle, composed of unstriped muscular fibres, is the chief agent of accommodation. Its nervous mechanism is thus summarized by Foster: "The ciliary muscle is governed by fibres which may be traced through the short ciliary nerves and in the ciliary ganglion, along the third nerve to a centre which lies in the extreme front of the floor of the aqueduct, or, rather, in the extreme hind part of the floor of the third ventricle, and which is especially connected with the extreme front of the nucleus of, and so with the anterior bundles of, the third nerve."

The relation of the various segments of the nucleus of the oculo-motor to the muscles has been referred to on page 774, and we know that the centres for the sphincter of the iris, accommodation, and convergence lie close to each other in the floor of the aqueduct.

The Normal Pupil. The size of the pupil in health varies with exposure to light and with accommodation and convergence. There is no physiological standard on which to base a measurement, the average diameter being a little over four millimetres. The position of the pupil, as already stated, is a little to the nasal side, and under equal illumination the pupils should be round and of equal size. The diameter of the pupil varies somewhat with the age of the patient, being usually larger in the young and smaller in the old, and also with the refractive condition of the eye.

Measurement of the Pupil. The pupil may be measured approximately by holding before it a rule marked in millimetres and noting the number of spaces its width occupies, or one of the variety of instruments known as pupillometres may be employed, a serviceable device being one which consists of a scale of circles held close to the observed eye, the scale being slowly rotated until that circle which matches the pupil in size is reached.

It is much to be desired that in making examination of the pupils a uniform light should be employed and the character of the light stated, and it is much to be regretted that in the recorded examinations such loose statements as "pupils dilated," "pupils contracted," "pupils medium-sized," have been frequently employed.

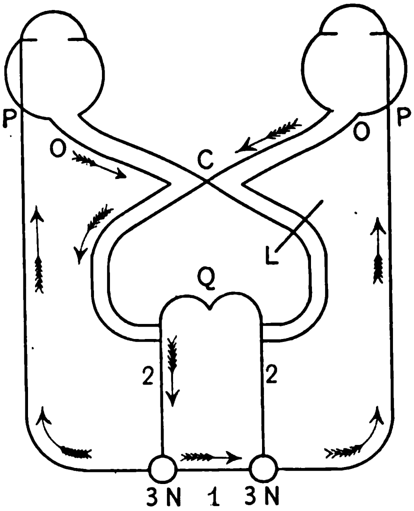
Method of Testing the Pupillary Reactions. Before attempting to record pupillary phenomena all errors of accommodation and convergence should be eliminated, and the absence or presence of attachment between the iris and the capsule of the lens (synechiæ), or immobility from atrophy of the iris, should be ascertained. Numbers of errors utterly vitiating the value of many recorded cases have occurred from negligence in regard to these points.

The light employed for testing the sensitiveness of the retina or visual centre should not be more intense, as Turner well observes, than that to which the eye is usually accustomed. Therefore, examinations made by a light reflected from a mirror, or by passing a flame in front of the eye, are not accurate, except under special conditions presently to be described. The following is the method:

The patient is placed before a window in diffuse daylight and one eye carefully excluded. He is directed to look into the distance with the exposed eye, which is then shaded, and if it is normal a considerable dilatation of the pupil will occur. On removal of the covering hand or card, when the light strikes the retina a contraction to the same size as that which existed before the test was applied takes place. This is the *direct reflex action of the pupil,* and is brought about by a muscular contraction of the sphincter of the iris following the stimulation of the optic nerve. If during this examination the other pupil, which has been shaded by a card or a covering hand, is observed, it will be found it has acted in unison with its fellow. This is the *consensual or indirect reflex action of the pupil.* Therefore, in normal eyes the pupils should be equal, not only with both eyes open, but with one eye shaded.[1]

Contraction of the pupil, or the response of the iris which occurs when the eye is exposed to light, is a reflex phenomenon, in general terms brought about by an impulse passing along the optic or afferent nerve to the third or efferent nerve, the centre being in the aqueduct at a level with the anterior corpora quadrigemina. The exact pathway of the reflex, however, is not definitely determined. Thus, light falling on one eye starts an impulse along the optic nerve, which, owing to semi-decussation of the fibres in the chiasm, passes with equal facility along each tract to the corpora quadrigemina, and thence by the communicating fibres between these bodies and the third nerve centre (Meynert's fibres) to the special centre of the sphincter pupillæ (pupil-contracting centre), and from there by the ciliary branches to each ciliary ganglion, the ciliary nerves and circular iridic fibres. This would explain the contraction (direct reflex) in the illuminated as well as that (consensual) in the non-illuminated eye. Fibres directly connecting the third nerve-centres are probably also present (Fig. 274).

FIG. 274.



3 N, centre of third nerve; 1, connection between nuclei of third nerves; 2, Meynert's fibres; Q, corpora quadrigemina; C, chiasma; O, optic nerve; P, myotic fibres of third nerve; L, seat of lesion; arrows show path of impulse in lesion of right tract at L. (SWANZY.)

Many other connections have been described, one of which, that given by Mendel (quoted by Swanzy), may be cited: Optic nerve, optic tract, to ganglion habenulæ of the same side, thence by the posterior commissure to the nucleus of the third nerve, and thence to the ciliary nerves. This may be spoken of as the optic oculo-motor reflex, and represents the sensory motor arc of the pupil.

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[1] The iris response to the stimulus of light should also be tested with both eyes open and exposed to the source of illumination; they are then alternately covered and exposed and the reactions noted.

If the eyes are directed to a near object—for example, the point of a pencil—contraction takes place under the influence of accommodation and convergence; that is to say, the sphincter of the iris contracts in association with the ciliary muscle and the internal recti. This is the *associated action of the pupils.* Accommodation increases pupillary contraction, but this does not take place under the influence of accommodation unassociated with convergence. It does occur with convergence without the act of accommodation. The nervous mechanism has been described on page 795.

Contraction of the pupil can also take place after division of the cervical sympathetic, section of the spinal cord (cilio-spinal centre), or depression of the pupil-dilating centre in the medulla (front part of the aqueduct).

Dilatation of the Pupil. The mechanism of dilatation is still in dispute, the three most prominent theories being that it is caused by the action of the sympathetic vaso-constrictor nerves, or by contraction of the radially placed muscular fibres (dilator pupillæ), or by inhibition of the sphincter through the action of the sympathetic, which presupposes the presence of elastic tissue in the iris (Gaskell). Recently Langley and Anderson[1] have reviewed the evidence, and the indications are that a dilator muscle is demonstrable.

The long ciliary nerves are known as the mydriatic nerves. They arise from the front of the aqueduct and are in connection with the cilio-spinal centre; they pass out with the first two dorsal nerves, reach the cervical and cavernous sympathetic and Gasserian ganglion, and finally the eye via the ganglionic branch of the fifth, the ciliary ganglion and its branches of distribution. The pupil-dilating centre is in the medulla and is very sensitive to stimuli of all kinds, *e. g.*, irritation of the skin of neck (*skin reflex*), the emotions, and deep respiration. Independently of the action of dilating fibres in the iris, as Foster puts it, the dilating mechanism is apparently tonic in nature, but subject to augmentation from various causes, and the cervical sympathetic is the efferent channel.

THE PUPIL IN DISEASE. While investigating the pupil-changes (see page 795), as William McEwen[2] insists, five possibilities should suggest themselves, namely: Are they caused by (*a*) the action of a drug; (*b*) ocular disease or optical defects; (*c*) spinal or sympathetic lesion; (*d*) localized cerebral lesion in special centres or tracts; (*e*) abeyance of brain function; (*f*) cerebral irritation.

In order to localize that portion of the reflex ring which is affected, this may be divided, according to Magnus[3] and other authors into the following three portions:

1. *The centripetal part, including the optic nerve, chiasm, tracts and connecting fibres to the cortex.* If the disease has rendered one optic nerve, for example, the right, impermeable to the transmission of impulses, the pupil on that side fails to react to light, but reacts normally when light falls upon the left or opposite eye; the left pupil has a normal light reaction, but fails to contract when light falls upon the right eye. The reactions to convergence and accommodation are normal.

Lesions affecting the chiasm and the tract are accompanied by hemianopia and the special pupillary phenomena which belong to this condition, while lesions, in the optical pathway between the corpora quadrigemina and the cortex, while accompanied by probable changes in the visual fields, are unassociated with pupillary disturbances.

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[1] Journal of Physiology, vol. xiii. No. 6. See also Juler's demonstration, Trans. Eighth International Ophthalmological Congress, 1894, p. 67.

[2] The Pupil in its Semeiological Aspect, American Journal of the Medical Sciences, new series, vol. xciv., July to October, p. 123.

[3] Klin. Monatsbl. f. Augenheilk., xxvi. p. 255.

2. *The part of the reflex ring which carries the light impulse from the corpora quadrigemina to the oculo-motor nuclei (Meynert's fibres).* If both sides are affected, neither pupil reacts to the impulse of light falling on either eye, but there is normal reaction to accommodation and convergence. (See Argyll-Robertson Symptom, page 800.)

3. *Lesion of the centrifugal portion of the reflex ring, namely, the nucleus of the sphincter of the iris, the third nerve and the termination of the third nerve in the iris* prevents, if the right nucleus is affected, the direct light reflex of the right pupil, and also the indirect reflex, *i. e.*, the one which should occur when light is directed into the left eye. A beam of light directed into the left eye is followed by reaction both in that eye and the eye upon the opposite side, although somewhat lessened in degree. There is normal reaction to accommodation and convergence. The pupils are unequal, the right being the wider.

If the trunk of the right oculo-motor is affected there is pupillary immobility under the influence of light directed into the right eye, and also when it is directed into the left eye, as well as loss of accommodation upon the right side. Light falling into the left eye produces normal reaction—a reaction which is also manifested on this side if the light is directed into the opposite eye. The pupils are unequal, the right being the larger. Similar conditions arise if the peripheral fibres of the oculo-motor at their termination in the iris are affected upon one side.

We have now to consider a little more in detail:

1. *Dilatation of the Pupil (mydriasis).* This occurs in ocular disease, for example, glaucoma, in cases of non-conductivity of light (optic nerve atrophy), in orbital disease, and under the influence of mydriatic drugs. It is further seen in fright, emotion, anæmia, in depressed nervous tone, neurasthenia, aortic insufficiency and irritation of the cervical sympathetic. It is noted in vomiting, forced respiration, and anæmia of the brain, for example, syncope, and is said to be present in those of low mental development, for example, idiots.

In disease of the nervous system dilatation of the pupil, when of cerebral origin, indicates extensive lesion; when of spinal origin, irritation of the part (McEwen). Systematic writers have divided dilatation into *irritation mydriasis*, caused by irritation of the pupil-dilating centre or fibres, and *paralytic mydriasis*, caused by paralysis of the pupil-contracting centre or fibres, or because stimulus is not conducted from the retina to the centre.

The former is apt to be seen in hyperæmia and irritation of the cervical portion of the spinal cord, in spinal meningitis, in cases of tumor of the spinal cord, and also, under certain circumstances, in tumor of the cerebral contents, in psychical excitement, for example, acute mania, and in tabes dorsalis and progressive paralysis of the insane. (See pages 799 and 800.)

The latter, which is also known as *iridoplegia*, is found in disease at the base of the brain affecting the centre of the third nerve, in pressure of the cerebrum when in great amount, as from hemorrhage, tumors, the late stages of thrombosis of the sinuses, or large abscesses; also in the late stages of meningo-encephalitis. It is said to be present in acute dementia when there is œdema of the cortex, and is found in cerebral softening. Hemorrhage into the centrum ovale and cerebral peduncles also produces mydriasis (McEwen).

2. *Contraction of the pupil (myosis)* appears in congestion of the iris, paralysis of the sympathetic and also of the fifth nerve, in certain fevers, in plethora, venous obstruction, mitral disease and under the influence of myotics.

If the myosis is of cerebral origin, it indicates an irritative stage of the affection; if of spinal origin a depression, paralysis, or even destruction of the part (McEwen). Systematic writers divide contraction of the pupil into *irritation myosis*, caused by irritation of the pupil-contracting centre or fibres, and *paralytic myosis*, caused by a paralysis of the pupil-dilating centre or fibres, or by a combination of both.

The irritation myosis, as just noted, is found in the inflammatory affections of the brain and its meninges, *e. g.*, meningitis, abscess (at the first myosis on same side as lesion) and beginning sinus-disease. According to the rule previously given, myosis may change to dilatation if the products of disease become excessive; hence the serious prognostic import of mydriasis under these circumstances. Myosis is seen in the early stages of cerebral tumor, in small hemorrhages into the cerebellum, and at the onset of cerebral apoplexy. Berthold, quoted by Swanzy, uses myosis as a diagnostic symptom between apoplexy and embolism. McEwen points out that the convulsions arising from meningo-encephalitis are accompanied by myosis, while those due to epilepsy are usually associated with mydriasis. Apoplexy of, or pressure upon, the pons is associated with myosis.

Paralytic myosis, often known as *spinal myosis*, occurs in lesions above the dorsal vertebræ. It is especially noteworthy in tabes dorsalis. At first the pupil reacts to light and convergence, but later exhibits the Argyll-Robertson phenomena (see page 800). Paralytic myosis is also met with in paralysis of the insane, pseudo-dementia paralytica of syphilitic origin, bulbar palsy when complicated with progressive muscular atrophy or sclerosis of the brain and spinal cord, and, according to Mills, in some forms of multiple neuritis.

Unequal pupils are rarely seen in health, although it is stated by one observer (Iwanow) that among one hundred and thirty-four healthy military recruits, the right pupil was larger in forty-nine and the left in fifty-three, equal width being found in only twelve. If there is recent wide dilatation of one pupil and no disease of the eye, the instillation of a mydriatic may be suspected. Unequal pupils occur in eyes with widely dissimilar refraction, if one eye is blind, in aneurism, dental disease, traumatism, and in diseases of the nervous system. If the disease is cerebral, unequal pupils denote unilateral or focal brain disease. They are not uncommon in tabes, disseminated sclerosis, and paralytic dementia.

Varying inequality of the pupil, or a mydriasis, now occurring on the one side and now on the other, is, according to Von Graefe, a serious premonitory symptom of insanity.[1]

The pupillary phenomena of certain well-known diseases, already several times referred to, are so important that the following special paragraphs are introduced.

The Pupils in Locomotor Ataxia. Unequal pupils, either with mydriasis or myosis, or of medium size, occur in fully 27 per cent. of the cases, according to Berger, a percentage which agrees entirely with the author's observations. They are slightly more frequent in the initial stage of the disease.

The pupil is often elliptical or pear-shaped, commonly associated with myosis, and, perhaps, as Berger suggests, this is due to palsy of the iris vessels, varying in different meridians. The iris reacts peculiarly to mydriatics, which dilate such a pupil (spinal myosis) only partially, and their effect is for a long time manifest. Myotics, however, contract it *ad maximum.* Heddaeus[2] states that the ordinary form of small pupil in reflex iridoplegia is dilated readily with cocaine. This indicates that its narrowness is due to stimulation of the third nerve and not to palsy of the sympathetic. It is possible that a spastic myosis may also occur in tabes. Irritation or spastic mydriasis may appear on account of irritation of the posterior columns, especially in the initial stages of tabes, while paralytic mydriasis is often one of its earliest symptoms.

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[1] The author is especially indebted to Swanzy's article, "The Motions of the Pupil in Health and Disease," Diseases of the Eye, 4th edition, chapter xi., in the prapartion of the sections devoted to the anomalies of the pupil.

[2] Archives of Ophthalmology, vol. xxiii. p. 8.

In tabes dorsalis the light reaction is first lost; later, there may be loss of reaction to accommodation, a peculiarity which is also present in the progressive paralysis of the insane, sometimes in cerebral syphilis, and is said to be produced by certain poisons, for example, bisulphide of carbon (Uhthoff).

Normal pupillary reactions are not common in ataxia, Berger having found them in only 3.7 per cent. of his cases, and he gives the following table to show the various phenomena which are present:

	In the stadium.		
	Preataxia.	Ataxia.	Paralytic.
Good reaction of the pupil	8	1	0
Gowers'[1] symptom	6	2	0
Slow light, good accommodation reaction	2	1	2
Argyll-Robertson symptom, unilateral	0	2	0
" " " bilateral	10	24	18
Pupil irresponsive to light and accommodation unilateral	0	1	2
" " " " " " bilateral	0	11	10

With the spinal myosis, the light reflex, and the contraction associated with accommodation and convergence may be preserved, when the lesion is still confined to the cilio-spinal region, or perhaps somewhat higher up.

Reflex Iridoplegia is the name given to a pupil responding only slightly or not at all to the light impulse, and when this exists without loss of the associated action of the iris (contraction in accommodation and convergence), the term "Argyll-Robertson symptom" is applied to the phenomenon. It may occur in any stage, being present in fully one-half of the patients. It is associated with mydriasis, myosis, medium-sized and unequal pupils, and may be unilateral or bilateral. The pathological lesion which determines the presence of this phenomenon is situated in the fibres which pass from the proximal end of the optic tracts to the oculo-motor nuclei of the same side. Turner,[2] however, prefers to assume that a single lesion situated in the Sylvian gray matter forming forepart of the oculo-motor nucleus[3] is the cause of both the myosis and the reflex iridoplegia.

Unilateral Reflex Iridoplegia, that is, a condition when one pupil is unaffected by varying degrees of illumination of both eyes, but acts to accommodation, the unaffected pupil responding to separate light stimulus of either eye, may exist with or without mydriasis, but usually (always according to Heddaeus) is wider than its fellow. It is not a common phenomenon, and is seen in tabes, and probably may be due to syphilis. It was formerly regarded as indicating a lesion affecting the iris-branch of the third between the sphincter and nucleus, but by Moebius and Seggel is regarded as an interruption in Meynert's fibres. Heddaeus (*loc. cit.*) suggests it may be explained by assuming that the iris-branch of the third nerve is formed of two roots, one from the sphincter-nucleus and one from the accommodation-nucleus; if the former is destroyed there is reflex iridoplegia and mydriasis, but preserved accommodative action unless the latter is also affected. This phenomenon should be distinguished from unilateral reflex blindness, *e. g.*, in embolism of central artery of retina (Heddaeus), in which there is pupillary response to light directed into both eyes, or into the unaffected eye, but failure of response in either pupil when light falls on the blind eye (see also page 797).

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[1] By Gowers' symptom Berger means after a marked contraction of the pupil a dilatation, followed by vacillation.

[2] Royal London Ophthalmic Hospital Reports, vol. xiii. part 3, page. 344.

[3] His studies have led him to suggest the following subdivision of the forepart of the oculo-motor nucleus: 1. A sphincter-inhibitory centre, closely associated with a sphincter-contracting centre; which two subserve the pupillary light reflex. 2. A centre for accommodation. 3. A centre for contraction of the pupil with convergence, in close association with the centre for the associated action of the internal recti muscles.

Schwarz[1] has recorded a case of right incomplete reflex iridoplegia and left incomplete accommodation palsy, a most unusual and perhaps unique observation, and accepts Heddaeus's explanation of the two roots of the iris-branch of the third nerve.

Occasionally the converse of the Argyll-Robertson symptom is observed, both with and without signs of tabes, *i. e.*, the pupils react to light, but contraction fails during attempts at convergence. This indicates disease in a special part of the oculo-motor nucleus, as, for example, in a case of Turner's (*loc. cit.*) with lesion in the second and third subdivisions of the nucleus (see foot-note, also Heddaeus's explanation, p. 800). According to Gowers, when the Argyll-Robertson pupils are not small, the skin-reflex persists, and when they are small, it is lost, a fact which Turner regards as confirming his view. This explains contracted Argyll-Robertson pupils, which have been observed without evidence of sclerosis of the cervical region of the cord.

Abolition of accommodative reaction, with preservation of normal power of accommodation and normal reaction to light, according to Heddaeus, has never been observed (see page 797).

The Pupils in Paralytic Dementia. Abnormal pupillary phenomena are common in this disease, and consist of inequality, sometimes of varying inequality, and of reflex iridoplegia.

Inequality of the pupils is seen not only in progressive paralysis of the insane, but in various types of mental disorder, and has been estimated to occur in half of the cases. Certainly this percentage is correct for paralytic dements. Uhthoff, who examined 4000 cases of mental disease, found 492 examples of reflex iridoplegia; of these 421, or 85.5 per cent., were paralytic dements.

Irregularly shaped pupils and irregular pupillary reactions are frequent; indeed, it is said that they exceed in frequency inequality of the pupil. Finally, accommodation may also fail and the pupil become motionless both to the stimulus of light and convergence (absolute iridoplegia). The dilatation upon irritation of the skin may be preserved for a long time. According to W. Bevan Lewis,[2] however the sequence of the morbid phenomena in the iris in this disease is (*a*) paralysis of reflex dilatation to cutaneous stimulation, (*b*) reflex iridoplegia, (*c*) partial and occasionally complete interior ophthalmoplegia.

Pupils in Epilepsy. The pupils of epileptics are not infrequently unequal, Browning[3] concluding that, on an average, one in every five or six epileptics will have some, perhaps only slight, inequality of the pupils. He divides the pupillary phenomena in this respect into three grades: decided inequality, probably depending upon localized intra-cranial trouble; slight but fairly constant inequality; and the so-called latent anisocoria, in which the inequality is evident only on faint illumination, a condition not uncommon in diseases other than those analogous to epilepsy.

PARALYSIS OF THE CILIARY MUSCLE. This may be present, and is known as *cycloplegia*, without co-existing affection of the pupil, and is most common after diphtheria. Sometimes there is slight mydriasis. As already noted, cycloplegia, with preservation of the light reflex of the pupils is seen in locomotor ataxia and in other allied pathological conditions. These cases probably depend upon affection of the centres for accommodation.

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[1] Centralblatt f. Prakt. Augenheilk, Dec., 1894.
[2] Trans. Ophth. Soc. United Kingdom, vol. iii. p. 219.
[3] Journal of Nervous and Mental Disease, January, 1892.

When both iris and ciliary muscle are paralyzed—as, for example, in third nerve palsy—we have the condition known as *cycloplegia with mydriasis.*

Ophthalmoplegia interna, or, in other words, cycloplegia and total iridoplegia, as intimated in preceding paragraphs, may be the ultimate result of a condition which begins as an ordinary failure of the reflex mobility of the pupil. It has always, as is well known, been described as a primary condition, and was attributed by Mr. Hutchinson to disease of the ciliary ganglion, and by Hulke to lesion of the intra-ocular ganglionic cells. We know from anatomical examinations that it is of nuclear origin, and may or may not be associated with an external ophthalmoplegia, and may result from allied lesions and causes (see page 784).

CERTAIN SPECIAL PUPILLARY PHENOMENA. *Hemiopic Pupillary Inaction.* This phenomenon, first described by Heddaeus in 1880, and designated by Wernicke (sometimes called Wernicke's symptom) hemiopic pupillary reaction sign, is the means for determining whether or not a lesion lies between the optic chiasm and the corpora quadrigemina or further on in the visual pathway.

Given a case of lateral hemianopsia, the examination is made as follows: One eye being carefully excluded, the patient being seated in a dark room, with the source of light somewhat behind him, the eye under examination is illuminated by a weak light reflected from a plane mirror. The observer then reflects a more intense beam of light by means of the concave mirror of the ophthalmoscope into the pupillary space, care being taken that the light falls obliquely, and is not diffused over the entire retina. If the beam of light falling upon the blind side of the retina causes no contraction of the pupil, it is assumed that the lesion is in that portion of the sensory motor arc of the pupillary reflex included between the chiasm and the corpora quadrigemina. If there is reaction of the pupil when the light strikes both the seeing and the blind side of the retina, the lesion is further on in the visual pathway. The examination must be made with great care.

It may be a transient, permanent, or recurrent phenomenon. Rothmann,[1] who has observed a transient case, concludes that this sign, when persistent, indicates basal disease in the region of the tracts, but, if transient, a distant lesion beyond the origin of the reflex fibres. Hemianopsia without the sign is presumptive evidence of disease outside the reflex arc, while late appearance of this pupil symptom in hemianopsia may mean secondary degeneration. Hemianopic inaction of the pupil may be present without hemianopsia if there is lesion between the third-nerve nucleus and its related tract on one side. Henschen's[2] conclusions, based upon a most careful study of the literature, as well as observations of his own, are as follows:

1. Hemiopic reaction (H. R.) does not appear with softening of the occipital, parietal, or temporal lobes, even when it is extensive, or has reached the neighborhood of the corpora geniculata, and is absent with tumors of these regions, even when they have destroyed the optic radiations, and pressed on the pulvinar or the corpora quadrigemina.
2. Mere pressure on the tract may cause the reaction.
3. Lesions of the tract produce the reaction, as a rule, even when very minute.

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[1] Deutsch. med. Wochenschrift, 1894, No. 15.

[2] Beiträge zur Pathologie des Gehirn's. Teil, III. s. 100-115. See also abstract in Ophthalmic Review, December, 1894.

4. Lesion of the outer corpus geniculatum seems not to produce the reaction.

5. The effect of lesions of the inner corpus geniculatum is uncertain.

6. Destruction of the pulvinar does not cause the reaction.

7. Destruction of the posterior corpus quadrigemina does not produce it.

8. Lesions of posterior segment of thalamus and pulvinar do cause the reaction—perhaps from pressure on the tract, or by destroying the brachium anterius.

9. Lesions of the chiasma produce it, though occasionally, for some unknown reason, it is not present.

10. It may occur in injury to the nerve, with monocular hemianopsia.

The Cerebral Cortex Reflex of the Pupil. If one sits in a darkened room a marked bilateral pupillary contraction will occur, without change of accommodation or convergence, if only the attention is directed to a bright object already present within the compass of the field of vision. The brighter the object the more pronounced the contraction.

Haab believes that this reflex must be cortical in nature. It is lessened in patients who are likely to have reflex iridoplegia, namely, tabetics and dements.

Hippus, or a rhythmical contraction and dilatation of the pupil, occurring without alteration of illumination or fixation—a normal phenomenon for a few seconds after light stimulus to the retina and optic nerve—is present in various nervous diseases. Damsch has reported the condition in multiple cerebro-spinal sclerosis, disseminated sclerosis, and in neurasthenia. In psychical disturbances, epilepsy, and in the early stages of acute meningitis and palsy of the oculo-motor it has also been observed. The most marked examples the author has seen were in a patient with recurrent mania and in one of grave hysteria.

The Skin Reflex. This is the second reflex action of the iris, the other being its contraction under the stimulus of a beam of light, and consists of a dilatation of the pupil when some cutaneous nerve is stimulated, especially in the region of the skin of the neck. The motor path for this action is in the cervical sympathetic, and in the connecting fibres with the spinal cord in the cervical region, and the centre probably beneath the corpora quadrigemina. The reflex is lost in disease of the cervical sympathetic and in certain lesions, especially those impairing sensibility in the upper portion (cervical) of the spinal cord.

Section of the fifth nerve is followed by pupillary phenomena similar to those seen in paralysis of the sympathetic, due probably to the fact that the trigeminus contains sympathetic fibres which are thus paralyzed, and a direct influence of this nerve upon a contraction of the pupil has been described, but, as Swanzy points out, this should be regarded purely as a reflex action.

In certain cases of reflex pupillary immobility, anæsthesia over portions of the distribution of the fifth cranial nerve will be found. This, as Turner insists, is suggestive of the presence of a sclerotic lesion, either in the trunk of the nerve or in the distribution of its roots, and he believes that the cause of this anæsthesia is connected with the distribution of the so-called ascending root of the fifth nerve. Such anæsthesia, then, in connection with pupillary phenomena, is strongly suggestive of early progressive degenerative lesion in the upper portion of the central nervous apparatus.[1]

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[1] Consult Turner, loc. cit., p. 342.

CHAPTER XXVII.

DISEASES OF THE CRANIAL NERVES

(CONTINUED).

BY C. A. HERTER, M.D.

MANY of the disorders of the functions of the cranial nerves are brought about by brain disease and not by disease located in the nerves themselves. Strictly speaking, disturbances arising in this way should not be treated in a chapter dealing with the diseases of the nerves, but, owing to the fact that the disturbances of function arising from central disease are of the same kind symptomatically as those due to peripheral disease, it is convenient in practice to touch upon the former in speaking of the latter.

AFFECTIONS OF SMELL.

ANOSMIA. Anosmia, or loss of the sense of smell, is much more often the result of disease of the olfactory mucous membrane (chronic rhinitis, nasal polypi) than of disease of the nerve or brain, and such local processes must be carefully excluded before a nervous lesion can be seriously considered. Any out of the following conditions may lead to anosmia: mere excess of nasal secretion; defective secretion, or changes in the mucous membrane, such as may occur in paralysis of the fifth nerve; blows on the head which mechanically tear the olfactory filaments from the bulb (very rare cause); pressure on the nervous bulb by tumor in the anterior fossa of the skull, or by caries of adjacent bone, or by syphilitic meningitis or by hydrocephalus; primary degenerative changes associated with locomotor ataxia; primary senile atrophy of the bulbs; congenital absence of the nerves; excessive olfactory stimulation.

Disease of the sensory part of the internal capsule (posterior limb) has been known to cause anosmia on the side opposite the lesion, and an extensive cortical or subcortical lesion in the area of the middle cerebral artery may produce the same result. Disease of the tip of the temporo-sphenoidal lobe, involving the olfactory centre, is probably a cause of anosmia. A functional loss of smell occasionally occurs in hysteria and in neurasthenic states.

Diagnosis. The diagnosis of anosmia of nervous origin depends on detection of loss of smell and on exclusion of disease of the mucous membrane. In testing the condition of the sense of smell it is important to make use of substances that stimulate the sense of smell only. It is convenient to use oil of cloves in various degrees of dilution. If there is loss of smell, with ability to distinguish flavors, the trouble is certainly due to disorder of the mucous membrane. In the very rare cases where the ordinary tests of smell do not suffice, an electrical current may be used to stimulate the nerves, a phosphorus-like odor being perceived if the nerves are normal.

Prognosis. The prognosis of anosmia of nervous origin is bad as regards recovery except in functional cases and some syphilitic and some traumatic cases.

Treatment. The treatment of nervous anosmia is practically the treatment of the cause of the anosmia, and is consequently unsatisfactory. But treatment should be attempted in the hope that the nerve-elements are functionally inactive rather than destroyed. One-thirtieth of a grain of strychnine in olive oil may be used as a snuff. A weak galvanic current should be applied with the negative pole to the nasal bones. Occasionally electrical treatment is of actual service.

HYPEROSMIA is an occasional occurrence in hysteria and insanity. It consists of a very remarkable acuteness of the sense of smell, comparable to that normally possessed by some animals. The condition is apt to be associated with a changed appreciation of the character of odors. A physiological hyperosmia is sometimes the result of cultivation, as in the case of the blind and those who follow certain occupations, such as tea-tasting.

Olfactory hallucinations occur in the insane and as the auræ of epilepsy. In rare instances olfactory hallucinations have resulted from tumor or softening in the anterior part of the temporo-sphenoidal lobe. Olfactory hallucinations are usually of an unpleasant character and are generally unassociated with anosmia.

PAROSMIA, or perversion of the sense of smell, is a condition in which all or most things smell alike. It may result from irritative disease of the nerve or brain, but is very rarely so caused. It is not a very uncommon condition in neurasthenics and in persons suffering from digestive derangement. In some persons a dose of purgative salts gives rise regularly to a perversion of the sense of smell, lasting many hours or even days. The odor perceived is usually disagreeable and may have a fecal character. The sense of taste may be coincidently perverted.

AFFECTIONS OF TASTE.

The path by which sensations of taste pass to the brain is still the subject of discussion, but the facts of pathology warrant the following conclusions: 1, that the sense of taste from the anterior two-thirds of the tongue, and in some cases from the back of the tongue as well, is conveyed in a circuitous course by the lingual branch of the fifth, then by the chorda tympani, then by the facial (between the stylo-mastoid foramen and the geniculate ganglion), and lastly by the great superficial petrosal to the second division of the fifth, and by it to the root of the fifth; 2, that the sense of taste from the back of the tongue and from the palate is conveyed by the glosso-pharyngeal nerve in most cases, but is switched off before reaching the glosso-pharyngeal root, to the second division of the fifth, by means of certain fibres of connection between the ninth and the fifth; 3, that the stimuli of taste, both from the anterior two-thirds of the tongue and from the posterior third of the tongue and the palate, having reached the second division of the fifth, pass by it to the root of the fifth, and thence by the fifth to the cortex of the brain (probably the general sensory region).

AGEUSIA, or loss of the sense of taste, may arise from disease in any portion of the taste-path just described. The most common cause of ageusia is facial neuritis between the geniculate ganglion and the point of departure of the chorda tympani from the facial. The loss of taste is then on the anterior two-thirds of the corresponding half of the tongue, and is associated with facial paralysis. Ageusia from peripheral lesions located elsewhere is not common, but disease of the chorda from tympanic disease, or from disease of the second division of the fifth, does not belong to the great rarities. Loss of taste from disease of the glosso-pharyngeal nerve alone probably does belong to the greatest rarities. Complete loss of taste probably never results from disease of the mucous membrane of the tongue, but a partial loss occasionally arises in this way. Loss of taste due to disease of the path within the brain is of rare occurrence. Such loss may, however, occur as part of a general hemianæsthesia from disease of the cerebrum involving the sensory crossway. Much more frequently ageusia forms part of the hemianæsthesia of hysteria. Disease of the pons has been known to cause complete one-sided ageusia under conditions that suggest the proximity of the taste-path in the pons to the motor root and nuclei of the fifth.

The recognition of ageusia is not difficult, but it should be remembered that the patient may be quite unconscious of a unilateral ageusia. The sense of taste includes only the perception of the following kinds of sensations: bitterness, sweetness, sourness, saltiness, and metallic sensations. Solutions of sugar, quinine, salt, and citric acid may be employed as tests, and a weak galvanic current applied to the tip of the tongue may be used as an actual test of the integrity of the nerve-elements. Caution should be used to confine the action of the test solution to the part of the tongue examined, and flavors (which are perceived through the olfactory nerve) should not be confounded with tastes.

The treatment of the symptom ageusia is the treatment of the condition on which it depends, this being most often a facial neuritis. The nerves of taste may readily be stimulated by galvanism, one electrode being placed on the tongue, the other on the mastoid. Such applications seldom do harm, but on the other hand are rarely of much service. In hysterical ageusia faradization of the tongue may be used.

PARAGEUSIA, or perversion of the sense of taste, is sometimes met with in insanity, hysteria, tabes, neurasthenia, and local catarrhal conditions. It consists in the perception of gustatory sensations different from those normally produced, sweet things tasting bitter, etc.

HYPERAGEUSIA, or increased sensitiveness of taste and subjective sensations of taste, occurs under conditions like those in which parageusia occurs. Subjective sensations of taste are sometimes part of the aura of epileptic seizures, and are not rare among the hallucinations of the insane.

AFFECTIONS OF THE AUDITORY NERVE.

ACOUSTIC PARALYSIS—nervous deafness.

Etiology. Deafness due to disturbance or imperfection of some part of the nervous mechanism of hearing, as distinguished from deafness due to middle or external ear disease, may be congenital or acquired. Congenital nervous deafness is the cause of four-fifths of all cases of deafmutism, the remaining one-fifth occurring from disease in early life. Inheritance is a very important factor in the causation of deafmutism, but of the pathology of the condition practically nothing is known. Nervous deafness other than deafmutism may arise from disease in any part of the auditory path, from the labyrinth to the cortex of the brain.

1. Disease of the labyrinth is the cause of the great majority of all cases

of nervous deafness, the morbid process being either primary in the labyrinth or extending to it from the middle ear. The morbid process may be an acute or a chronic inflammation, a syphilitic exudate, a degeneration, or a hemorrhage, or, as secondary changes, fibrous or calcareous degeneration. Exposure to cold, gout, or a toxic blood state may appear to determine the occurrence of the labyrinthine disease. Drugs which, like quinine, are capable of causing deafness probably do so by their action on the labyrinth, by acting either on the nerve-endings of the acoustic nerve or on the structure in which these terminate. A very loud noise, *i. e.*, one with much concussion, may give rise to complete deafness, temporary or permanent, probably by causing hemorrhage into the labyrinth. Persons subject to jarring or continuous noise, as locomotive engineers and boilermakers, not rarely suffer from deafness of labyrinthine origin.

2. Lesions of the auditory nerve, either in the course through the temporal bone or at the base of the brain, are much less frequently the cause of nervous deafness than disease in the internal ear. The following are the chief forms of disease of the nerve: pressure on the nerve from new growths outside the nerve, or from syphilitic or other forms of meningitis, or from narrowing of the meatus, inflammation through extension from adjacent structures, hemorrhage or tumor, or calcareous degeneration within the nerve (very rare), primary degeneration of the nerve (as in locomotor ataxia or senility), and, possibly, primary neuritis and the so-called rheumatic neuritis.

3. The auditory nuclei in the pons are rarely the seat of disease. Occasionally nervous deafness is the result of hemorrhage, acute softening or a new growth involving these nuclei.

4. Disease in any part of the auditory path above the nuclei in the pons is an occasional cause of nervous deafness. Thus disease of the superficial layer of the tegmentum of the crus, or of the sensory portion of the internal capsule, or of the cortex of the temporo-sphenoidal lobe, or of the white substance just beneath the cortex, has been known to give rise to deafness. Functional loss of hearing is met with in hysteria as part of hemianæsthesia, in anæmia (the defect being then partial), and in cases of profuse hemorrhage. In these conditions the cortex is probably the seat of nutritional or vasomotor disturbance.

Symptoms. The most important characteristic of nervous deafness relates to the conduction of sound through the bones of the head. Normally, the vibrations of a tuning-fork can be heard longer through the air than through the bone, *i. e.*, when the subject can no longer hear the tuning-fork held to the skull he can still hear it if it is placed just opposite the external auditory meatus (this is "Rinne's test"). If there is disease of the middle ear, conduction through the bone is better than conduction through the air. But if there is some deafness to sounds conducted through the air, and, notwithstanding this, sounds are heard better through the air than through the bone, the deafness depends on labyrinthine or nerve disease, *i. e.*, it is nervous deafness. The deafness in these cases is especially marked for short sounds of high pitch.

Considerable study has been devoted to the characters of the electrical reactions in nervous deafness, but the changes in reaction found in such cases are neither distinctive nor do they occur in all cases. Moreover, the application of the test is extremely disagreeable. The test is thus impracticable. The position of the disease which causes nervous deafness is to be determined by means of the associated symptoms. If there is facial paralysis with nerve deafness, and there is no evidence of middle-ear or bone disease, the auditory nerve is involved at the base of the brain or in the internal meatus. Deafness of sudden onset, associated with paresis of the arm and leg of the opposite side, and other bulbar or pontine symptoms, is probably due to disease of the auditory nuclei, but if these bulbar symptoms exist with deafness of slow onset this inference cannot be made. If nervous deafness is associated with hemiplegia or hemianæsthesia on the same side the lesion is probably in the internal capsule, in the cortex, or just beneath the cortex.

Bilateral nervous deafness is less common than unilateral affection, but is nevertheless common. Its chief causes are bilateral disease of the labyrinth (common), symmetrical disease of the auditory nerves (very rare), destructive lesions of the superficial layer of the crustæ of the crura (very rare), and disease of the auditory centre in each temporo-sphenoidal lobe (very rare).

Treatment. The treatment of deafness due to labyrinthine disease is described in text-books of otology. When the symptom is due to a central or nerve lesion the principles which govern treatment are those which relate to the treatment of the same process elsewhere. When deafness is acute in onset counter-irritation does good. Chronic cases are not usually amenable to treatment, but slight improvement is sometimes observed during the use of counter-irritation, hypodermic injections of strychnine, and galvanism to the nerve.

AUDITORY HYPERÆSTHESIA. The term auditory hyperæsthesia is used in a general way to designate increased action of the auditory nerve or centre and various perversions of their function.

TRUE HYPERÆSTHESIA (hyperacusis, oxyecoia), or increased keenness of hearing, is, when well pronounced, a condition of great rareness. In this state, of which there are authentic examples, all sounds or particular sounds (as the high notes of a Galton's whistle) are heard with undue loudness. Usually the state is a transient one of a few hours' duration. It is generally associated with other disorders of hearing. The following are the chief morbid states in which hyperacusis has been observed: in hysteria (associated with increased acuteness of other senses); in the mental excitement induced by alcohol (Politzer); at the onset of acute cerebral and general diseases; during recovery from tubal catarrh; just preceding the development of central disease of the auditory path (Moos).

AUDITORY DYSÆSTHESIA, or dysacusis, is a more common condition in which sounds cause discomfort, although they are not heard with undue loudness. Such discomfort is experienced in a variety of morbid conditions of the middle ear, in labyrinthine disease, in partly deaf persons, and in some who are completely deaf (Jacobson), in trigeminal neuralgia, migraine, neurasthenia, and various organic cerebral diseases (meningitis, general paralysis). The discomfort is probably not produced by direct irritation of the acoustic nerve.

The treatment of these hyperæsthesiæ varies with the nature of the primary affection. The symptom itself is almost always relieved by full doses of the bromides.

TINNITUS AURIUM. The subjective sounds to which the name tinnitus aurium is given constitute a rather common and often a most distressing ailment. The affection is much more frequent in the second than in the first half of life, and is almost unknown in children. It is probably of nearly equal frequency in the two sexes.

Although tinnitus aurium varies much in its character and arises under a variety of conditions, it is always dependent in part upon irritation of some portion of the auditory tract or upon some one of the various sound-producing processes in the neighborhood of the normal ear. Of all cases of tinnitus, labyrinthine disease is by far the most common. There are distinct evidences of nerve-deafness in four-fifths of all the cases that come under the physician's notice (Gowers), and this large proportion of cases would be increased by including the cases of occasional tinnitus in which there is continually very slight nerve-deafness.

The following are the chief direct causes of tinnitus: 1. Various forms of ear disease: (*a*) disease of the external ear, as new growths in the meatus, accumulations of cerumen; (*b*) disease of the middle ear, acute and chronic suppurative otitis, catarrhal otitis, spasm of the stapedius muscle; (*c*) disease of the internal ear, including the various causes of nerve-deafness (*vide ante*). The mechanism by which these causes are operative in producing tinnitus is a matter of conjecture.

2. Irritation of the auditory nerve or of its central path by organic changes, hemorrhage, softening, degenerations, concretions in the nerve, etc., usually associated with nervous deafness.

3. Irritation of the auditory nerve or centre dependent on functional or nutritional disorder in these parts. This is probably the origin of the subjective sensations of sound which are perceived in some cases of epilepsy as an aura, in rare cases of migraine, and in some cases of functional derangement of a neurasthenic character. In the former cases the tinnitus doubtless depends on cortical irritation due to perverted nutrition of the nerve-elements.

4. Intense stimulation of the auditory mechanism through loud or long continued sounds. The report of a cannon, a loud railway whistle, have each been followed by long-continued tinnitus. Sounds less loud, but long continued, are capable at times of causing tinnitus, usually of moderate degree and short duration. Piano-tuners, boiler-makers, etc., are sometimes affected in this way.

5. The movement of the blood in the vicinity of the ear. Normally the blood-currents there and in the ear give rise to no sound, but they may be so changed as to be perceived. The most common form of such disturbance is the murmur in the ears sometimes heard in anæmia, and due probably to vibrations of the blood-current in the carotid. The pulsation may be lessened by pressure on the carotid in the neck. Pressure on the cervical sympathetic has seemed, in some instances, to cause subjective sounds, doubtless by causing dilatation of the labyrinthine vessels. In very rare cases an intra-cranial aneurism causes a murmur similar to that of anæmia.

Any disorder of the general health which leads to defective nutrition of the brain, especially its cortex, constitutes a most important predisposition to the development or intensification of tinnitus. It is further most important to recognize the fact that the continuous overaction of the auditory centres leads to nutritional changes there, which, after a time, may become as potent an influence in the maintenance of tinnitus as the aural changes themselves.

Symptoms. The sounds vary much in character, intensity, duration, and in the situation to which they are referred. Generally they are simple in nature, that is, of a low degree of elaboration, and are spoken of as "singing," "ringing," "hissing," "buzzing," "humming," "whistling," etc.

Much more rarely the sounds are more elaborate in character and are likened to the ringing of bells, the rustling of wind in the trees, or the mingling of voices. Faint sounds are often low pitched, loud sounds are generally high pitched. The sound may be intermittent or constant, pulsatory or continuous. In cases of slight labyrinthine disease, the sounds may recur at long intervals and last only a few minutes. The subjective character of the sounds is generally realized, but sometimes at the commencement of the affection a slight but elaborate sound is thought to have an actual objective existence. The sounds are more often unilateral than bilateral, and generally referred to the ear, but, strangely enough, sometimes to the head, generally, or to a particular part of the head at a distance from the ear. In the same patient considerable variations may occur in the intensity and character of the subjective sounds. Tinnitus is usually influenced to some extent by external sounds, sometimes decreased, sometimes increased. Usually a loud continuous noise (like that of a railway car) reduces the subjective sounds, at the same time, in some cases of labyrinthine disease, diminishing the degree of deafness. The condition of hearing varies much in different cases. Owing to the frequent labyrinthine origin of tinnitus, nerve-deafness on one or both sides is common. Slight continuous deafness may be temporarily increased by transient tinnitus. In some cases there is deafness only while the tinnitus lasts. In some hearing is quite normal at all times, and in a few there is hyperacusis.

Clicking sounds in the ear generally depend on contraction of muscles connected with the Eustachian tube, vibratory sounds on contraction of the stapedius or tensor tympani. Tinnitus is frequently associated with headache, neuralgia, insomnia, nutritive disorders, and neurotic states generally. (See chapter on Neurasthenia). In many cases these conditions are gradually developed as a consequence of the depression in health induced by the distressing and persistent nature of the tinnitus. The depression that results in some obstinate cases leads to suicide. Occasionally an intractable tinnitus is at least the immediate cause of the development of insanity.

Diagnosis. The diagnosis of the origin of tinnitus rests mainly on the associated symptoms. Of these the chief is deafness, and the cause of deafness is usually also the cause of the tinnitus. The various causes of nerve-deafness have been discussed elsewhere.

In trying to distinguish between tinnitus from labyrinthine disease and tinnitus from disease of the nerve, the enormous preponderance of the former should be mentioned. The character of the tinnitus gives little help, as a rule. Roughly, speaking elaborate sounds are of central origin; but simple sounds are also frequently of central origin.

Prognosis. The prognosis depends on the ability to recognize and remove the cause of the symptoms. Where a case depends on external or middle-ear disease or constitutional derangement of a remediable nature, the chances of recovery are very good if the case is recent. But when the trouble depends on labyrinthine or central disease, the outlook is uncertain. There is no way of predicting which cases will prove intractable and which will be benefited by treatment.

Treatment. While there are many cases in which treatment is of no avail there are also many in which intelligently planned treatment gives considerable relief. The first step in treatment is to remove any conditions which may directly or indirectly result in tinnitus: the treatment of local morbid states on the one hand and constitutional states on the other. The treatment of anæmia, gout, or rheumatism, where these exist, may in itself be sufficient to effect improvement. Where the tinnitus comes on only after fatigue or excitement, as is sometimes the case, the indication is plain. Where digestive disorder exists it should be carefully remedied; even when there is no evidence of indigestion the diet should be simple and nourishing. Attention should be given to the state of nutrition in all cases where uric acid is excreted in excess. The action of a blue pill is sometimes followed by considerable relief, lasting perhaps for days. Those who are exposed to continuous noise and suffer in consequence should have rest in a quiet place. But all efforts to combat the causes of tinnitus may fail, in fact do fail in a majority of cases. Treatment must then be addressed to the symptom itself.

The drugs which have been found useful in the treatment of tinnitus act either upon the functions of the labyrinth or upon the cortical centres. Quinine and salicylate of soda both act upon the internal ear, and in physiological doses cause tinnitus deafness and vertiginous sensations. Both drugs exert a considerabe influence in some cases in mitigating the intensity of the sounds, and at times the effect lasts after the drugs have ceased to be administered. Quinine should be given in doses large enough to produce physiological effects—10 to 15 grs. t. i. d. Salicylate of soda is serviceable in a much larger proportion of cases and rarely fails to do some good. The dose should be from 10 to 20 grs. t. i. d. The drug should be given a thorough trial if the patient can take it without gastric disturbance. Neither of these drugs should be employed in the tinnitus of acute ear disease. Of the drugs which reduce that overaction of the central nerve-elements, which in many cases of tinnitus comes to be a most important factor, the bromides are the most useful. There are a few cases of tinnitus that are not in some degree benefited by the administration of bromide of sodium or ammonium in doses of 10 to 20 grs. twice or three times daily. When giddiness accompanies the sounds this also is relieved. The combination of the bromide with belladonna is sometimes advantageous. The bromide treatment may often be continued indefinitely without any reduction in its effect. In very severe cases of tinnitus it may be necessary to use morphine hypodermatically.

Counter-irritation is also of service in many cases. A fly-blister over the mastoid often reduces the noise for days after its use. The painting of the cartilaginous part of the external ear with a liniment containing the tincture of valerian is frequently of service (Politzer). Daily exposure of the patient to a sound of an opposite character to that heard—to a low note, where the sound heard is high, and *vice versa*—sometimes mitigate the trouble (Lucae).

DISEASES OF THE FIFTH NERVE.

PARALYSIS.

Etiology. The fifth nerve may be damaged by disease in any part of its course. The following are the chief causes of its involvement:

1. The three divisions of the nerve, ophthalmic, superior, and inferior maxillary, are exposed to different lesions, owing to the difference in their course. The ophthalmic lies in the cavernous sinus, where it may be damaged by tumors in the pituitary region, and within the orbit may be injured by new growths or inflammatory processes. The superior and inferior maxillary divisions lie in the spheno-maxillary fossa and are liable to suffer from wounds, and the new growths, which frequently invade this region (osteo-sarcomata, parotid tumors, etc.).

2. At the base of the brain (in the posterior or middle fossa) the fifth nerve is liable to suffer from tumors, syphilitic meningitis, or caries of the temporal bone.

3. Disease in the pons, as hemorrhage, acute softening, and new growths, occasionally also a patch of sclerosis, may damage the fifth. When the root-fibres are implicated the paralysis may be complete in both motor and sensory portions of the nerve.

When the sensory nuclei of origin are affected the paralysis is partial, as a rule, because the sensory nucleus is so extensive that it is rarely affected throughout. The motor nucleus is apt to escape degenerative changes, even when these are widespread in the pons and medulla. The descending root is occasionally involved in a degenerative process, which leads to facial hemiatrophy.

4. Certain traumatisms to the mouth and nose (pistol shots, punctured wounds) are liable to damage the branches of the nerve.

5. Primary neuritis of the nerve is rare, probably because its deep course protects it from the influence of cold. Such a neuritis may occur, however, in persons in depressed health, especially in the gouty, rheumatic and syphilitic. The neuritis which causes herpes zoster is not uncommon in the fifth nerve.

Symptoms. The chief symptoms of paralysis of the *sensory* division of the fifth nerve are as follows:

1. Loss of sensibility in the parts supplied by the fifth. The loss may involve the entire region supplied by the fifth when there is disease of the root of the nerve, or when all three branches (ophthalmic, superior maxillary, and inferior maxillary) are implicated in a lesion at the base of the brain. When individual branches are involved the anæsthesia is limited to the regions supplied by them. The tactile sense is commonly lost before the pain sense, but eventually both are involved. Besides loss of sensibility of the skin, sensation is abolished in the mucous membranes supplied by the nerve (nose, tongue, and mouth on the side of the lesion). In some cases the back of the tongue and the hard and soft palate share in the sensory loss, but in others (even when the entire nerve is probably involved) only the anterior two-thirds of the tongue loses its sensibility.

2. Pain, neuralgic in character, in the distribution of the nerve, may precede the development of anæsthesia. There may also be tender points in the course of the nerves, and hyperalgesia.

3. Loss of taste on one side of the tongue and palate results from severe damage to the sensory portion of the fifth. When taste is not involved in lesions of the fifth it is probably because the disease is partial or the lesion is within the pons. Loss of taste confined to the anterior two-thirds of the tongue occurs where the lingual branch of the fifth is diseased between the peripheral distribution and its junction with the chorda tympani.

4. Various trophic and vasomotor disturbances may result from lesions of the fifth nerve. Diminution in lachrymal and salivary secretion is observed in destructive lesions; increase in these secretions may occur where there is irritative disease. Occasionally the face is distinctly paler on the affected side, owing to decreased vascularity. The most important trophic change is that which occurs in the nutrition of the eye and results in inflammation of the eyeball. The cornea becomes dry and opaque, ulceration occurs, and this may go on to perforation and eventual destruction of the globe of the eye. These ocular changes are especially frequent in irritative disease involving the Gasserian ganglion or the nerve anterior to it, and are rare in lesions between the ganglion and the superficial origin of the nerve from the pons. Another trophic change that results especially from lesions that affect the Gasserian ganglion or the nerve anterior to it is herpes zoster, which most often affects the superior maxillary distribution of the nerve. It is thought by some that common catarrhal herpes of the lips depends on neuritis of the peripheral branches of the fifth; but this view is not supported by satisfactory pathological evidence.

Hemiatrophy of the face is certainly due, in many cases at least, to disease of the descending or trophic root of the fifth.

The principal symptoms of disease of the *motor* portion of the nerve are weakness of the masseter, temporal, and external pterygoid muscles on the side of the lesion. The weakness of the masseter and temporal muscles is evident when the finger is placed over these muscles and the patient brings the upper and lower teeth forcibly together. When the loss of power is considerable it is shown by the weakness or complete absence of contraction, when the loss is slight the weak muscle may contract a little later than the normal one. The pterygoid paralysis is shown by defective lateral movement of the jaw, which can be moved toward but not away from the paralyzed size, and also by deviation of the jaw, when depressed, to the paralyzed side. The tensors of the palate and tympanum have been supposed to be paralyzed by a lesion of the fifth, but there is no satisfactory clinical evidence that this is actually so. There may be some sinking in of the temporal and zygomatic fossæ in consequence of the muscular atrophy, and some limitation of the downward motion of the jaw from shortening of the muscles.

Diagnosis. Complete paralysis of the fifth nerve, including paralysis of motor portion of the nerve, can hardly be confounded with any other condition. When, however, the sensory portion is alone affected and there is anæsthesia of adjacent areas, difficulties in diagnosis may arise. In hemianæsthesia the loss of sensibility includes the very parts supplied by the sensory portion of the fifth. But in hemianæsthesia the loss involves also the back of the head, the limbs and the trunk on one side, and often there is hemianopsia. When pain is the only symptom of disease of the fifth, as it may be for a time, it is important to avoid confounding the condition with neuralgia. When anæsthesia appears, corresponding in area to that supplied by the fifth or one of its branches, the nature of the affection is plain. The appearance of loss of taste upon one side is also an important aid in diagnosis.

The diagnosis of the locality of the disease which gives rise to paralysis of the fifth rests entirely on the extent of the anæsthesia and the condition associated with this loss of function. Paralysis of all three divisions of the sensory portion, with paralysis of the motor portion of the nerve, points to disease at the base of the brain, or, perhaps, at the Gasserian ganglion. If there is also paralysis of the sixth nerve the lesion is surely near the point of emergence of the fifth at the side of the pons. If the loss of function is confined to the distribution of the superior maxillary division of the nerve, the lesion is probably at the sphenoidal fissure or in the orbit, and this belief is strengthened if there is also paralysis of the nerves to the eyeball. If the inferior maxillary division of the nerve is alone affected the lesion is probably in the spheno-maxillary fissure or in the superior maxillary bone. Paralysis of the second and third division of the nerve, without involvement of the first division, is generally due to disease in or near the spheno-maxillary fossa.

Crossed paralysis of the fifth nerve (fifth on one side, anæsthesia of arm and leg on the other) always depends on a lesion in the lower half of the pons or upper part of the medulla, if the onset is sudden. The association of conjugate deviation (to the side of the lesion) with paralysis of the fifth is positive evidence of a pontine lesion.

Treatment. Success in the treatment of paralysis of the fifth nerve depends largely on the ability to recognize and remove its cause. In many cases the nature of the lesion is such (new growth, traumatism) that little can be done. The treatment of a syphilitic process involving the fifth is that of a similar process elsewhere in the nervous system. Where there is simple neuritis vigorous counter-irritation at the occiput or over the mastoid may do good, at least by relieving pain. If the neuritis follows exposure to cold the application of a large linseed poultice over the head and face of the affected side may be of service. Care must be taken to avoid setting up trophic changes in the skin.

Pain may be very severe, especially at the onset. If it is not relieved by local applications of heat and the use of phenacetine or antifebrine, morphine should be injected hypodermatically. The galvanic current is of very little service in the relief of such pain. If there is local anæsthesia this may be much benefited (where the anæsthesia does not depend on destruction of nerve-fibres with a break of continuity) by frequent stimulation of the insensitive area with the faradic current through a wire brush. The current should be strong enough to be felt, if this is possible. Frequent gentle rubbing of the anæsthetic area helps to maintain the nutrition of the skin. If there is any evidence of altered nutrition of the eyeball the greatest care must be taken to exclude all causes of irritation from the eye. The eye should be kept constantly covered. The conjunctiva should be washed from time to time with a saturated solution of boric acid. But the irritation of the diseased nerve may be so great from the beginning that no measures can prevent the destruction of the eyeball, and perhaps the involvement of the other eye.

NEURALGIA OF THE FIFTH NERVE.

Trigeminal or Trifacial Neuralgia; Tic Douloureux; Prosopalgia.

The fifth nerve is by far the most important nerve of common sensibility, and, owing probably to the highly differentiated structure of its central connections, is particularly prone to suffer from disturbances of nutrition. Neuralgia of the fifth nerve is considerably more frequent than all other forms of neuralgia taken together. It is not, therefore, surprising that the etiological characteristics of this form of neuralgia and most of the facts relating to prognosis and treatment should apply to neuralgia in general.

Etiology. Neuralgia of the fifth is essentially a disease of adult life. It is rare in childhood and old age. Some of the most distressing and intractable cases, however, begin after sixty. Women are certainly somewhat more liable to the disease than men, but the difference in sexual incidence is not so striking a feature as some authors have stated. In a very small proportion of cases a direct neuralgic heredity can be traced. Much more frequently there is general neuropathic heredity as shown by a history of migraine, of epilepsy or insanity.

Persons of so-called "nervous" temperament, who are excitable, irritable, and excessively emotional are very liable to be sufferers from neuralgia of the fifth. The disease is very much more common in persons of feeble constitution than in those who are robust. It is true that the condition is met with among persons in excellent general health, but it is also true that in most severe cases where the health is apparently good there is in reality some disturbance of nutrition. Persons who have what are known as rheumatic tendencies are very prone to trigeminal neuralgia. It may probably be said that in almost all cases of an intractable nature the excretion of uric acid is habitually much increased both absolutely and in comparison with the amount of urea.

All influences that impair the general health may operate as exciting causes of neuralgia of the fifth. Overfatigue, bodily and mental, prolonged emotional excitement, excessive venery, overlactation, etc., are all factors of the highest moment in determining the development of neuralgia of the fifth. Excessive use of the eyes without errors of refraction is competent to bring on a seizure in some persons. Errors of refraction and pronounced insufficiency of the extrinsic ocular muscles (especially esophoria) are occasionally important factors in bringing about a trifacial neuralgia, but it must not be forgotten that these conditions very often depend on reduction of the general health.

Exposure to cold, local or general, may excite a neuralgic seizure. In some persons a rapid lowering of the temperature of the air is apparently sufficient to determine a seizure, although there has been no exposure. Peripheral irritation, of whatever origin, is an important cause of neuralgia of the face.

The important influence of carious teeth should always be borne in mind. It is an important fact that the pain may not be greatest in the nerve-root irritated, but in a neighboring nerve-distribution. Traumatism of a branch of the fifth, with or without resulting neuritis, occasionally operates as a cause of neuralgia of the fifth. The various toxæmic influences, as alcoholism, lead-poisoning, diabetes, malaria, and the grippe, are potent and not infrequent causes of the disease. Malarial neuralgias of the face are not so common as some writers would have us believe. The neuralgias which follow the grippe are occasionally very severe. There is little evidence to favor the view that active syphilis is ever a cause of true neuralgia. Old standing syphilis may of course operate through its influence in depressing the general health. Acute gastro-intestinal derangement has been rapidly succeeded in some instances by neuralgia of the fifth. The nature of the relation is obscure. A toxæmic state probably underlies the symptoms.

Symptoms. The pain of trigeminal neuralgia has no distinctive characters; it is spontaneous, paroxysmal, and felt in certain regions, usually limited in extent of nerve-distribution, thus resembling the pain of neuralgia generally. Further, it is usually unilateral, is usually constant for a time in its position, and is usually referred, not to the skin, but to more deeply lying structures, the pain often corresponding to the position of the nerve-trunk or branch. Trigeminal neuralgia is about equally common on the two sides, and much more commonly affects one or two divisions of the nerve than all three. Neuralgia of any one of the three divisions of the nerve is apt to lead to the establishments of tender spots. During the paroxysms the pain is commonly most intense at certain points, and pressure at these points is especially painful. After a time these spots remain tender in the interval between the paroxysms.

Neuralgia of the *first* or *ophthalmic division* of the fifth involves mainly the supra-orbital branch, and is usually referred to as supra-orbital neuralgia. It is also known as "brow ague," owing to the fact that it is, perhaps more frequently than any other form of neuralgia, due to malarial infection. The pain is felt radiating over the front of the head from the supra-orbital notch. It may be felt also in the eyelid, in the eyeball itself, and at the side of the nose, high up on the same side. A tender point often exists at the supra-orbital notch or a little above it in the course of the nerve. Occasionally the following painful points occur: a palpebral in the upper eyelid, a nasal at the exit of the long nasal branch at the junction of the nasal bone with the cartilage, and, according to some authors, an indefinite focus within the globe of the eye. Pain above the eyebrows is not very uncommon as the result of morbid processes in the frontal sinuses. Such pain may be neuralgic in character, and depends in some cases on certain influences which act at a distance, as, for instance, in the cases where neuralgic sinus pains are caused by eating ices. Sometimes there is neuralgic pain referred to the soft parts just below the supra-orbital nerve-trunk.

Ocular neuralgia, neuralgic pain referred to the eyeball itself, is a particularly distressing and not very infrequent form of neuralgia. The pain is often severe, may be spontaneous or excited by use of the eyes, and may be accompanied with slight or considerable dimness of vision and lachrymation. The pain is usually one-sided, but is sometimes bilateral. It may or may not be associated with other neuralgic pain in the region of the fifth. Sometimes it seems to depend upon an error of refraction, but often none can be discovered. The pain is sometimes described as a tearing pain deep in the orbit. The general health is probably always impaired in severe ocular neuralgia.

Not rarely the subjects are anæmic young women. The past subjects of rheumatic iritis are prone to this form of neuralgia.

In neuralgia of the *second* (superior maxillary) *division* of the fifth, the pain is referred to the area lying between the orbit and the mouth and often to the side of the nose. In infra-orbital neuralgia the chief painful spot is at the emergence of the nerve from the infra-orbital foramen. Not rarely there are the following painful foci; a molar over the prominent part of the bone, a gingival line along the gums of the upper jaw, and a nasal point at the side of the nose. It is said that a palatine point is occasionally the seat of intolerable pain (Anstie). When the *inferior maxillary division* is affected the following are the chief foci of pain: a temporal at a point on the auriculo-temporal branch just in front of the ear (a common focus) or in the posterior part of the temple, an inferior dental opposite the emergence of the nerve from the foramen, a parietal just above the parietal eminence at the point of conjugation with the branches of the great occipital nerve (a very small point and the most frequent of all), and occasionally a lingual point at the side of the tongue. In many cases of neuralgia of the third division, the pain is not confined to a small area, but extends throughout the various branches to the temple, parietal eminence, ear, lower jaw, and tongue. Sometimes the pain is confined to the tongue, and is then very intense. In the temporal region the pain is apt to be boring in character.

While it is comparatively rare for neuralgia of the fifth to occupy all three divisions of the nerve, it is by no means rare for pain to extend, from time to time, from the division chiefly affected to the branches of a neighboring division or even to the branches of other nerves. Thus in superior maxillary neuralgias the pain may extend to the branches of the ophthalmic, to the distribution of the great occipital over the occiput, to the lingual branch of the inferior maxillary, or even to the cervical nerves. The pain of trigeminal neuralgia is apt to be excited by movement and by contact. Mastication is often difficult or impossible in neuralgias of the inferior maxillary nerve. Occasionally there is reflex muscular facial spasm ("tic convulsif") when the neuralgic pain is acute and intense. Slight paralysis of the third nerve has sometimes followed paroxysms of the supra-orbital neuralgia. Supra-orbital neuralgias are sometimes associated with marked vasomotor disturbances. When trophic disturbances are pronounced and of long duration, it is probable that some portion of the nerve has undergone structural change. Migratory pains are often felt in various parts of the scalp, and may be associated with tenderness of the skin during and after the paroxysms. These pains may bear no distinct relation to the nerve-trunks, and cannot, perhaps, be strictly designated neuralgias. In some cases such pains alternate with true neuralgia.

The neuralgias of the fifth may be advantageously classified according to their characteristics and causes. The term *epileptiform* is applied to an agonizing form of neuralgia in which attacks are characterized by the great suddenness and severity of their onset, their short duration, and frequent occurrence. This form of the disease belongs especially to the second half of life, and may develop after sixty. The maxillary divisions are more frequently affected than the ophthalmic division of the nerve, and more than one branch is usually affected. Facial spasm may accompany the pain. A neuralgia may, perhaps, be termed *reflex* when the pain is felt in a nerve distribution different from that in which its cause lies. Thus a carious tooth may cause neuralgia in a distant part of the fifth, and an injury of the ulnar may be the cause of a trigeminal neuralgia. Some neuralgic pains in the ophthalmic region of the fifth are accompanied with a herpetic eruption over the forehead, and such cases have been designated *herpetic* neuralgia, but it is probable that an actual neuritis exists in such instances. The fifth nerve is often the seat of what are known as *degenerative* neuralgias—intractable neuralgias—which come on late in life and are associated with evidences of degeneration of the central nervous system. Epileptiform neuralgia sometimes belongs to this category. The causative relations of rheumatism, gout, diabetes, anæmia, malaria, and syphilis to neuralgia have led writers to speak of a rheumatic neuralgia, a gouty neuralgia, etc.

Diagnosis. The recognition of the nature of neuralgic affections of the fifth is usually easy; the relation of the pain to a nerve trunk or branch or area, its unilateral distribution, distinctly paroxysmal and migratory character, and the absence of all evidence of organic disease of the nerve involved (absence of tenderness and swelling along the course of the nerve affected), are unmistakable features. Sometimes it is difficult to say whether a pain referred to the skull should be called headache or neuralgia. A head-pain is to be considered neuralgic only when there is a distinct correspondence on the part of the pain to the course of the branch or branches of a nerve or to an entire nerve-area. Actual neuralgia may, however, cause a diffuse head-pain in its neighborhood, which is sometimes called a "neuralgic headache."

The trigeminal pains that occur in the course of organic brain disease are to be recognized by their associated symptoms.

Prognosis. The outlook for cases of trigeminal neuralgia is influenced by the same indications that determine the prognosis of neuralgia generally. The prognosis is best in cases where there is a conspicuous and remediable constitutional cause, as anæmia. Other things being equal, it is poor where after careful examination no cause whatever can be found. Cases in which there is a pronounced neurotic heredity yield much less readily than where there is no hereditary taint. Most cases are intractable in proportion to their duration and severity. The fact that the pain has during many years been shifting from place to place is an unfavorable feature. The chances of relief by treatment is much greater in youth and middle life than after sixty. Epileptiform neuralgia is the most intractable of all varieties. It is probably incurable by means of drugs. In all varieties of neuralgia the liability to recurrence is very great if the affection has been well established for many months. Even the most agonizing forms of neuralgia appear not to shorten life apparently, though they may render it difficult to endure.

Treatment. In any case of trigeminal neuralgia it is necessary to direct treatment, first, to the pain itself; secondly, to the removal of the cause of the neuralgia. The measures taken to remove the cause of the affection are of the first importance, but the relief of pain is naturally apt to be the first step in treatment. Where the condition is not "idiopathic," but neuritic in character, the treatment is that recommended in connection with neuritis.

Of the measures which are employed for the relief of pain it is convenient to consider, first, the use of drugs which operate by entering the general circulation, and, second, the use of purely local measures. A large number of drugs have been employed for their sedative or anodyne action, but reliance can be placed on few. The bromides (especially the bromide of potassium) have been extensively used for their sedative action. They have surprisingly little influence upon the pain and are apt to increase the digestive disturbance which often exists. In irritable, nervous patients the drug may be useful in moderate doses (5 to 10 grs. t. i. [illegible] dering more endurable the pain of paroxysmal neuralgia. Whe [illegible] severe the bromides cannot be counted upon. Salicylate [illegible] of use in "rheumatic" neuralgias. Its control over [illegible] ted, and it not merely does not remove the "rhe [illegible] ; may aggravate it. The antipyretics, antipyrine, antifebrine, phenacetin, and exalgin, have been extensively and somewhat recklessly employed in neuralgias of the fifth. In moderate doses each of these drugs (especially antifebrine) has a remarkable influence over the pain, even when this is severe. The promptness with which relief may be obtained and the absence, in most instances, of distinctly and immediately objectionable after-affects has popularized the use of these agents, not merely with practitioners, but with the laity. But the fact should not be lost sight of that these drugs employed in even moderate doses, continuously for weeks or months, are capable of modifying nutrition profoundly, probably by increasing hæmolysis or checking hæmogenesis. The writer has met with several instances in which a considerable grade of anæmia and complex disorders of nutrition were referable to such use of these antipyretics. The fact that these drugs may be readily abused for the relief of pain does not of course condemn them, but should render the practitioner exceedingly cautious to keep the dose as small as possible and to intermit frequently in the course of their administration. All of these drugs are liable, after prolonged use, to grow less efficacious in the relief of pain.

Nervine stimulants (alcohol, valerian, sulphuric ether) sometimes abort a neuralgic attack, but are of little use after the pain has become established. The repeated use of alcohol is of course to be deprecated. The use of small doses of nitroglycerin ($\frac{1}{100}$ gr. t. i. d.), alone or combined with tonic treatment, may be very serviceable for the relief of neuralgia. It probably acts, in a measure at least, by improving the nutrition of the nerve-elements concerned in initiating painful stimuli, by flushing them with blood.

Opium and morphine are the drugs which give the quickest and most certain relief to the pain of neuralgia. The hypodermatic injection of a moderate dose ($\frac{1}{8}$–$\frac{1}{4}$ gr.) of sulphate of morphine gives relief to the severest forms of neuralgia, especially if the injection be made into or near the seat of the pain. But the danger of forming the morphine habit is so great that the drug should be used with the greatest caution and only in cases where pain is so severe as to be unendurable, as, for example, in cases of epileptiform neuralgia. Even here all other resources, including those which surgery affords, should be employed before making repeated use of morphine. The injection of cocaine ($\frac{1}{10}$–$\frac{1}{8}$ gr.) into the seat of pain usually brings rapid relief from neuralgic pains, but the danger of establishing a habit is hardly less great than in the case of morphine. In no case should the patient be permitted to make the injection. At times the use of morphine and cocaine does more than palliate; the formation of a "pain habit" may be avoided, for the repeated relief of pain helps to prevent its recurrence.

The use of belladonna or of atropia beneath the skin is effective in some cases of trigeminal neuralgia, but the dryness of the throat and constitutional symptoms that are produced render these drugs objectionable for general use. Chloral has little influence over neuralgic pains. Croton-chloral, in doses of five grains or more, is said to be serviceable in some neuralgias of the fifth, but it is doubtful if much reliance can be placed upon it. Indian hemp likewise has been recommended highly, especially in neuralgias in which the pains are sudden, sharp, and of short duration, but only of moderate severity. The dose is from a quarter of a grain to a grain three times daily. The readiness with which the drug brings on hallucinations in some persons and the frequency with which it deranges digestion are very objectionable features.

All things considered, aconitia is probably the most satisfactory drug at our command for the relief of trigeminal neuralgias. In a large majority of cases, including even some of the severest type, progressively increasing doses of aconitia greatly modify or stop the pain. The beginning dose is $\frac{1}{200}$ gr. In slight cases this amount, or double this amount, often gives distinct relief. When the pain is severe the administration of the drug must be pushed to the point of producing distinct physiological effects: numbness of the tongue, slow heart-action, and perhaps nausea. The dose should be increased from day to day by $\frac{1}{200}$ gr. until these effects are produced. The relief afforded is, in many cases, more than temporary. There is no evidence to show that the long-continued use of aconitia impairs the general health. In a small proportion of cases it is without effect.

In cases of neuralgia of the fifth that are of malarial origin and in many that are not, Warburg's tincture is exceedingly efficacious. Its administration should be preceded by a mercurial purgative. The fluid extract of ergot in doses of one drachm (twice repeated, if necessary) has been highly recommended in cases of severe periodic (usually daily recurrent) neuralgias of the fifth. The drug may also be given by the rectum (ʒij, water ℥ij), if its use by mouth causes nausea or vomiting. Ergot is said to succeed frefrequently where aconitia, quinine, and the antipyretics have failed.

The irritant or sedative effects of local treatment are often of service in trigeminal neuralgias of moderate severity. Blisters and sinapisms cannot usually be conveniently employed about the head, but the actual cautery may be of great service, especially in supra-orbital neuralgia. The application should be superficial and should be made with the instrument heated to a dull red. Acupuncture and aquapuncture are not suitable for use about the head. The hypodermatic injection of osmic acid cannot be recommended, although it is sometimes very efficacious. Camphor chloral and chloroform liniment sometimes do good. Menthol is useful only when the pain is slight. The ointments of belladonna, veratrine, and aconitia may give temporary relief. The oleate of morphine has little efficacy. Local heat and moisture frequently give a great deal of relief from pain and a sense of comfort. A warm saline nasal douche may relieve frontal pain that is associated with neuralgia. The ether spray is not, as a rule, satisfactory. A strong ointment of cocaine made up with lanoline may be very useful, but should not be used near mucous membranes. Occasionally electricity (faradism or galvanism) gives a temporary relief to a trigeminal neuralgia, but it cannot be regarded as an effective and reliable therapeutic measure. If it seems desirable to try electricity, the best effect will usually be obtained by placing the anode over the seat of pain and the cathode at some indifferent point. The current should be weak, and should never cause pain, but merely a slight sensation of tingling or burning. From two to five milliampères will usually suffice.

The use of electricity for the purpose of diffusing a medicinal substance at the seat of pain is far more efficacious. Special electrodes are made for the purpose of effecting this diffusion, but any metal electrode will answer. The cataphoric action is effected by the positive pole (anodal diffusion). Chloroform, the tincture of aconite, or a strong solution of cocaine may be employed. Cocaine is most satisfactory. A piece of tissue-paper is saturated with a 20 per cent. solution of cocaine, the paper is placed over the metal electrode and the latter is applied to the painful spot (Peterson). This method is economical and secures a degree of accuracy in dosage. A weak current is used for five or ten minutes. Relief is usually pr
last for many hours. Although the direct relief of pain fir
tention, nothing is more important for the successful treatme
neuralgia than those measures which operate through their
general health by the removal of a local cause of irritatio
of any distinct cause of irritation, such as carious teet

should not be delayed. The effect upon the pain is often most striking, especially if the local condition has not been long operative, but it is important to realize that the cases are not rare in which the removal of a local condition which seems sufficient to explain the existence of a neuralgia does not in itself materially benefit the patient.

In every case of trigeminal neuralgia the character of the patient's environment should be carefully studied, and every effort should be made to render his surroundings as hygienic as possible. All influences that tend to depress the general health—sedentary habits, sexual excess, excessive use of alcohol, tobacco, tea, and coffee, emotional excitement, worry, and over-fatigue—should be removed so far as possible. An out-of-door life should be advised where this is practicable; in any case the patient should be out of doors at least two hours daily. In some severe cases of neuralgia a removal of residence to a warm, equable climate (Southern California, Florida) may be necessary. When possible the patient should exercise; but, if the exercise of walking or horseback riding causes exhaustion, massage is to be preferred. It is very important that exercise should never be carried beyond the degree that causes an agreeable sense of fatigue. Unless there are exceptional reasons (diabetes, excessive intestinal putrefaction) for restricting the diet, this should be full and representative of the various types of food-stuffs. Nitrogenous food should be taken, chiefly as meat, and where the pain is very severe beef is to be distinctly preferred. Contrary to general opinion, gouty and rheumatic neuralgias are no exception to this rule. Cod-liver oil, iron, arsenic, and strychnine are valuable aids in treatment. Quinine is much less valuable than strychnine.[1] Warburg's tincture is an excellent tonic in many cases. Zinc phosphate and phosphorus have been highly recommended, but have little effect. Mixed treatment must be employed where there is recent syphilis.

It occasionally happens, especially in persons in the second half of life, that all attempts to relieve a trigeminal neuralgia are without avail. In such cases the only measure which offers a prospect of success is the interruption of the passage of all impulses along the nerve, from its peripheral distribution to the cortical centres. This may be accomplished by means of nerve section, by neurectomy, by evulsion, or by the removal of the Gasserian ganglion, and perhaps by nerve-stretching. Nerve-stretching has been of temporary benefit in some forms of neuralgia, but is unsuitable for trigeminal neuralgias. Nerve-section is apt not to give permanent results, in part because union is likely to take place between the divided ends, which remain in contiguity. The most satisfactory operation upon nerves is that of neurectomy, in which a short piece of the trunk of the nerve is removed, although in some situations, as in the case of the inferior dental nerve, it is better to practice evulsion and remove as much as possible of the peripheral end of the nerve. Cases are on record in which long relief has followed neurectomy, but in a considerable proportion of cases, probably a majority, the pain has recurred in from six to eighteen months. In many cases the relief from operation is slight and transitory, the pain soon returning with unabated intensity and rendering the life of the patient unendurable. In such cases the question of removing the Gasserian ganglion arises. Several cases are now upon record

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[1] A tablet triturate (Fraser's) having the following composition is often serviceable as a tonic and as a means of relieving pain in cases of moderate severity :

Quin. sulph.	1/2 gr.
Morph. sulph.	1/30 gr.
Strych. sulph.	1/120 gr.
Acid. arsen.	1/60 gr.
Ext. aconite	1/8 gr.

This tablet is not suitable for continued use, on account of the morphine it contains.

in which this has been successfully done, and in which there was immediate and entire relief from pain. As regards the length of time for which relief from pain may be expected, the observations are at present far too few to enable us to arrive at a conclusion. The operation as devised by Hartley, of New York, is not likely to prove one from which the mortality is great, and, while there is still considerable uncertainty as to the duration of the relief afforded, it may be recommended without hesitation in cases where all other means have failed and the morphine habit is the only alternative. The anæsthesia of the face is of slighter extent than might be expected after removal of the Gasserian ganglion. The sense of taste is apt to be lost in the anterior two-thirds of the tongue upon the corresponding side. Trophic disturbances of the skin and eyeball were absent in two cases reported by Thomas, and probably do not usually occur. The masseters are apt to be paralyzed, and some branches of the facial nerve are liable to be cut in the course of the operation. (See also Chapter XXXIII.)

It seems probable that the chances of success from neurectomy are much increased by doing the operation as early as seems justifiable.

Cutting operations upon the extrinsic muscles of the eyeball have of late come into vogue in certain quarters for the relief of trigeminal neuralgias. The practice is based on the belief that many neuralgias of the fifth are occasioned by insufficiency of the ocular muscles, and that the correction of this defect is essential to recovery. There is some evidence that the correction of a high grade of insufficiency has benefited a small number of cases where the neuralgia has been distinctly related to the use of the eyes. These defects can usually be overcome by the use of prisms. The operation of cutting has been extensively performed without the slightest justification, and often with distinct injury to the patient. If the operation has any legitimate use, it is certainly in an exceedingly limited class of cases. Errors of refraction should, of course, receive careful attention in all cases of neuralgia of the first division of the fifth, although it is only rarely that a causative influence can be ascribed to this condition.

FACIAL PARALYSIS.

The muscles of the face are paralyzed by a lesion anywhere in the facial path which lies between the motor centre for the face in the lowest third of the Rolandic area and the face muscles of the opposite side. But the nature of the paralysis differs according to the position of the lesion—according as the lesion affects the facial path between the nucleus of the facial in the pons and the cortex (supra-nuclear facial paralysis), or involves the nucleus of the facial or the fibres of the nerve itself (nuclear or infra-nuclear paralysis). In the supra-nuclear form the upper muscles of the face (orbicularis palpebrarum and frontalis) retain their power, voluntary movements may be more impaired than emotional movements, and the electrical reactions remain little or not at all changed. In the nuclear or infra-nuclear form all the muscles of one side of the face (including the orbicularis and frontalis) are paralyzed, emotional movements are lost equally with voluntary ones, and the electrical reactions are altered in character.

Supra-nuclear paralysis is often referred to as "central," in distinction from paralysis due to nerve lesions, which is termed "peripheral." Paralysis from disease of the nucleus or the nerve-roots in the pons is sometimes called "central," sometimes "peripheral." The character of such palsies are peripheral, although the lesions which cause them are central. Only the peripheral palsies (using the term in the broad sense) are considered here;

supra-nuclear paralysis is treated with the hemiplegia, with which it is commonly associated.

Etiology and Pathology. Peripheral facial paralysis, though not so frequent in its occurrence as some other form of peripheral palsy (*e. g.*, musculo-spinal paralysis), is yet one of the most common of peripheral paralyses. Probably two-thirds of the cases, including cases of every origin, occur between the twentieth and fortieth years, and the condition is more often seen in men than in women, and has been, owing to their greater exposure, met with in children. The following are the chief causes of peripheral facial paralysis:

1. Neuritis of the facial nerve is the cause of a very large proportion (probably more than 80 per cent.) of all cases of peripheral palsy. It is the cause of Bell's palsy, using that term in its original sense. The neuritis is commonly termed "rheumatic." Whatever that may mean, it is true that the palsy generally comes on after exposure to cold. The exposure may be considerable in degree and special in kind, as sitting by an open window in a passenger car, standing in an open doorway, or driving in the cold (one side of the face being usually more exposed than the other), or it may be more or less habitual, and consists of frequent exposure to draughts. Occasionally the subjects are distinctly rheumatic or gouty, but usually there is no evidence of either condition. It is extremely probable that the general nutrition of the patient is somewhat impaired as a rule, at least temporarily. There are, however, cases in which there is no reason even to suspect impairment of general nutrition; the palsy may occur in the midst of apparently the best of health. Probably the exposure in these cases is unusually severe.

It was formally thought that exposure to cold causes facial paralysis by paralyzing the terminations of the facial nerve in the muscles. There is now good reason to believe that the affection always depends on an inflammation of the trunk of the nerve (perhaps involving chiefly the sheath) within the Fallopian canal. In some cases the nerve-fibres undergo complete degeneration. There is no evidence that the inflammation ever involves the nerve after its emergence from the canal.

2. Injury is an important cause of facial paralysis. The nerve may be torn or compressed in fracture of the base. The disability is then immediate. In other cases of fracture of the base the paralysis has come on in the course of several days, in one case at the end of five days. Here the involvement of the nerve is due to secondary meningitis, with implication of the nerve, or perhaps to secondary inflammation of the nerve alone. Some rare cases of congenital facial paralysis have probably been due to meningeal hemorrhage. The facial is apt to suffer outside the skull, in some of its branches, in operations about the ramus of the jaw. A blow upon the nerve in the parotid region has caused permanent paralysis of the face. Rarely the nerve of one side has been compressed by the blade of a forceps during delivery, and very rarely such injury has been bilateral. Parotid tumors and tubercular nodes in the neck occasionally give rise to facial paralysis.

3. In children the nerve is apt to suffer in the temporal bone. Otitis media, with or without bone disease, is the common cause of involvement here. The nerve may be damaged in consequence of bone disease limited to the mastoid, probably owing to extension of inflammation along the chorda tympani or stapedius branch of the nerve. In rare instances facial paralysis has followed sore-throat.

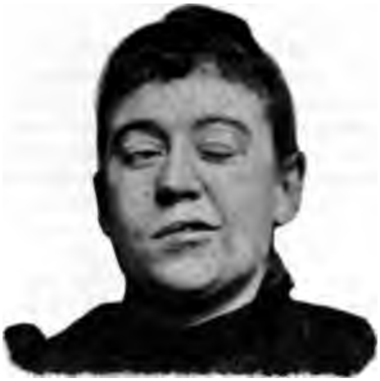
4. The facial nerve is not rarely compressed at the base of the brain in tumors or in meningitis. Very rarely it is implicated in meningeal hemorrhage. Owing to its proximity the auditory nerve is apt to suffer with the facial in this situation.

5. Within the pons the facial (nucleus and root-fibres) is apt to suffer from gross lesions, especially acute softening and hemorrhage, and occasionally from a patch of sclerosis. The nerve may suffer alone, but commonly there is implication of the motor path to the limbs of the opposite side, causing a crossed hemiplegia. The sixth nerve is liable to suffer with the facial in pons lesions. The nucleus of the facial is very rarely damaged in acute myelitis and in chronic glosso-labio-laryngeal paralysis. The facial may suffer in diphtheritic paralysis, possibly from involvement of both cells and fibres. When the facial suffers in alcoholic paralysis (it very rarely does), the lesion is probably in the trunk of the nerve. Among the rarer causes of facial paralysis may also be mentioned the following: Syphilis, which may operate by involving the root-fibres in a meningitis or a gumma, or may cause a neuritis without a considerable inflammatory exudate. Syphilis acts also by reducing the general health and thus predisposing to ordinary neuritis of the facial. In the rare cases of facial paralysis, in which the onset is sudden and there is no evidence of central disease, there is probably hemorrhage into the nerve-sheath or Fallopian canal or thrombosis in a vessel of the nerve. In a few cases emotion or fright has been followed by paralysis of sudden onset, and it is possible that the mechanism here is thrombosis.

Double facial paralysis is of rare occurrence. It is observed as a consequence of (1) bilateral disease of the pons (as from symmetrical softening from basilar disease); (2) disease of nerves at the base of the brain, other nerves suffering; also (3) double otitis media (this is perhaps the least infrequent cause); (4) neuritis from cold; (5) alcoholic multiple neuritis and multiple neuritis from toxic blood-states; (6) nuclear degeneration or symmetrical cortical disease (here the palsy is partial); (7) erysipelas of the face.

Symptoms. In cases of complete facial paralysis there is loss of power and tone in the muscles of the side of the face involved. There is loss of emotional as well as voluntary movements. Owing to the elasticity of the skin in youth the resulting change in the facial expression is far less in the young than in those in whom the skin is wrinkled. In the latter the wrinkles are smoothed out or change their position. The transverse wrinkles of the forehead cease abruptly near the median line, the lower eyelid falls forward,

FIG. 275.



Complete facial palsy. Patient unable to close the eye of the affected side.

and the tears collect so that the eye "waters." The loss of power in the face renders the patient unable to close the affected eye. (See Fig. 275.) It remains somewhat open even during sleep. The inability to close the mouth permits liquids to run out of the mouth unless the head is inclined to one side. Whistling and accurate spitting are impossible. Food is apt to accumulate between the teeth and the lip on the affected side because the buccinator is relaxed. Speech is apt to be a little muffled. The paralysis of the external muscles of the ear, of the stylohyoid and posterior belly of the digastric, and of the dilator naris, does not cause important symptoms.

It was formerly believed that the palate is sometimes paralyzed upon the same side as the face in lesions of the facial nerve, but it is doubtful if this is so. The levator palati is innervated by the spinal accessory nerve, and it is probable that in the very rare cases in which the palate has actually been paralyzed coincidently with paralysis of the facial the palsy has depended upon a lesion of the palatine branch of the spinal accessory or upon disease of the trunk of this nerve. Mere deviation of the uvula must not be mistaken for paralysis of the palate.

In a small proportion of all cases of facial paralysis taste is lost in the anterior part of the tongne or the side affected. In these cases the facial nerve is involved between the origin of the chorda tympani and the geniculate ganglion of the facial. In lesions affecting other parts of the facial the sense of taste is not lost. Taste is involved in a large proportion of all cases of facial paralysis arising from cold. Sometimes it is lost where the facial has been damaged externally to the stylo-mastoid foramen, but in such cases there is doubtless a neuritis extending along the nerve to the chorda. The sense of hearing may be impaired or lost. This occurs especially in the facial paralysis of middle-ear or mastoid disease and in lesions at the base of the brain, which implicate both auditory and facial. Slight loss of hearing is not rare in facial neuritis from cold. Tinnitus aurium also may be present in these cases. When the nerve to the stapedius is implicated the sensitiveness of the ear to certain musical tones may be increased.

It is stated that in lesions involving the facial nerve at the geniculate ganglion there is loss of lachrymal secretion and of the reflex flow of tears upon the side of the lesion.

The changes in the electrical reactions in facial paralysis are identical with those observed in the paralyses of other peripheral nerves. They merit careful study in every case, owing to their bearing upon prognosis. In severe cases the reaction of the nerve to the faradic and galvanic currents is rapidly decreased and may be entirely lost in from one to two weeks. While the muscles lose their faradic irritability, they react more readily than normal to galvanism at first, and show a reversal of the ordinary formula of contraction (the anodal closure contraction (AnClC) being greater than the cathodal closure contraction (CaClC)).

In less severe cases the irritability of the nerve to both currents is reduced but not lost, the loss of faradic irritability in the muscles does not occur so rapidly, and instead of the formula being reversed to galvanism there may be merely an equality in the two contractions. In very slight cases there may be little or no reduction in the irritability of the nerve, or there may be slight initial increase in irritability which is not succeeded by a reduction of irritability below the normal. But even in cases of slight severity the muscles show some reduction of faradic irritability for a time. As the nerve recovers there is a gradual return of the normal irritability of nerve and muscle. There may be considerable recovery of power before the full irritability of the nerve is established.

Atrophy of the muscles of the face always follows degeneration of the nerve, but in most persons there is enough subcutaneous fat to prevent the wasting from being noticeable. In all cases of severe type contracture comes on in the affected muscles when voluntary power begins to return. The contracture comes on in the course of five or six months, increases for six or eight months, and then remains unchanged or lessens slowly. It is most marked in the zygomatici, which may cause the naso-labial fold to be more pronounced than in the normal side while the face is at rest, and thus lead to the impression that the paralysis is upon the side opposite the contracture. With this contracture is associated overaction of the muscles, especially of the zygomatics and the orbicularis, which may contract the face during voluntary movement. The zygomatics may be the seat also of slight spasmodic involuntary twitching.

Pain in the region of the ear is very apt to attend the onset of neuritic cases of facial paralysis. It may be associated with some tenderness to pressure below the ear about the ramus of the jaw. When the paralysis is due to a new growth from the parotid there may be constant intense pain back of the ear. Other symptoms which occasionally attend the onset are swelling about the ear (due to cellulitis), giddiness, and neuralgia of the head, face, or neck.

The onset of the paralysis is usually acute, but very rarely sudden. In some cases the paralysis does not reach its height for forty-eight hours. In slight cases the palsy may entirely clear up in a week or ten days. Most cases last from two to four months, and then recover entirely. Where the loss of power lasts more than six months ultimate recovery is partial only; indeed, it may be said that some permanent loss remains whenever complete paralysis undergoes no improvement after a month's duration.

So far as the facial paralysis itself is concerned its characters are essentially the same whatever may be the position or nature of the lesion. But in cases of facial paralysis due to disease of the facial nucleus the orbicularis oris escapes entirely. This is because the fibres to this muscle arise not from the facial nucleus, but from cells near the hypoglossal nucleus, although the fibres soon join those of the facial nerve.

Diagnosis. The first question in diagnosis that arises upon seeing any case of paralysis of the face relates to the position of the lesion: Is the paralysis of central (supra-nuclear) origin or is it of nuclear or infra-nuclear origin? Cases of cerebral origin are ruled out by means of the following facts: (1) cerebral cases do not show persistent paralysis of the eyelid; (2) emotional movements are little or not at all impaired; (3) reflex action is unchanged; (4) the electrical reactions are normal or slightly changed; and (5) the difference in the course and associations of cerebral cases.

Having determined that the lesion is not central, it remains to fix the seat of the lesion in the lower segment of the motor path. To do this the associations of the paralysis must be taken into account. If no other nerve than the facial is implicated and taste is unimpaired, the lesion is probably just within the stylo-mastoid foramen or outside the skull. If the sense of taste is involved in the anterior two-thirds of the tongue on the side of the lesion, the seat of the disease is in the Fallopian canal at some point between the junction with the chorda tympani and the geniculate ganglion (where the great superficial petrosal nerve containing the taste-fibres joins the trunk of the nerve). Of course the disease may have spread to this region from a more peripheral region. Disease of the nerve at the geniculate ganglion has been thought to cause paralysis of the palate, but it is extremely doubtful if this is so. Inequality of the two sides of the palate has no localizing value in connection with facial paralysis. If there is complete deafness in the ear on the side of the lesion, and if this deafness dates from the time of the facial paralysis, the disease is in the trunk at the base of the brain, or (in rare cases) in the internal auditory meatus. If paralysis of the sixth nerve is associated with paralysis of the facial, the lesion is no doubt in the pons, and in all probability in its posterior part, where the fibres of the seventh pass around the sixth nucleus. Crossed hemiplegia also points to a partial lesion. Disease of the facial nucleus is probably indicated where all muscles supplied by the nerve are paralyzed except the orbicularis oris (which is probably innervated by the twelfth nerve). There is one simple point in the diagnosis of facial paralysis, the neglect of which sometimes leads to error. When contracture develops in the paralyzed muscles the zygomatici draw up the angle of the mouth on the affected side. Hence an error may be made as to the side affected, unless the patient is made to move both sides of the mouth. Patients and their friends can seldom be relied upon in their statements regarding the side affected; they usually think the palsy is on the side they describe as "drawn," *i. e.*, the normal side, on which the features appear to them distorted.

Prognosis. By far the most important indications as to prognosis are derived from the electrical reactions, which enable us to make a forecast in many cases before there is any recovery in power. If there is no loss in irritability of the nerve to galvanism or faradism at the end of ten days the paralysis will clear up in a few weeks or a month. If nerve irritability is much lowered, but not lost, after the lapse of two weeks, there will probably be complete recovery of power in the course of two or three months from the onset. Where the excitability of the nerve is entirely lost by the end of a fortnight, the paralysis will probably endure many months at least. The possibility of slight recovery is not gone until both faradic and galvanic irritability in the muscles has been lost more than a year. There may be return of power long before the electrical reactions become normal. There is some persistent loss of power in almost in all cases where there has been complete paralysis of the face for more than one month.

Occasionally the electrical reactions are misleading. A severe facial paralysis may occur without any evidence of the R. D., and the R. D. may be well marked in mild cases.

The nature of the lesion causing the paralysis does not greatly affect the prognosis, with the following exceptions: The prognosis is bad in the rare cases where the suddenness of onset justifies the diagnosis of hemorrhage into the nerve; good when due to a syphilitic lesion, when it is an uncomplicated palsy, and when the cause is a multiple neuritis (if death does not occur).

Treatment. In every case of facial paralysis an effort should be made to remove or modify the pathological process on which the loss of power depends. In cases where there is suppurative ear disease or mastoid disease the pus should be given free exit. In any case where there is even the smallest likelihood that there has been syphilitic infection, iodide of potassium should be given freely, and if the infection has been recent, mercury should be added. In all neuritic cases of rapid onset the patient should be given a diuretic and a brisk purge, and a fly-blister should be placed over the exit of the nerve from the skull or over the mastoid. The blister should be followed by hot fomentations over the nerve, the applications being made for half an hour several times daily for two or three days. After the patient has been purged, salol should be given in doses of five grains hourly until forty or fifty grains have been taken in the course of the day. This should be continued, if possible, until the end of the first week. In severe cases the patient should remain in the house, if possible, during the first week. The diaphoretic effect of a hot bath daily during the first days may be of service in reducing the local inflammation.

There are no facts to show that the therapeutic application of electricity in any form exerts any influence either in checking the progress of degeneration in the nerve-fibres or in aiding their regeneration. Nevertheless the stimulation of the paralyzed muscles by the galvanic current causes them to contract, and it is highly probable that their nutrition is improved by such stimulation; and, owing to this improved nutrition of the muscles they are in a condition more favorable to rapid repair when the nerve has become regenerated than if they had remained unstimulated. It is upon this ground alone that the use of electricity is to be recommended in facial and other peripheral palsies. Electricity should not be employed from the beginning in facial paralysis, but only after the lapse of a week, when the galvanic current may be used for about ten minutes daily, or twice daily, in strength just sufficient to cause the muscles to contract. The positive electrode should be placed below the zygoma over the nerve and the negative electrode stroked over the various paralyzed muscles in turn. After a month or six weeks the applications may be reduced in number to three or four per week. It is well to continue the use of the galvanic current after power has begun to return, but it should be stopped when contracture begins. There is no advantage in substituting faradism for galvanism when the muscles begin to react to the former current. When contracture sets in it may probably be lessened by means of local massage. Other treatment directed to the contracture is useless. If the eye cannot be closed it should be protected from injury by means of a shade. (For facial spasm see p. 270.)

DISEASES OF THE GLOSSO-PHARYNGEAL NERVE.

The functions of the glosso-pharyngeal nerve and the symptoms that result from morbid processes involving it have not as yet been satisfactorily determined. One reason for this want of definite knowledge is that the nerve is very seldom involved by lesions affecting it exclusively.

The glosso-pharyngeal nerve is distributed to the back part of the tongue, the soft palate, the upper part of the pharynx, and the Eustachian tube and the cavity of the tympanum.

It has been generally thought that the glosso-pharyngeal is the nerve of taste for the back of the tongue, palate, and fauces. But so far as the back of the tongue is concerned, there is satisfactory evidence that the taste-fibres which have this distribution come not from the root of the glosso-pharyngeal, but from the root of the fifth, though they are distributed with the former nerve. It is, moreover, probable that the sensory fibres to the anterior part of the soft palate and fauces come from the fifth and not from the ninth. The sensibility of the upper part of the pharynx and of the tympanic cavity, on the other hand, is probably subserved by the ninth. A good deal of uncertainty exists also as to the motor functions of the glosso-pharyngeal. The pharyngeal plexus, to which the ninth contributes, supplies the upper pharyngeal muscles, but it is not certain whether the motor fibres come from the ninth or the tenth nerve. It is likely that they come from the ninth. It is even more doubtful whether the ninth supplies any of the fibres of the palate.

The glosso-pharyngeal nerve may be implicated by disease within the medulla or at the base of the brain in the posterior fossa.

The chief intra-medullary lesions in which it suffers are hemorrhage, acute softening, and degeneration, with resulting acute or chronic bulbar paralysis. The pharyngeal symptoms in such cases are probably referable to the affection of the glosso-pharyngeal nuclei. At the surface of the medulla, the nerve is liable to suffer (together with the pneumogastric) from meningitis and new growths.

Isolated disease of the glosso-pharyngeal (especially if bilateral) probably causes difficulty in swallowing.

DISEASES OF THE PNEUMOGASTRIC AND SPINAL ACCESSORY NERVES.

The vagus or pneumogastric nerve has the widest distribution of any cranial nerve, supplying, as it does, the vocal and respiratory organs, the heart, the greater portion of the alimentary canal, and some of the abdominal viscera (liver, spleen). Certain of the functions pertaining to the trunk of the nerve depend not upon the pneumogastric, but upon the fibres which it receives from the accessory portion of the spinal accessory nerve. Hence, it is convenient to consider pathological conditions pertaining to the accessory fibres in connection with those affecting the root of the pneumogastric, and to defer the consideration of disease of the spinal portion of the eleventh nerve.

Owing to the wide distribution of the pneumogastric the symptoms resulting from its derangement are exceedingly numerous and varied. Very often the symptoms of derangement of the pneumogastric nerve generally depend, not on demonstrable lesions of the nerve, but upon functional or nutritional disease. The consideration of this very important class of symptoms belongs properly to the diseases of the organs supplied by the pneumogastric, but it is well to touch upon certain of these symptoms in the present connection. For convenience, we may review the derangements of the pneumogastric generally before passing to a more detailed description of the symptoms of its local derangement.

AFFECTIONS OF THE PNEUMOGASTRIC GENERALLY.

In spite of the long course of the pneumogastric nerve it does not suffer from structural disease with great frequency. The nucleus of the nerve in the medulla sometimes is damaged by the acute vascular lesions, softening and hemorrhage, or by degenerative processes. The adjacent nuclei of the glosso-pharyngeal and hypoglossal nerves regularly suffer when the vagus nucleus is involved. Within the medulla the root-fibres may suffer from the processes just mentioned and from tumor. At the side of the medulla the root-fibres sometimes suffer compression from chronic meningeal disease and from growths from the skull, very rarely from aneurism of the vertebral artery. The nerve-trunk in the neck occasionally suffers from deeply-seated tumors and from wounds, including gunshot wounds. The rare punctured wounds are much more apt to be recovered from than lacerated or incised wounds, which generally cause death by injuring the carotid or jugular. Sometimes the nerve suffers in surgical operations; it has been included in a ligature of the carotid and cut in removal of deep tumors of the neck.

The recurrent laryngeals are liable to suffer in the operation for the removal of goitre. In the thorax, aneurisms, tumors, and enlarged lymph-nodes are among the causes of damage to the nerve. Neuromata of the nerve are very rare. It is thought that exposure to cold may sometimes cause neuritis of the vagus. It is certain that the trunk of the nerve is occasionally involved in the multiple neuritis that depends on toxæmic states, such as diphtheria and septicæmia. Rarely it is implicated in alcoholic neuritis. It is probable that functional derangement of the nerve, temporary in duration, but pronounced in character, sometimes depends on the absorption of toxic substances from the intestinal tract.

Symptoms. The symptoms of pneumogastric derangement are referable either to paralysis or irritation, or to both combined. Spasm of the larynx, vomiting, and slowing of the heart are the chief irritative symptoms. Cases have been recorded of persons who could voluntarily arrest the action of the heart for a few seconds by pressure upon the vagus. The chief paralytic symptoms are rapid heart action, with or without slowing of respiration, and paralysis of the larynx. In some cases the pulse has risen to 200 beats per minute, while the respirations have fallen to 10 per minute. In such cases the pneumogastric has probably been implicated upon both sides (as in cases of multiple neuritis), and a fatal termination has usually taken place.

Examples of functional derangement of the pneumogastric are seen in Cheyne-Stokes respiration, in the irregularity of the heart's action observed in emotion, for instance, in anger and fear (this derangement being doubtless dependent on the cortical connections of the vagus), in the "epigastric auræ" of epilepsy, and in the globus hystericus and laryngeal spasm of hysteria and epileptiform seizures. The close anatomical connections of the vagus nucleus with the nervous mechanism of equilibrium explains the common association of vomiting with severe vertigo. The relation of the vagus and the equilibrial portion of the auditory to vomiting is further illustrated by the fact that derangement of the vagus from gastric disturbance may bring on vertigo where there is slight pre-existing disease of the semicircular canals.

DISTURBANCES REFERABLE TO THE PHARYNGEAL BRANCHES OF THE VAGUS. Branches of the vagus take part in the formation of the pharyngeal plexus, through which the muscles and mucous membrane of the pharynx are supplied. The pharyngeal muscles may be the seat of paralysis or spasm.

Paralysis of the pharynx results from disease of the vagus nuclei, from meningeal disease of the vagus roots, from bone disease at the base, and sometimes from diphtheritic neuritis. Pharyngeal paralysis causes difficulty in swallowing. If the affection of the nerve is one-sided there may be little disability; if bilateral, food entering the pharynx from the mouth lodges there instead of passing down to the œsophagus.

Spasm of the pharynx is always of functional origin and is generally a symptom of hysteria. It is seldom the sole manifestation of the hysterical condition. During the spasms of hydrophobia spasm of the pharynx usually occurs. In pharyngeal spasm, as in paralysis, there is inability to swallow food, but this inability is transient.

DISTURBANCES REFERABLE TO THE LARYNGEAL BRANCHES OF THE VAGUS. The larynx receives sensory fibres from the vagus, but its motor fibres are derived exclusively from the accessory portion of the spinal accessory. The superior laryngeal nerve supplies the crico-thyroid muscle; the inferior laryngeal supplies all the remaining laryngeal muscles which act on the glottis. The vocal cords are abducted and the glottis opened mainly by the posterior crico-thyroid muscle; the cords are adducted, with closure of the glottis by a number of muscles, but chiefly by the lateral crico-arytenoid.

Paralysis of the larynx may originate in many ways; first, from organic disease of the nerve-centres or of the nerves external to the larynx: cortical disease of the laryngeal centre (Semon and Horsley), chronic nuclear degeneration, growths or meningitis involving the nerve-roots, damage of the nerve-trunk by trauma, tumors or multiple neuritis, damage to the recurrent laryngeal by aneurism, growths, or enlarged nodes within the chest or enlargement of the thyroid; second, by paresis of the laryngeal muscles not due to lesions of the vagus centres or nerves (hysterical paresis, weakness from anæmia or prolonged exhaustion, local congestions and inflammations); third, from disease involving minute branches of the nerves in the larynx and causing paralysis of a single muscle (laryngeal growths, cellulitis, perhaps multiple neuritis); fourth, without known cause.

Symptoms. There are three kinds of evidence of laryngeal paralysis: (1) alteration or loss of voice; (2) derangement of the regulation of the entrance of air during respiration; (3) defects in the movement of the vocal cords.

According to the degree to which movement is impaired there are three main types of laryngeal paralysis: (1) bilateral abductor paralysis, a dangerous form of palsy resulting from cold, or local catarrh, or bilateral central or nerve disease; (2) unilateral abductor paralysis, due to pressure on one recurrent laryngeal (often by aneurism); (3) adductor paralysis, usually hysterical in character, though often excited by laryngeal catarrh or overuse of the voice.

The following well-known table of Gowers gives the most important characters of the various forms of laryngeal paralysis:

Symptoms.	Signs.	Lesion.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Spasm of the larynx is not an uncommon affection in children who are rachitic or suffer from some other disorder of nutrition (tetany, for example). Occasionally paroxysmal attacks of laryngeal spasm occur in adults (usually young women). The seizures both in children and in adults are apt to be nocturnal and may be accompanied with severe dyspnœa and cyanosis. Seizures of laryngeal spasm are said occasionally to replace migraine paroxysms (Liveing). The "laryngeal crises" of locomotor ataxia are probably examples of adductor spasm. Very rarely laryngeal spasm is excited by attempts to phonate.

Anæsthesia of the larynx is a rare condition sometimes met with in bulbar disease and in diphtheritic neuritis. Occasionally it is seen as a hysterical manifestation. When it is of functional origin reflex action is preserved; in central or nerve lesions reflex action is lost.

Disturbances Referable to the Pulmonary Branches. There is little positive information as to the effects of disease of the pulmonary branches of the vagus. The muscular fibres of the bronchi are supplied by the vagus, and the paroxysmal contractions which give rise to asthma are thought to originate through the agency of these nerves. There is some reason to think that the pneumogastric exerts a trophic influence over the lungs, and that acute pulmonary congestion and hemorrhage are sometimes occasioned in part by its disease. Various disturbances in the rhythm of respiration are referable to changes in the vagus centres.

Disturbances Referable to the Cardiac Branches. The fibres which inhibit and regulate the action of the heart run in the cardiac branches of the vagus. Instances are recorded in which pressure upon one vagus in the neck caused arrest of the action of the heart for a few seconds (Czermak, Concato). There are also cases where the heart could be inhibited for a few beats by an effort of the will. Slowing of the heart's action has been known to follow ligation of one vagus. The slow heart of certain forms of brain disease (meningitis, rapid compression) probably depends on irritation of the vagus nuclei. A slight reduction of the frequency of the heart's action is common in disturbances of the digestive tract, especially in dilatation of the stomach. This slowing is thought by some to often depend on auto-intoxication (Jacoby).

Excessive rapidity of the heart's action is brought about through paralysis of the cardiac branches of the vagus. This has been noted in some cases of diphtheritic neuritis and in injury of the nerve from various causes. Toxic influences acting upon the the vagi seem competent to greatly accelerate the action of the heart for a time. Loss of function of one vagus may cause only temporary disturbance or none at all. The irregularity of the heart's action observed in sexual neurasthenics and in some cases of chronic alcoholic intoxication depends probably upon a disturbance of nutrition in the vagus (trunk, cardiac branches or centres).

Sensory disturbances referable to the cardiac branches are frequent and varied. A tumor of one vagus has seemed responsible for anginal attacks (Blondin). Neuritis of one vagus has appeared to be the cause of pseudo-anginal seizures (Obolonsky). Nothing is known of the relation of the cardiac branches to angina pectoris. Owing to the occurrence of fatty degeneration of the heart after vagus lesions, in certain cases, a cardiac trophic function has been ascribed to the pneumogastric.

Symptoms Referable to Œsophageal and Gastric Branches. The œsophageal branches of the vagus are rarely disturbed in function. Occasionally disease of the vagus or its centre has occasioned difficulty in deglutition. More frequently spasm of the œsophagus, of functional origin, has been observed. Vomiting results not merely from reflex stimulation of the vagus through its gastric branches, but also by direct irritation of the vagus roots, as in the case of basal meningitis. Gastralgia is thought to originate as a pure neuralgia in some cases; in others as a result of direct irritation of the peripheral endings of the gastric branches. Hunger and thirst have been lost as a consequence of vagus disease (Johnson). Excessive appetite has been present, however, where there was atrophy of both vagi (Swan).

It is extremely probable that the sensation of emptiness experienced in some digestive derangements comes into existence through the agency of the vagus in consequence of the imperfect digestion and absorption of food.

Section of the vagi lessens, but does not arrest the movements of the stomach. Both reflex and central irritation of the nerves are competent to induce vomiting. Paroxsymal vomiting has resulted from intermitting pressure of a tumor on the pneumogastric (Gowers). Pressure on one vagus in the neck as it lay exposed during an operation was found to cause vomiting when the pressure was being exerted (Boinet). The gastric crises of locomotor ataxia are probably due to the irritation of a nuclear lesion. Intestinal symptoms have not been noted as the result of vagal disease.

Prognosis. The prognosis in affections of the pneumogastric depends naturally upon the cause of the derangement of the functions of the nerve. The outlook is bad in all cases of pneumogastric disease of organic origin. In functional disturbance the prognosis as regards life may be good, but may be bad as regards the comfort of the patient.

Diagnosis. The four most characteristic symptoms of vagus disease are paralysis of the larynx, slow respiration, rapid or slow heart action, and vomiting. In order to determine the seat of the lesion, it is necessary to take into consideration the distribution of the symptoms and the nature of the associated symptoms. Disease of the trunk is rare as compared with disease of the branches or roots of the nerve. One-sided laryngeal palsy depends generally on disease in the chest (with implication of other cranial nerves, especially the hypoglossal). A high degree of bilateral laryngeal paralysis suggests nuclear disease; a slight degree may be peripheral in origin. With the exception of chest tumors, it is usually not difficult to make out the existence and situation of compressing lesions of the vagus.

Treatment. To remove the pathological process by which the nerve is damaged is the chief indication of treatment, but this can be accomplished in comparatively few cases. Nuclear disease is usually beyond the reach of treatment. Root disease is often syphilitic and yields to mixed treatment.

The laryngeal divisions of the vagus often call for special treatment, although the causes of recurrent laryngeal paralysis are frequently not within the range of treatment of any kind. When tubercular nodes are suspected to be the cause, tonic treatment, including the use of cod-liver oil, is indicated. The use of electricity is of doubtful value, except in hysterical laryngeal paralysis, where faradism, applied to the interior of the larynx, may cause immediate improvement. When laryngeal paralysis is secondary to inflammation, blisters to the exterior of the larynx, together with hypodermatic injections of strychnine, appear to do good.

PARALYSIS OF THE SPINAL OR EXTERNAL PORTION OF THE SPINAL ACCESSORY NERVE.

The large or external portion of the spinal accessory nerve is essentially a series of fibre-bundles of the motor cervical nerves that ascend to the cranial cavity, only to leave it again with a cranial nerve to be distributed to the trapezius and sternomastoid muscles. The nuclei of the nerve may be implicated in progressive degeneration of the motor nuclei of the cord. The nerve is apt to be involved in the exudate of basal meningitis. It may also suffer in cervical caries from tumors outside the skull and from wounds of the neck. Occasionally it is the seat of "rheumatic" neuritis.

Symptoms. Paralysis of one sternomastoid is shown by difficulty in rotating the head to the opposite side. The trapezius is supplied in part by cervical nerves, but paralysis of the accessory causes loss of function in the upper (occipito-acromion) portion of the trapezius and weakness of the middle portion. The shoulder drops a little, and elevation of the arm is impaired.

Treatment. In nuclear disease nothing can be done. When the paralysis depends on pressure, the treatment consists in the removal of its cause. Electricity and massage should be employed in all peripheral cases.

Spasm of the spinal accessory is considered in Chapter IX., p. 272.

DISEASES OF THE HYPOGLOSSAL NERVE.

PARALYSIS OF THE HYPOGLOSSAL NERVE.

Etiology. Paralysis of the tongue is the chief result of disease in the path of the hypoglossal nerve, and may result from a lesion anywhere in the path between the cerebral cortex and the tongue; *i. e.*, from a supra-nuclear, nuclear, or infra-nuclear lesion.

Paralysis of the tongue from supra-nuclear disease—that is, from disease anywhere in the hypoglossal path, between the nucleus in the medulla and the lowest part of the ascending frontal and the base of the third frontal convolutions—is by far the most common form. Ordinarily supra-nuclear paralysis forms part of the paralysis of hemiplegia, with which it is fully discussed. The lesion is most often hemorrhage or acute softening, but may be any one of the processes that give rise to hemiplegia.

Paralysis of the tongue from nuclear disease is the least common of the varieties mentioned. The hypoglossal nuclei lie so close to the nuclei for the lips, pharynx, and larynx, that some or all of these parts generally suffer with the tongue in consequence of the implication of their nuclei. The hypoglossal nuclei of opposite sides are also so close together that a lesion rarely involves one nucleus exclusively. This is true, not only of chronic lesions like degenerations, but of acute lesions like hemorrhage and softening.

Infra-nuclear disease may be located in the medulla, at the base of the brain, outside the medulla, or external to the skull. Disease within the medulla is usually acute softening or tumor. The lesion usually involves also the motor tract of the same side; hence a crossed hemiplegia arises in such cases. Outside the medulla the roots of origin of the nerve may be damaged by tumors and by the various forms of meningitis that occur in this situation. Outside the skull the hypoglossal is occasionally damaged by penetrating wounds or deep tumors of the neck or by vertebral disease. The spinal accessory is liable to be injured with the hypoglossal external to the skull. Rarely the hypoglossal is compressed by narrowing of its foramen in the skull.

Symptoms. The chief effect of disease of one hypoglossal nerve is loss of power in the tongue. When the tongue is at rest in the mouth, its base is a little higher on the paralyzed than on the unparalyzed side. When the tongue is moved *within* the mouth there is imperfect power of motion *to* the paralyzed side; whenever the tongue is protruded, it *deviates to the paralyzed side*, because it is pushed to that side by the fibres of the genio-hyoglossus on the normal side. In disease of both hypoglossal nerves the tongue cannot be moved in the mouth and cannot be protruded. In such cases articulation is very defective. There is also difficulty in mastication. In supra-nuclear paralysis there is no wasting of the tongue; in nuclear or infra-nuclear paralysis the tongue wastes on one or both sides, according as the lesion is one-sided or bilateral. The muscular tissue above suffers, and sensation and taste are essentially unimpaired. The reaction of degeneration may be demonstrated in the wasted half of the tongue.

Diagnosis. The recognition of the existence of hypoglossal palsy offers no difficulty, though it should be borne in mind that the tongue always deviates toward the weaker side, being pushed over by the genio-hyoglossus. The position of the lesion is shown by associated conditions. If there is hemiplegic weakness on the side of the paralysis of the tongue, the paralysis is supra-nuclear. The existence of nuclear disease should be suspected if there is bilateral atrophic paralysis associated with weakness of the lips and pharynx. Crossed paralysis—that is, paralysis of the tongue on one side and of the arm and leg of the opposite side—makes it probable that the root-fibres are involved in the medulla. Unilateral paralysis associated with corresponding unilateral paralysis of the vocal cord and palate suggests disease at the side of the medulla, although a partial and irregular paralysis of the vocal cord and palate may be associated with hypoglossal palsy due to a lesion external to the skull.

Prognosis. There is usually in time considerable or entire recovery of the tongue palsy where the lesion is supra-nuclear and the patient does not die. In nuclear and infra-nuclear paralyses the prognosis is generally bad, owing to the incurable nature of the process that affects the nucleus or nerve. The outlook for recovery of some power is rather better in syphilitic cases, but is uncertain even then.

Treatment. The treatment of hypoglossal paralysis is chiefly the treatment of the morbid process. If the lesion is syphilitic in nature, mixed treatment should be employed. In all other conditions general tonic treatment is indicated. In inflammatory conditions counter-irritation at the occiput may be tried. The application of electricity to the tongue involves considerable discomfort to the patient, and is of such doubtful efficacy that there is less inducement to use it than in peripheral paralysis elsewhere.

Hypoglossal spasm, spasm of the tongue, is considered in Chapter IX., p. 275.

CHAPTER XXVIII.

DISEASES OF THE SPINAL NERVES AND THEIR PLEXUSES.

By C. A. HERTER, M.D.

DISEASES OF THE CERVICAL NERVES.

CERVICO-OCCIPITAL NEURALGIA. In this condition the pain is referred to any part or all of the distribution of the first four cervical nerves, but it is the posterior branch of the second cervical nerve, the great occipital, that is most often affected. This nerve supplies the entire occipital region and the posterior part of the parietal region, and the pain may be felt in this entire area or be limited to its posterior part. The pain is usually constant and dull in character, with occasional attacks of sharper pain. Rarely the pain intermits. The scalp may be excessively tender to pressure and even to movements of the hairs. Tender spots may be found in the following positions: (1) At the exit of the great occipital between the mastoid and the spine; (2) in the triangle between the trapezius and sternomastoid occupied by the cervical nerves; (3) above the parietal boss. Cervico-occipital neuralgia is very liable to extend to the distribution of the fifth, either above to the branches of the first division, or in the neck to the third division of the fifth over the lower jaw. In the latter case there may be swelling of the submaxillary and cervical glands. This form of neuralgia has, further, the following characteristics: It is frequently bilateral, occurs especially in those who have had neuralgia elsewhere (Anstie), generally follows exposure to cold, and is apt to be intractable when once established. Rarely it has been clearly dependent on a carious tooth, or on pressure upon the neck from a heavy load. It may be associated with stiff neck or with torticollis. In one case it was associated with symptoms of a destructive lesion of the cervical sympathetic (Johnson). It is one of the rarer forms of neuralgia.

It should be remembered that occipito-cervical neuralgia may result from caries of the cervical vertebræ. In such cases the pain only rarely extends to the occipital region, and there is early limitation of movement and cervical pain when the patient drops heavily from the tip-toes to the sole of the foot.

The prognosis in primary cervico-occipital neuralgia is good except where the disease occurs in the second half of life and has become established.

Treatment is to be carried out on the principles described under neuralgia of the fifth. Counter-irritation to the neck and occiput is the most effective local measure. The Paquelin cautery should be used over the painful area and on either side of the vertebral spines. A fly-blister may be used instead of the actual cautery. The general health should receive careful attention. In one case adhesions of the superior cervical ganglion of the sympathetic and the cord below were freed by operation (Johnson). The occipital and vertical pain, which had been severe, entirely ceased for six weeks, when it returned as before.

AFFECTIONS OF THE PHRENIC NERVE. Paralysis of both phrenic nerves causes entire inaction of the diaphragm. Owing to this inaction the abdominal viscera do not descend with inspiration, and the upper part of the abdomen does not advance or may even be retracted. The action of the thorax may be increased. Such bilateral paralysis does not embarrass respiration during rest because of the action of the thoracic muscles, but on exertion there is dyspnœa and weak voice. This paralysis disposes to congestion of the base of the lung and renders an existent bronchitis dangerous. One-sided phrenic paralysis causes little or no inconvenience in breathing, and the inactivity of one side of the diaphragm is readily overlooked owing to the movement of the normal side.

Phrenic paralysis is usually the result of a lesion of the cervical spinal cord or its nerve-roots. Sometimes the nerve-roots are compressed in bone disease. The affection is bilateral in all such cases. The nerves themselves are protected from injury by their deep course. Still, they occasionally suffer unilaterally from neck wounds or tumors in the neck or chest. They are also involved on both sides in multiple neuritis, especially in beri-beri and diphtheritic neuritis. It is said that the phrenic may be the seat of a simple neuritis the result of cold. When due to disease of the cord or membranes phrenic paralysis is associated with other paralyses. Mistakes in diagnosis may arise if the following facts are not kept in mind: (1) That hysterical and nervous persons, especially women, often breathe for a time without using the diaphragm ("upper costal type" of breathing); (2) that diaphragmatic pleurisy or peritonitis may cause inaction of the diaphragm owing to the pain caused by motion; (3) that degeneration of the diaphragm itself may impair its power. The prognosis in phrenic paralysis depends on the cause and extent of the paralysis. When due to diphtheria or beri-beri, paralysis of the phrenics is usually fatal. Treatment must be directed to the cause of the paralysis. Electrical treatment is useless.

DISEASES OF THE BRACHIAL PLEXUS AND OF ITS BRANCHES.

BRACHIAL NEURITIS.

Pathology. The term brachial neuritis is used to designate a primary inflammation of several or all the nerves that enter and make up the brachial plexus. By a broader use of the term it might be made to include the inflammatory affections of individual nerves entering the brachial plexus, to be hereafter described, but it is best to restrict it to the condition above indicated.

The pathological process in brachial neuritis is an inflammation of the nerve-sheaths—a perineuritis. Usually it involves in an irregular manner a considerable part of the plexus. In some cases the plexus is little involved, the process being located chiefly in the nerve-roots that belong to the plexus. Such a process is known as a radicular neuritis. The existence of such a form of neuritis is inferred from clinical grounds, and has not been demonstrated. Indeed, a satisfactory description of brachial neuritis based on pathological findings cannot be written at present owing to the infrequency with which autopsies have been made. The clinical grounds for the existence of a radicular neuritis are very strong, and, as will be seen from the discussion of diagnosis, it is of the greatest importance that they should be recognized.

Etiology. Primary brachial neuritis is a rare disease, if we admit as evidence of rarity the fact that many neurologists with wide clinical opportunities have not met with a dozen cases in as many years. The liability of women is probably somewhat greater than that of men. The disease belongs distinctively to the second half of life, five-sixths of the cases being estimated to occur after fifty (Groves). It is a striking fact that brachial neuritis is seldom met except in persons whose nutrition is to some degree perverted. There is sometimes a history of gout or muscular rheumatism, but more frequently there is merely general debility and digestive disorder of long duration. The subjects of brachial neuritis have, in a good many instances, been sufferers from sciatica or lumbago, and sciatic neuritis has been known to precede the development of the brachial affection.

Symptoms. The pain which characterizes neuritic processes in all sensory or mixed nerves is the prominent feature of brachial neuritis and renders it one of the most distressing of painful diseases. The seat of the pain at the commencement of the trouble is usually at a distance from the plexus itself, and affects especially the back of the forearm, the wrist (and perhaps with it the hand), and the region of the scapula. But soon, and in some cases from the first, the chief seat of pain is the region of the plexus itself, especially the axilla or the supra-clavicular region. As the disease increases the pain travels down the nerves of the arm, to which the lines of pain may accurately correspond. In character and intensity the pain varies considerably in the same and in different cases. At first the pain is generally slight and occasional. As the process becomes established the pain grows more severe and often becomes continuous. It is especially aggravated by movement. Usually the pain is paroxysmal; there is more or less continual dull-aching pain with frequent or infrequent attacks of a sharp, lancinating, stabbing nature. When the acute pain subsides the skin of the greater part of the arm may be the seat of a prolonged burning or tingling sensation. Usually the paroxysms of acute pain appear to be spontaneous.

Hyperalgesia is commonly present after the acute seizures in the skin near the plexus. Slight temporary and limited anæsthesia may be present in the skin near the plexus, but complete and lasting anæsthesia is very rare even in the severest and oldest cases.

Loss of power in the arm, especially in the hand and forearm, may occur after a time, but it is usually slight in degree. There is frequently considerable *apparent* motor loss, owing to the pain caused by motion. Trophic disturbances in the arm of the affected side are seldom absent. Wasting of the muscles is present in all severe cases, but is generally slight and irregularly distributed. The wasted muscles show the R.D. in various degrees. The skin in places may be glossy and thin, as in other forms of neuritis, and slight local œdema is common. In severe old-standing cases, arthritic changes in the joints of the fingers occur and cause serious deformities.

Diagnosis. There may be little difficulty in the recognition of fully developed cases of brachial neuritis, but in some cases the condition is frequently confounded with quite different states. From brachial neuralgia the chief distinctive features are the presence in brachial neuritis of points of persistent local tenderness in the nerves, the marked increase of pain on movement, especially abduction of the arm, and evidence of damage, even slight damage, to the nerve fibres of the brachial plexus (muscular atrophy, R. D., and trophic changes in the skin). In neuralgia there may be points of tenderness, but they are shifting and temporary. A history of neuralgia in the patient does not aid in the diagnosis, but a history of gout favors the neuritic nature of the trouble.

Cases of slight neuritis occur when the region of the plexus is not painful, but when there is pain referred to the extremity, the pain being paroxysmal and not constant. If occurring on the left side such pain may be mistaken for the pain of angina pectoris. The important point of distinction is the presence in neuritis of persistent tender points on the nerves. Sometimes the pain of brachial neuritis has led to the suspicion of an aortic aneurism; but aneurism should be thought of only when there is intense, increasing, and constant nerve-pain in the arm, *without* marked nerve tenderness. The late joint changes of neuritis may be mistaken for the changes of rheumatoid arthritis, but this can happen only where the history is not taken into account.

The pains of radicular neuritis may be readily confounded with those of bone disease or a meningeal tumor of the cervical region. Persistent marked tenderness of the nerves of the arm is absent in both the latter. The bone tenderness and slight deformity of bone disease are, of course, distinctive if present. The presence of rigidity and the symptoms of slight unilateral involvement of the cord cannot be misinterpreted and should not be overlooked. Still, cases arise where a diagnosis can be made with reasonable certainty only after watching the progress of the case.

Prognosis. The large majority of cases of brachial neuritis are of long duration, pain and disability of the arm lasting many months, and, in the most obstinate cases, as much as a year. The pain which is suffered in these cases is apt to last long after subsidence of the inflammatory process in the plexus, and this post-neuritic pain is particularly prolonged in elderly persons and those whose nutrition is bad. Scarcely any movement of the arm is possible which does not involve pressure on the sensitive plexus, and hence voluntary movement of the affected arm is greatly restrained or impossible for a long period. Relapses sometimes occur. In the severest cases the nerve-fibres sustain permanent damage.

In bad cases of neuritis a permanent reduction in the size and form of the limb occurs. Changes in the joints of the shoulder, elbow, wrist, and fingers are very apt to take place, and lead to deformity and disability, often of a serious nature. Persons who have recovered from the more acute troubles of brachial neuritis are prone to suffer from neuralgia of the arm and vasomotor disturbances.

Treatment. Roughly speaking, the treatment of brachial neuritis is that of neuritis in general; but in dealing with cases of brachial neuritis it is necessary to bear in mind the extreme sensitiveness of the brachial plexus and the long duration of the pain and tenderness. The first point in every case is to secure immobility in the position which causes least pain and will permit least deformity from contracture. Immobility of the shoulder is particularly important. The arm should be bandaged to the side with the forearm across the chest.

Immobility increases somewhat the chances of joint-stiffness, but, notwithstanding, this must be employed, since the evil effects of motion during the acute stage are far greater. Absolute rest having been attained, the next object is to keep the patient comfortable. The pain may be greatly decreased by mere rest. The spontaneous pain which remains should be combated, if severe, with hypodermatic injections of cocaine in the neighborhood of the most painful places. For this purpose as much as ¼ grain of cocaine hydrochlorate may be necessary twice daily during the acute stage. This is an efficacious way of relieving the pain, and is, on the whole, preferable to the hypodermatic use of morphine. But the danger of a cocaine habit should not be overlooked, and the evil constitutional effects of the drug must be avoided. It is scarcely necessary to insist that its use should not be prolonged a day beyond the time the pain becomes bearable. The first dose of cocaine should not be greater than $\frac{1}{10}$ grain.

When the acute stage of inflammation subsides, gentle massage to the entire arm should be commenced. The massage should be cautiously increased, particular care being taken to avoid increasing pain in the plexus. Properly applied this is the most efficacious measure that can be employed. It helps to relieve pain, to improve the nutrition of the muscles, and to prevent contracture. The contractures should be further combated by placing the contractured muscles in hot water twice a day for ten or fifteen minutes, and gently overcoming the deformity by passive motion.

During the period when there is acute pain electricity should not be employed, but may be used at a later period for the relief of pain (galvanic current) and to help in restoring the nutrition of the muscles by exercising them. For the latter purpose massage is far more efficacious.

COMBINED PARALYSIS OF THE BRACHIAL NERVES. Brachial palsies, in which all or nearly all of the branches of the brachial plexus are involved, are of frequent occurrence, being about one-fifth as common as all single-nerve paralyses (Dana).

Etiology. The following are the chief causes of combined brachial paralysis from conditions external to the spinal canal:

1. Pathological conditions in the neck, especially new growths, which compress the upper part of the brachial plexus or the nerve-roots outside the spinal canal. 2. Obstetrical and other mechanical injuries. 3. Dislocations of the humerus. 4. Fractures of the bones of the arm. 5. Ascending neuritis. 6. Primary brachial neuritis (already considered). Taken as a whole, the brachial palsies are much more common in men than in women, and occur especially in adult life. To infancy belongs an important class of palsies, those due to injury during birth.

Symptoms. The symptoms of combined brachial paralysis vary widely with the distribution and degree of the lesion. According to the degree of the lesion there may be merely transient "heaviness" and numbness of the arm, the weakness disappearing in a few hours at the longest, or the degree of neuritis or pressure may be such as to cause considerable weakness for several months, or again the nerves may be quite severed or torn. The symptoms resulting from such damage are, of course, simply those common to nerve injuries in general; paralysis, atrophy, R. D., together with varying sensory, trophic, and vasomotor disorders. While the precise loss of motion that is met with varies much in different cases there are certain movements which are especially apt to be affected, viz., abduction and elevation of the arm (dependent on the circumflex nerve, and, after elevation to the horizontal position, on the lower cervical, upper dorsal, and posterior thoracic nerve), extension of the arm (dependent on the integrity of the musculo-spinal nerve acting on the triceps), and flexion of the forearm on the arm (dependent chiefly on the integrity of the musculo-cutaneous nerve, acting through the biceps and brachialis anticus). It is important to recognize the following forms of brachial paralysis, which are based partly upon the etiological facts already enumerated, partly upon the seat of the lesion.

Brachial Paralysis from Primary Brachial Neuritis. The paralyses resulting from this well-defined clinical condition are described elsewhere (see Primary Brachial Neuritis).

Brachial Paralysis from Ascending Neuritis (neuritis "migrans"). Here the paralyses are due to a neuritis, perhaps due to infection, which commencing in a single nerve in consequence of an injury, ascends to the brachial plexus, where it spreads to other nerves, often in an irregular way. Thus the inflammation may pass up the ulnar, and reaching the plexus spread to the roots of the median. The spread of the process is usually slow, occupying many weeks, and is generally accompanied with a good deal of pain in the sensory areas whose nerves are involved.

Brachial Paralysis from Fracture or Dislocation. Dislocation of the humerus is a not uncommon cause of brachial paralysis. When the dislocation is subcoracoid the head of the humerus can hardly fail to compress the nerves, which may suffer in any degree. As a rule, the paralysis is severe and extensive, with rapid atrophy and R. D. Trophic and vasomotor disturbances are likely to be pronounced. Fractures of the humerus are particularly apt to damage the musculo-spinal nerve, with or without the ulnar. Rarely the median suffers. The median and ulnar may together suffer in fractures of the bones of the forearm.

Brachial Paralysis from Morbid Processes in the Neck (malignant or tubercular bone disease or syphilitic cellulitis) is usually associated, as might be expected, with irritative symptoms in the region of the nerve affected, especially severe pain, hyperæsthesia, and irregular and considerable muscular contractures. Sometimes a new growth compresses the subclavian artery and so causes weakening of the radial pulse on the same side.

Brachial Paralysis of the Upper-arm Type (Erb's paralysis). This peculiar and distinctive type of brachial paralysis depends on injury to the roots of the cervical nerves, probably the fifth and sixth, at the side of the neck just in front of the edge of the trapezius. The condition (which is not common) is met with in adults, almost exclusively men, and in infants. In adults it depends usually on downward pressure on the neck, as from carrying a heavy weight on the shoulder, but it sometimes arises from non-traumatic processes causing neuritis. In infants it is especially apt to arise from traction on the neck by the finger during birth. It thus contributes a variety of "obstetrical paralysis."

The paralysis regularly involves the deltoid, biceps, brachialis anticus, and supinator longus, sometimes the supinator brevis, and the supra and infraspinati. There is frequently anæsthesia on the outer side of the arm in the distribution of the circumflex and external cutaneous nerves, and there is often much muscular wasting. The character of the palsy differs considerably in adults and infants. In adults the loss of power is often complete, sensory symptoms are pronounced, and the affection is commonly of long standing. In infants the paralysis is more apt to be slight in degree. In fact, the slighter degrees of the palsy may escape notice for some time after the birth of the child. In well-marked cases the paralysis cannot escape detection; the arm hangs by the side with the forearm in extreme pronation. Wasting cannot be detected until several months after the injury, and may appear very slight even then, as it is masked by the large amount of fat over the muscles. Sensation is rarely impaired in infants, and may escape notice when it is. The palsy generally wears away in a few weeks or months; it rarely persists.

Brachial Paralysis of the Lower-arm Type is due to the involvement of the nerves arising from the seventh and eighth cervical and first dorsal roots. In this form of paralysis the arm can still be elevated and the forearm flexed and supinated; the loss of power is located in the triceps, the flexors of the wrist, the pronators, the flexors, and extensors of the fingers, and the muscles of the hand.

Klumpke's Parlaysis (paralysis of the lower roots of the brachial plexus). Lesions which involve the first dorsal root of the brachial plexus and the communicating branch of the second dorsal are characterized by atrophic paralysis of the thenar, hypothenar, and interossei muscles, anæsthesia in the ulnar area of the forearm and arm, and certain ocular symptoms. These ocular symptoms comprise myosis on the side of the lesion, sluggish contraction of the pupil, diminution in the size of the palpebral fissure, and recession of the eyeball, and are thought to depend on implication of the ramus communicans of the first dorsal nerve (Klumpke, Pfeiffer).

This form of brachial paralysis is of interest, chiefly on account of the localization of the lesion to which it must be referred. It probably has no claim to be regarded as a distinct type upon pathological grounds. It may arise as a primary neuritis or as the result of pressure by a new growth arising from the chest or vertebræ. In the former case the lesion may spread to the roots of the plexus, in the latter case it is apt to invade the spinal cord and cause symptoms of pressure and destruction of the cord elements.

Diagnosis. The diagnosis of the seat of the lesion in combined arm paralysis is not difficult, if the facts regarding the formation and distribution of the brachial plexus be borne in mind. The diagnosis of the character of the lesion may be difficult if the paralysis be non-traumatic. The process is usually neuritic in character, but whether this be primary or secondary to some other pathological condition it may be impossible to say at first.

The distinction of peripheral from central brachial palsy is not usually difficult. Focal processes in the cervical spinal cord give rise to paralysis, wasting, and sensory disturbances in the arm. But there can be no error if the following considerations be taken in account: in spinal cord disease there is usually no correspondence between the disturbances of function and the functions of particular peripheral nerves; there are no local pathological conditions which would explain a nerve-lesion, and, most important of all, there are evidences of impairment of the conducting functions of the cord (weakness and sensory disturbances and alteration in reflex action below the lesion), not rarely bilateral in character.

Treatment. There is little to be said of a special nature regarding the treatment of brachial nerve paralysis; treatment consists in removing so far as possible the cause of the paralysis, whether this be neuritic or traumatic. The treatment of neuritis has been already considered. Any source of pressure must be got rid of. Divided nerves must be sutured, primary suture always being used when practicable. Electrical treatment and massage should be employed in all cases where there is serious damage to the nerves. If electricity be employed to obtain muscular contraction it should be applied at least once daily. (See also Prognosis and Treatment of Paralysis of the Spinal Nerves).

CERVICO-BRACHIAL AND BRACHIAL NEURALGIA. These terms are applied to neuralgic affections of the four lower cervical and first dorsal nerves. The pain is most often in the distribution of the ulnar nerve, but it is not rarely most marked in the axilla or on the shoulder. It may be referred to any part of the arm or hand. Tender points are commonly found. An axillary, a circumflex over the deltoid, a superior ulnar at the level of the elbow, and an inferior ulnar where the nerve passes in front of the annular ligament of the wrist, are the chief painful points. As a rule, there is some dull continuous pain in addition to the paroxysms of sharp pain. It is very common for cervico-brachial neuralgia to be increased or excited by movement, and some of the severest cases are of the nature of occupation neuroses, and are seen in pianists, violinists, and those who write to excess. But of course the mere fact that a movement like that of writing or piano-playing excites the pain does not show that the case is an occupation neurosis. The pain may radiate to the side of the chest and thus suggest angina pectoris, or it may be associated with neuralgia of the fifth. If trophic changes occur in the skin this is evidence of neuritis.

Brachial neuralgias are not so often associated with diathetic states as neuralgias elsewhere, but are often the consequence of injury (especially blows on the shoulder and injuries to the ulnar). An extensive neuralgia without neuritis may result from a slight injury to the arm. Injuries to the finger-nerves are seldom followed by neuralgia. "Median cephalic" neuralgia is said to have been comparatively common in the days of phlebotomy (Anstie). Carious teeth are said to be an occasional cause of brachial neuralgia (Salter). The prognosis is good where there is a removable cause. Where no such cause is found the affection may be very obstinate. Rest for the affected part is often an essential part of treatment. Sometimes immobilization of the arm by means of a plaster bandage is of much service. (For general treatment, see Neuralgia of the Fifth).

PARALYSIS OF SINGLE NERVES. *Nerves of the Upper Extremity.*

Paralysis of the Posterior Thoracic Nerve. This nerve is derived from the fifth and sixth cervical nerves, and injury to it causes paralysis of the serratus magnus muscle. The main effects of the paralysis of this muscle are: 1. Rotation of the scapula on its vertical axis when the arm is put forward, with recession of the edge of the scapula from the thorax, so-called "winged scapula." 2. Rotation inward and upward of the lower angle of the scapula when the arm is advanced. 3. Weakening of the power of elevating the arm above the shoulder. There is often severe pain in the neck and shoulder during the onset of the paralysis.

The damage to the nerve is generally in the neck, either by direct pressure of a heavy angular object on the shoulder or by a violent muscular effort, as in lifting a heavy hammer. Occasionally the nerve is injured by a wound or contusion, and sometimes a neuritis is set up by exposure to cold. The palsy is generally one-sided; very rarely it is bilateral. It is very much more common in men than in women. Usually the affection is right-sided. Serratus paralysis occurs in progressive muscular atrophy, but then there are other muscles involved. Paralysis of the posterior thoracic nerve is of long standing where the loss of power is complete. It should be remembered that it is a very rare form of palsy.

Paralysis of the Supra-Scapular Nerve. Damage to the supra-scapular nerve causes paralysis of the supra-spinatus and infra-spinatus muscles. The paralysis of the former gives rise to obtrusive symptoms, but paralysis of the infra-spinatus causes a loss of outward rotation of the humerus. An important effect of this loss is the inability to carry the hand from left to right as in writing.

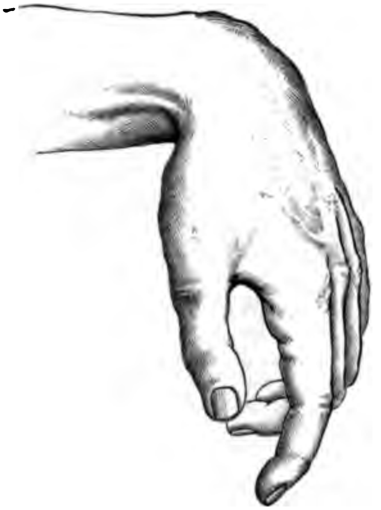
There is seldom isolated paralysis of the supra-scapular nerve, but it is not rarely affected together with the circumflex, in consequence of dislocation of the head of the humerus.

Paralysis of the Circumflex Nerve causes loss of power in the deltoid and teres minor muscles. The paralysis of the former is far more important. Its chief sign is inability to raise the arm. In some cases there is loss of sensation on the outside of the upper part of the arm over the muscles. The deltoid wastes, and this alters the contour of the shoulder. After a time trophic changes occur in the shoulder-joint (the circumflex sends filaments to the joint), and adhesions may form. Paralysis of the circumflex nerve is easily recognized. It is impossible to confound it with the loss of motion that is seen in anchylosis of the shoulder-joint if it is remembered that in the latter state passive motion of the arm moves the scapula as well as the arm. The circumflex nerve is often injured by falls on the shoulder and by dislocations of the head of the humerus. Rarely it is the seat of "spontaneous" neuritis. It is sometimes paralyzed with other nerves belonging to the brachial plexus (see Combined Paralysis of the Brachial Nerves) in a highly characteristic manner.

Paralysis of the Musculo-cutaneous Nerve (External Cutaneous, Perforans Casserii) causes loss of power in the biceps and brachialis anticus muscles, the effects of which are unmistakable (loss of flexion of elbow, especially marked when the forearm is supinated and the supinator longus cannot act as a flexor). There may or may not be anæsthesia on the outer half of the forearm in front and behind, and over the arm in its lower part and outer side. The musculo-cutaneous is rarely paralyzed by itself.

Paralysis of the Musculo-spiral Nerve in the vicinity of the brachial plexus causes loss of power in all the extensors of the forearm and wrist and the supinators. Extension of the elbow is impossible, the wrist drops, and the fingers are flexed at their distal joints. The fingers, can, however, be extended by the interossei and lumbricales if the first phalanges are flexed. Supination, though not entirely lost (the biceps being active), is greatly weakened. After a time the excessive flexion of the carpus leads to undue prominence of the carpal bones and the synovial sacs at the back of the wrist. When the damage to the nerve is serious there is in a few weeks a perceptible diminution in the size of the forearm, due to the atrophy of the paralyzed extensors, and the muscles present the R. D. in various degrees.

FIG. 276.



Wrist-drop in musculo-spiral paralysis. (LEUBE.)

If the damage to the nerve is in the middle of the arm the biceps is generally involved. The supinator longus escapes only in rare cases of musculo-spiral paralysis.

The loss of sensation in the parts supplied by the musculo-spiral varies considerably in different cases. In actual division of the nerve above its cutaneous branches there is usually loss of sensation in the outer part of the arm (about one-quarter of its circumference) from the level of insertion of the deltoid to the external condyle of the humerus, and on the back of the forearm on the outer side above, fading into normal sensation in the lower third of the forearm. The skin on the dorsal surface of the hand is anæsthetic over the thumb and metacarpal bones of the thumb, index, and middle fingers. In many cases of musculo-spiral paralysis there is no anæsthesia either in the hand or arm.

Musculo-spiral paralysis is of frequent occurrence, the course of the nerve exposing it to various kinds of injury. High up the nerve may be damaged by the pressure of a crutch. Indeed, crutch paralysis is usually due to musculo-spiral injury. The nerve is apt to be torn in cases of fracture of the humerus, and may be pressed upon by callus. The most common cause of the paralysis, however, is damage to the nerve during sleep. The patient lies on a hard bed or on the floor with the arm under him and receiving his weight. The nerve suffers as it passes around the humerus about the middle of the arm. But there is very good reason to think that the majority of these sleep palsies of the musculo-spiral are due not to pressure, but to stretching of the plexus from extension of the arm above the head. This is also the origin of the palsy that occurs during extension of the arm in some cases where the patient is anæsthetized. So often does this occur in patients who have fallen asleep after excess in alcohol that it is known as "Saturday-night paralysis" or "Sunday-morning paralysis," from the times at which the paralytic effects usually arise or are detected.

It is necessary to distinguish musculo-spiral paralysis from some forms of multiple neuritis—notably from lead paralysis and alcoholic neuritis. In lead paralysis the muscles supplied by the musculo-spiral nerve are involved, but the affection is almost invariably bilateral (though the two sides may suffer very unequally), and the supinator longus muscle is almost exempted from the palsy. In musculo-spiral paralysis, on the contrary, the paralysis involves only one nerve (in rare cases a cause of musculo-spiral paralysis operates bilaterally), and the supinator longus is almost invariably included in the palsy. Moreover, the onset of lead palsy is gradual and the development of musculo-spiral paralysis is rapid or sudden. The cause of musculo-spiral paralysis is usually readily elicited. The distinction from alcoholic neuritis is usually extremely easy (see Multiple Neuritis). The bilateral character of the paralysis, its extensive distribution, and the alcoholic history will prevent error even when the paralysis affects chiefly the upper extremities.

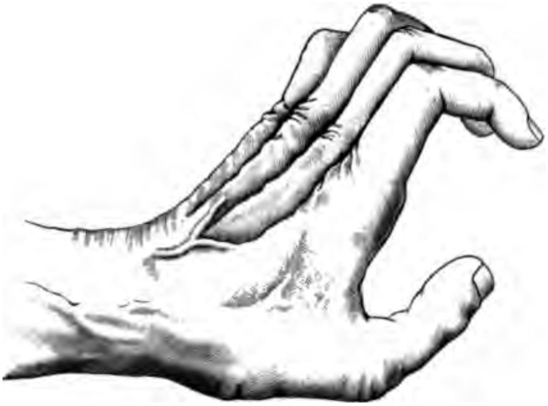
Paralysis of the Median Nerve. Severe damage to the median nerve above its muscular branches causes loss of power in the flexors of the fingers (excepting the ulnar half of the flexor profundus), in the pronators, in the flexor carpi radialis, in the two outer lumbricales, and in all the muscles of the ball of the thumb except the abductor pollicis and the ulnar half of the flexor brevis pollicis. In consequence of this loss of power the ability to flex and pronate the forearm is greatly diminished, but not abolished. Flexion at the wrist to the ulnar side is still possible by the action of the flexor carpi ulnaris; pronation is feebly performed by permitting the weight of the hand to rotate the forearm after it has been supinated, the supinator longus being capable only of pronating the arm to a position midway between supination and pronation. The thumb is extended and abducted in a characteristic manner, and cannot be brought in contact with the tips of the fingers. The second phalanges can no longer be flexed on the first, and in the first and second fingers there is also loss of flexion of the third phalanges. The first phalanges are flexed by the interossei. The most characteristic distribution of anæsthesia is as follows: On the palmar surface loss of sensation on the radial side of the palm and of the thumb, index and middle finger, and the radial side of the ring finger; on the dorsal surface, loss of sensation on the index and middle fingers and on the radial side of the ring finger for a variable distance, and perhaps on the ulnar side of the thumb. (See Figs. 241 and 242, p. 747.)

The degree and extent of the anæsthesia vary much in different cases. Sometimes there is no affection of sensation whatever. In a case of severe damage to the median the appearance of the hand and forearm soon becomes highly characteristic. The forearm is much atrophied on the radial side in front, the wrist is inclined to the ulnar side and perhaps hyper-extended, the ball of the thumb is greatly wasted, the head of the metacarpal bone is prominent, and the thumb is usually rotated out, so that its palmar surface is on a plane with that of the hand, as is the case in apes.

The median nerve is often injured. It suffers most frequently just above the wrist-joint, where it is more superficial than in the rest of its course, and is readily divided. It may, however, be damaged in almost any part of its course. In the forearm it is not rarely injured in fractures of the ulna and radius. In the upper arm it is most often invaded just above the bend of the forearm. It is said to be in some cases the seat of primary neuritis. Very rarely it is injured by violent contraction of the pronator radii teres.

Paralysis of the Ulnar Nerve. When the ulnar nerve is divided or severely damaged above the origin of all its branches there is loss of power in the ulnar half of the flexor profundus digitorum, in the flexor carpi ulnaris, in all the muscles of the little finger, in all the interossei, in the two ulnar lumbricales, in the abductor pollicis, and in the inner head of the flexor brevis pollicis. When this paralysis has lasted some time (three or four weeks or longer), the action of the unparalyzed opposing muscles brings the hand into a very characteristic position. (See Fig. 277.) The wrist is slightly bent backward and to the radial side of the forearm by the action of the extensor carpi radialis, extensor carpi ulnaris, and flexor carpi radialis. The hand is considerably thinner than normal owing to the wasting of the interossei and the muscles of the little finger, which leaves the metacarpal bone of that finger very prominent. There are depressions between the metacarpal bones, but there is a particularly marked depression on the radial side of the metacarpal bone of the index finger on the back of the hand, owing to the wasting of the first dorsal interosseus.

FIG. 277.

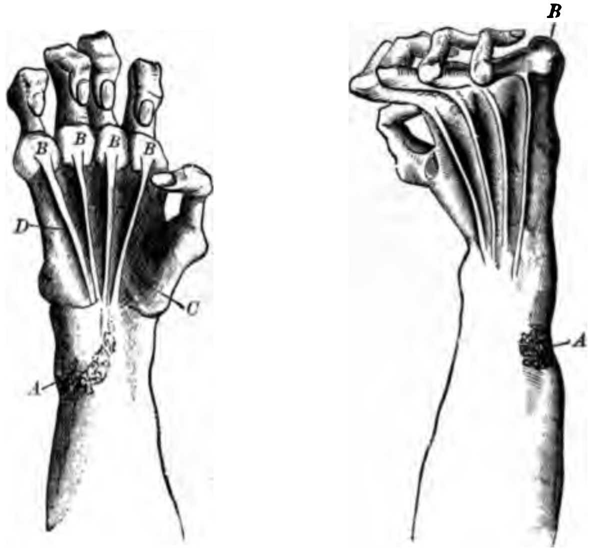


Position of wrist, hand, and fingers in ulnar paralysis. (LEUBE.)

The paralysis of the interossei leads to a deformity which is almost distinctive of ulnar paralysis. The fingers cannot be flexed at the first, or extended at the second and third phalanges, and, in consequence of this the extensor communis digitorum, flexor sublimis digitorum, and part of the flexor profundus digitorum by their contracture, overextend the first phalanges and flex the second and third.

This deformity of the hand is known as the "bird-claw hand," or the "claw-like hand." (See Fig. 278.) The deformity is especially marked in the third and fourth fingers; the first and second are less affected because their lumbricales escape paralysis. This deformity occurs not only when the ulnar nerve is damaged high up above its muscular branches, but also in injuries of the wrist, though it is perhaps less extreme in the latter class of cases.

FIG. 278.



Position of hands and fingers in ulnar paralysis of long standing; "bird-claw hand," "main en griffe." A, wound of the ulnar nerve; B, ends of the metacarpal bones; D, tendons of the flexor sublimis; C, muscles of the ball of the thumb. (DUCHENNE.)

The state of sensation in ulnar paralysis varies considerably; in some cases there is no anæsthesia; in others of severe damage to the nerve the loss involves, on the palmar surface, the little finger and the ulnar half of the ring-finger, together with the corresponding portion of the palms, on the dorsal surface, the little finger, the ulnar half of the ring-finger, and the corresponding region of the dorsum of the hand. (See Figs. 243 and 244, p. 748.)

The ulnar nerve is probably more often damaged than any other spinal nerve. It is frequently injured in wounds of the forearm, especially in wounds at the wrist, where the nerve is superficial. When the nerve is injured at the wrist it is generally above the origin of the dorsal cutaneous branch. Higher up in the forearm the nerve may be hurt by fractures of the ulna and radius. At the back of the elbow, just external to the olecranon, the nerve is very liable to suffer from wounds, and is occasionally injured by pressure or contusion. Long-continued flexion at the elbow sometimes suffices to cause ulnar paralysis. Sometimes paralysis arises in this way during sleep. The nerve is very rarely injured in the arm above the elbow. Sometimes the symptoms of an apparently spontaneous ulnar neuritis are observed in persons in reduced health.

The diagnosis of ulnar paralysis is simple. Error may possibly arise in rare cases of disease of the cervical enlargement, in which the ulnar nerve distribution is chiefly affected. Other evidences of spinal cord disease are never wanting in these cases.

In cases where nerves of the arm, and especially of the forearm, have been injured, and tendons and muscles have been injured with the nerves, it may be difficult to distinguish the effects of the nerve injury and the effects of injury to the tendons and muscles. This is because there is often considerable cicatricial change in muscles and tendons, leading to deformity, which may simulate closely that of nerve injury, and because certain of the most important signs of nerve injury, namely, changes in the irritability of muscles and nerves, may be obscured by the contraction that sets in. Even the most careful attention to every detail in the history and examination of such cases may not enable the observer to arrive at a correct conclusion as to extent and situation of the nerve damage.

DISEASES OF THE DORSAL NERVES.

The affections of the dorsal nerves are mainly sensory. The motor fibres of individual dorsal or intercostal nerves sometimes suffer in wounds, but such localized damage does not produce recognizable symptoms. When there is extensive motor paralysis of the dorsal nerves, it is in consequence of vertebral or spinal-cord disease.

INTERCOSTAL NEURALGIA (Dorso-intercostal Neuralgia). This rather common form of neuralgia is especially frequent in women. Frequently it follows exposure to cold or a contusion or other injury. It probably does not occur spontaneously, except in persons whose nutrition is impaired. It may involve one or more of the intercostal nerves, from the third to the ninth, and is characterized by acute stabbing pains, which shoot along one or more spaces. In the intervals between the exacerbations there is usually a dull, constant intercostal pain. When the neuralgia has existed for some time tender points may be found at the exit of the branches of the intercostal nerve, which is the seat of pain, near the vertebræ, near the median line anteriorly, and in the axillary line.

Intercostal neuralgia of the type just described is often obstinate and of long duration.

MAMMARY NEURALGIA (Mastodynia) is a variety of intercostal neuralgia which occurs in neurasthenic or anæmic women, with or without disturbance of the function of the gland. In some cases it is referable to over-lactation. The pain is most often on the left side, is sharp in character, and limited to the anterior part of the upper intercostal spaces. Another variety of intercostal neuralgia is the trifling neuralgic pain known as pleurodynia. In such cases the pain is migratory, and is unassociated with points of tenderness. It is necessary to distinguish all these forms of neuralgia from the sharp pleuritic pains that occur so often in neurasthenics and in those who have had pneumonia. The pain in these cases is apt to recur in the same positions, and is influenced by the movements of respiration. Probably it depends on slight localized dry pleuritis. It is seen most often in persons who are free from pulmonary tuberculosis. In many cases this pain varies with extreme regularity with the well-being of the patient, often coming on only when he has been fatigued. In the primary intercostal neuralgias, which are comparatively infrequent, this relation to the changing vitality of the patient is not so clearly seen.

In every case where the diagnosis of primary intercostal neuralgia is made great care must be taken to exclude the influence of organic conditions of the chest, mammæ, vertebræ, and spinal cord.

Treatment. Counter-irritation over the affected space usually gives at least temporary relief; sometimes it renders recurrence of the pain less frequent. The actual cautery (Paquelin) seems the most effective means of applying counter-irritation. The point should be applied superficially and quickly. If this is not available, a mustard plaster or fly-blister should be applied. Chlorodyne in small doses often gives relief, but its repeated use cannot be recommended. It is exceedingly important to employ tonic treatment and rest, and to keep the bowels freely open. In rare cases section of the affected intercostal nerve has been resorted to and has given relief.

Herpes Zoster (Shingles). Although herpes zoster occurs in various parts of the body, it affects with especial frequency the distribution of the dorsal nerves. The disease is characterized by the rapid development of groups of vesicles in the course of a nerve, associated with neuralgic pain in the same region. The development of the vesicles reaches its height in about ten days. The pain may precede or follow the development of the eruption. The pain which precedes the eruption is usually moderate in severity, and grows less as the vesicles develop. The pain that succeeds the eruption is usually acute, lancinating, severe, and accompanied with tenderness of the skin. The severity and persistence of this pain is apt to be proportioned to the age of the patient. In the young it is transient; in those past fifty there is always a liability that the pain will prove intractable, or at least of long duration. In rare cases intercostal neuralgia precedes for a long period the appearance of the eruption. The dermatitis is usually one-sided, and is generally located in the lower intercostal region.

There is satisfactory evidence that the eruption of herpes zoster depends on irritation of the nerve or nerves along whose distribution it occurs. Probably the intercostal nerves are always the seat of neuritis, and there is good reason to think that this neuritis is due to inflammation of the corresponding ganglion of the posterior nerve-roots (Baerensprung, Weidner, Wyss, Charcot). The neuralgia is thus symptomatic.

Exposure to cold, injury, and the medicinal use of arsenic have seemed to account for some cases. Usually we are wholly ignorant of the nature even of the exciting cause.

The affection lasts a few weeks in most cases. The chief factor in the prognosis is age. In some cases where the neuralgia subsides with the eruption it recurs from time to time for a long period.

Treatment is not satisfactory.

Galvanism relieves the pain of herpes zoster, as a rule. Ointments of opium and belladonna and dusting powders of camphor and morphine sometimes give relief. Internal medication appears to be useless, except where the general health is much impaired. The vesicles should be protected from irritation.

DISEASES OF THE LUMBAR AND SACRAL PLEXUSES AND OF THEIR BRANCHES.

With the exception of sciatic neuritis, lesions of the nerves of the lower limb are considerably less frequent than those of the upper extremity.

The *lumbar plexus* (which is made up of the first three lumbar roots and one-half of the fourth) is only occasionally damaged by disease. Abdominal tumors (ovarian tumors, growths, and tuberculosis of the abdominal lymph-nodes) and psoas abscess are among the more frequent causes of such damage. The nerve-roots from which the plexus arises may suffer pressure in vertebral caries or malignant disease or from meningeal tumors or inflammations. Very rarely the plexus is the seat of an apparently primary neuritic process, and very rarely, also, a lumbar neuritis is due to upward extension of inflammation along the lumbo-sacral cord.

The lumbar plexus supplies the skin of the lower part of the abdomen, of the front and lateral aspects of the thigh, and of the inner surface of the leg and foot. Through its branches, the obturator and anterior crural nerves, it supplies the numerous muscles, the flexors and adductors of the thigh, and the extensors of the knee. When the lumbar plexus is the seat of disease, the symptoms are referred to the parts just mentioned, but it is rare for all parts of the plexus to suffer, and the parts involved generally suffer unequally.

Paralysis of the Obturator Nerve rarely occurs alone. When it does, it is the result of pressure during labor. The chief symptom of such paralysis is loss of the power of adducting the thigh; the affected leg cannot be put across the other. Most cases of paralysis of the obturator nerve depend on damage to the lumbar plexus.

Paralysis of the Anterior Crural Nerve causes loss of power and atrophy in the extensors of the knee and loss of knee-jerk from damage to the reflex arc. When the nerve is damaged within the pelvis the branch to the iliacus muscle is involved and there is impaired power of flexing the hip, as well as loss of extension of the knee. Paralysis of the anterior crural nerve also causes anæsthesia, which involves the entire thigh, with the exception of a strip of variable width along the back of the thigh (supplied by the sacral nerves) and the inner side of the leg and foot. The anterior crural may be damaged in the thigh or groin, may suffer from pressure during parturition or from dislocation of the hip, or may be implicated in disease of the lumbar plexus.

Paralysis of the Superior Gluteal Nerve causes loss of abduction and circumduction of the thigh, from paralysis of the gluteus minimus and medius. As an isolated paralysis, apart from affections of the plexuses, it is a very rare condition.

SCIATIC NEURITIS (*Sciatica*). Sciatic neuritis is an inflammation of the great sciatic branch of the sacral plexus. The inflammation involves principally the sheath of the nerve, but the pathological changes often extend to the interstitial tissue of the nerve. The nerve-fibres themselves are damaged secondarily. During the acute stage the sheath is red and swollen and may be the seat of minute hemorrhages. All these inflammatory changes (described more minutely under "Neuritis in General," Chapter XXV.) are most pronounced at two points in the course of the nerve, near the sciatic notch and about the middle of the thigh. The pathological changes may be limited to these spots or may extend with less intensity to a large part of the course of the nerve.

Sciatic neuritis gives rise to the clinical condition known as sciatica, and is, indeed, its chief cause. The word sciatica is, however, legitimately employed to include cases of pain probably in the sciatic nerve due, not to organic changes, but to slight alterations in the nutrition of the nerve—sciatic neuralgia. The word is less strictly used to designate all painful conditions in the neighborhood of the sciatic nerve; as, for example, the sciatic pain due to tumor in the pelvis pressing on the plexus. It is best that the term should be restricted to sciatic pain arising from causes within the sciatic nerve.

Etiology. Sciatic neuritis is a disease especially of middle adult life, but is not rarely met with between sixty and seventy. Occasionally it is met with during adolescence. It is more than twice as common in males as in

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Etiology. Sciatic neuritis is a disease especially of middle adult life, but is not rarely met with between sixty and seventy. Occasionally it is met with during adolescence. It is more than twice as common in males as in females. A disturbance in general nutrition, inherited or acquired, is probably present in all cases of sciatic neuritis. The nature of this disorder is but imperfectly known. In some cases the sufferers are the subjects of gout or the descendants of gouty persons. In other cases there is a marked disposition to rheumatism, especially muscular rheumatism. In some cases, probably in many, there is an excessive excretion of uric acid as compared with urea—an evidence of imperfect digestion though there may be no appreciable local digestive disorders.

Lumbago is sometimes followed by sciatic neuritis about the ischiatic notch, and in these cases the neuritis arises probably by an extension of interstitial inflammation from the muscles to the nerve. It is doubtful whether syphilis acts as a cause of sciatic neuritis, except by lowering the general health.

There is commonly an exciting cause for sciatic neuritis. The most frequent exciting cause is exposure to cold, either local exposure (as from sitting on wet grass, or a draughty water-closet, or from standing in the wet) or, less frequently, general exposure, as to a draught of cold air. Mechanical causes are sometimes competent to start up a sciatic neuritis. The pressure on the legs by the edge of a hard seat is such a cause, and a common one. Violent muscular contraction and over-exertion are much rarer causes.

Symptoms. The cardinal symptom of sciatic neuritis is pain in the course of the nerve-trunk, and, less frequently, along the branches of the nerve or in its distribution. The onset of the pain is usually gradual, occasionally rapid. At first the pain is experienced only upon motion, particularly such motion as puts the nerve on the stretch. This pain grows more severe and recurs from slighter causes until a time may arrive when almost any movement of the affected leg causes severe pain. While the pain on movement gradually increases spontaneous pain is added. This spontaneous pain is most frequent and most severe in two places, at the exit of the nerve from the sciatic notch and at a spot about the middle of the back of the thigh. The pain is not always confined to the neighborhood of the nerve, but extends to its areas of distribution, especially to the following points: (1) Just above the hip-joint, below the posterior superior spine of the ileum; (2) in the popliteal space; (3) below the head of the fibula; (4) back of the outer malleolus; and (5) on the dorsum of the foot. In character the pain may be sharp or dull. It is often described as burning. Usually it is aggravated at night. In some cases the pain seems to shoot downward from the uppermost part of the nerve. As the pain grows more pronounced, tenderness of the nerve-trunk to pressure appears. This tenderness can be brought out even when it is slight in degree, though stretching the sciatic nerve slightly as the patient lies on his back by flexing the fully extended leg on the body.

Other disturbances than pain are met with in the distribution of the sciatic nerve. Numbness, tingling, and formication are of common occurrence. Areas of complete anæsthesia occur only in severe cases. Somewhat more frequently irregular areas of partial anæsthesia and analgesia are found on the back of the thigh or on the leg. When the sensibility at the back of the thigh is affected this is an indication that the neuritic process has extended to a point above the origin of the small sciatic nerve or that there is a neuritis of this nerve.

Involvement of the motor functions of the nerve is of common occurrence, more common, probably, than is generally supposed. A large proportion of all cases of sciatic neuritis are accompanied with some loss of power in the distribution of the sciatic nerve or of the branches of the sacral plexus. The loss of power may be slight or considerable and is often associated with slight wasting and flabbiness of the muscles with alterations, which are rarely considerable in the electrical reactions. Most often the paresis involves the hamstring muscles (Mann). To elicit this weakness the patient should lie upon the belly and flex the leg on the thigh while one hand of the examiner resists the effort and the other feels the insertions of the muscles. Much less commonly there is atrophic paralysis of the calf-muscles, the anterior tibial or peroneal muscles, or of the gluteus maximus. Paralysis of the latter causes little or no difficulty in walking, but interferes with rising from a chair and with walking upstairs. Occasionally the palsy extends to the erector spinæ, with resulting typical scoliosis (convexity to the side of the neuritis). In rarer instances the pareses are not confined to the side on which there is sciatic pain.

Persons with sciatic neuritis hold the leg stiffly in walking and slightly flexed, owing to the pain caused by changing the tension of the nerve in walking naturally.

Slight fever may accompany the onset of acute cases of sciatic neuritis; there is rarely any elevation of temperature when the condition is established.

Marked trophic or vasomotor disturbances occur in some severe cases. The most important of these are œdema of the leg and a herpetic eruption in the distribution of the nerve. Another and more serious complication is the ascent of the neuritic process along the sciatic to the lumbar plexus. The anterior crural nerve may be thus involved, causing pain in the front of the thigh and weakness and wasting of the quadriceps extensor.

Diagnosis. The diagnosis of sciatica is based on the presence of pain in the nerve or its distribution, and on tenderness of the nerve-trunk, and usually is not difficult. The distinction from sciatic neuralgia depends chiefly on the presence of marked and persistent tenderness of the nerve-trunk. The characteristics of sciatic neuralgia, which is actually a rare condition as compared with the frequency of sciatic neuritis, are as follows: (1) The pain is spontaneous from the start; movement does not itself cause pain; (2) the pain is apt to be referred to the branches rather than the trunk of the nerve; (3) tenderness of the trunk is slight or absent; (4) the patients are apt to suffer from neuralgia elsewhere. Diseases within the pelvis may give rise to severe sciatic pain—secondary sciatica. The pain in these cases is usually referred more to the branches than to the trunk of the nerve; there is also little trunk-tenderness in proportion to the pain. But the recognition of the nature of the pain may be difficult from the symptoms alone; a rectal examination, which should be made in every case of sciatica, is of great aid in determining the nature of the case. In sciatic neuritis there may be pain about the hip-joint (owing to the distribution of filaments from the sciatic nerve to the joint), but there is never any excuse for confounding such widely different conditions as hip-joint disease and sciatic neuritis. Sciatic neuritis is seldom double, hence double sciatica should direct suspicion to other disorders, especially disease of the nerve-roots or cord. The writer has known double sciatica to be due to extensive disease (sarcoma) of the bodies of the lumbar vertebræ, the first suspicion of the nature of the sciatica being excited by the appearance of lumbar abscess, first one-sided, later double. The sciatic pain of locomotor ataxia (which may be due to neuritis) is to be distinguished by its transitory character and its association with cardinal tabetic symptoms.

Prognosis. As regards the ultimate subsidence of pain and the restoration of the function of the nerve, the prognosis of primary sciatic neuritis is good in every case. The duration of the symptoms is extremely variable, lasting in some cases for a few weeks only, in others for many months or a year. Relapses are common in severe cases and may prolong the period of suffering to one or two years. In general the more acute and severe the symptoms the longer the duration of the period in which there is pain. Most cases last for several months. It is a good practical point to remember that in cases where the nerve-tenderness is such that the patient cannot get about, the duration of the trouble will be several months at least. The subsidence of the neuritic symptoms may be followed by obstinate neuralgia. Occasionally a neuritic process is set up in the nerve just as the patient appears to be making a recovery. These statements apply to primary neuritis; the cause and prognosis in secondary sciatic neuritis, of course, depend upon the character and position of the primary morbid process.

Treatment. The first condition of improvement in all cases of sciatic neuritis is rest for the affected limb, and it is important that this should be secured from the beginning. A week's thorough immobilization at the beginning of the trouble is probably more effective than a month's rest when the neuritic process has become established. Every position or movement of the leg that causes pain should be absolutely avoided. This element of rest in treatment is negative in character, but it is, nevertheless, of greater importance than any more positive measures. Just how the rest should be enforced varies according to the acuteness and severity of the case. In cases of mild degree it is not essential that the patient should go to bed; it suffices to keep the leg somewhat flexed, to avoid sitting on chairs with hard edges, and to walk little and only with crutches. When the nerve-tenderness is such as to prevent standing the patient should remain in bed with the leg extended upon a long splint after the manner recommended by Weir Mitchell, until the tenderness of the nerve has much subsided. This is probably the most satisfactory means of securing immobilization and gives excellent practical results.

The second cardinal indication in the treatment of sciatic neuritis is the relief of spontaneous pain. This may often be done at the commencement of a severe attack by the application of a hot poultice, or by the use of ice-bags over the course of the nerve for several hours daily. The use of cold is particularly well adapted for use in the cases where the long heel and axilla splint is employed. In the less severe cases counter-irritation by means of a mustard-plaster or a fly-blister may give much relief, at least temporarily. But the most effective form of counter-irritation is that obtained by means of the actual cautery applied lightly without blistering over the entire painful area. In chronic cases the cautery may be used to advantage to blister. The cautery is preferable to the ointments and liniments that are often employed. If the means of alleviating pain (and they probably also exert some influence upon the neuritic process) have been tried with little or temporary effect, it may be necessary to resort to hypodermatic medication. The hypodermatic injection of distilled water sometimes gives great relief, and should always be tried. If it fails, cocaine hydrochlorate should be injected, bearing in mind the precautions mentioned under brachial neuritis. Injections of morphine should be reserved for the last, and should then be used as sparingly as possible, and should be replaced by cocaine when practicable. In all cases where hypodermatic medication is employed for sciatic neuritis the injection should be made over the most painful spot, at a depth of about two inches, and never into the nerve itself. Antipyrin, antifebrin, and phenacetin should be avoided entirely, if possible. Simple acupuncture in the line of the nerve may give temporary relief, but is in itself a painful proceeding, and cannot be recommended.

When the neuritis is subsiding galvanism may be cautiously employed, but probably its chief value is the mental effect of its use upon the patient. If the muscles are atrophic or flabby, or if there is any contracture, massage properly used is exceedingly valuable. It may also do much to relieve pain.

Nerve-stretching has done good in some cases. It may be recommended where everything else has failed; but under these circumstances it too is likely to fail. It acts probably by (1) breaking up adhesions, (2) enforcing rest, (3) as a counter-irritant.

The possibility of doing good by constitutional treatment should never be lost sight of. The diet should be carefully studied and regulated. In rheumatic cases a largely meat diet should be used if the patient is not reduced in weight. Where no specific disorder of nutrition can be detected a tonic containing wine, quinine, and a little strychnine may be advantageously used for a considerable period of time. In the majority of cases of sciatic neuritis the treatment outlined is of material benefit; only in rare cases is treatment wholly unsatisfactory.

The *sacral plexus* is liable to suffer from various forms of pelvic disease, especially pelvic tumors and pelvic cellulitis. Rarely the plexus suffers compression during delivery, and occasionally it is the seat of neuritis in consequence of an extension of inflammation from the sciatic nerve. The plexus is said to be the occasional seat of a "primary" neuritis.

The symptoms of sciatic plexus disease are usually at first irritative in character (pain, paræsthesia, etc.); later they are indicative of a destruction (anæsthesia, paralysis, atrophy). The precise distribution of these symptoms naturally varies in different cases, since the various muscular and sensory distributions of the different elements of the plexus may be variously and very unequally affected. The manifestations of disease of the plexus are rarely confined to the distribution of any single nerve arising from it, although a single nerve (as the anterior crural) may for a time be the chief or exclusive seat of the symptoms. Plexus disease is almost invariably *unilateral*, but in rare cases of pressure-paralysis it is *bilateral*. In all cases where plexus disease is suspected a careful rectal exploration is of the utmost importance, and may reveal the presence of a pelvic tumor, an aneurism, or an abscess which exerts pressure on the sacral plexus.

Paralysis of the Sciatic Nerve causes symptoms which vary considerably with the seat of the lesion. Damage to the nerve is usually below the upper third of the thigh, and, if severe, gives rise to paralysis of all the muscles below the knee, and anæsthesia of the sole and outer side of the foot and the outer side of the leg. The gait in such cases is much like that observed in many cases of poliomyelitis in children. If the lesion is above the middle third of the thigh, the flexors of the knee and extensors of the hip are included in the paralysis. The sciatic nerve external to the pelvis may be damaged in wounds of the thigh, by disease of the femur, by adjacent tumors, and occasionally by dislocation of the hip. The nerve is often the seat of primary neuritis.

Paralysis of the External Popliteal (Peroneal) Nerve causes loss of power in the tibialis anticus, extensor longus digitorum, extensor brevis digitorum, and peronei, in consequence of which there is loss of flexion of the ankle and of extension in the first phalanges of the toes. The patient in such cases has "drop-foot," and in the course of time talipes equinus develops. In cases of severe damage to the nerve there is anæsthesia on the outer half of the front of the leg, and on the greater part of the back of the foot. The external popliteal nerve is very superficial in its course, and passes over the fibula. It is consequently exposed to all kinds of injury from wounds, from fracture of the fibula, from pressure, etc. It is also sometimes the seat of primary neuritis.

Paralysis of the Internal Popliteal Nerve, causes loss of power in the posterior tibial group of muscles (including the tibialis posticus and popliteus) and the long flexors of the toes, and in the muscles of the sole of the foot.

Extension of the ankle-joint is impossible, and if the branch to the popliteus is involved there is loss of inward rotation of the leg when it is flexed. When the damage to the nerve is sufficiently severe to cause anæsthesia there is loss of sensation over the outer part and posterior aspect of the lower part of the leg and on the sole of the foot. The posterior tibial nerve is rarely injured except in fractures of both bones of the leg.

The branches of the internal popliteal nerve, the external and internal plantar nerves, are rarely involved alone.

Paralysis of the External Plantar Nerve causes loss of power in the muscles of the little toe, the flexor accessorius, the interossei, the two outer lumbricales, and the adductor of the big toe. Certain of these muscles (lumbricales and interossei, abductor and flexor minimi digiti) flex the first phalanges, and extend the second and third, an action of much importance in walking in the propulsion of the body forward just before the foot leaves the ground. The loss of this action is a hindrance in walking, as is the later contracture of the opponents of the interossei, which leads to flexion of the second and third phalanges. The sensory loss in cases of external plantar paralysis includes the skin of the outer half of the sole of the foot and of the little toe, and that of the adjacent half of the fourth toe.

Paralysis of the Internal Plantar Nerve causes loss of power in the short flexor of the toes, the intrinsic muscles of the big toe (with the exception of the adductor), and of the inner lumbricales. It gives rise also to anæsthesia on the inner part of the sole of the foot and the plantar surface of the three inner toes and the adjacent half of the fourth toe.

Paralysis of the Small Sciatic Nerve causes paralysis of the gluteus maximus, with consequent interference with the power of rising from a seat and loss of sensibility in an area of variable size on the posterior surface of the thigh. The small sciatic nerve is damaged only in disease of the sacral plexus and is seldom the only nerve involved.

NEURALGIAS INVOLVING BRANCHES OF THE LUMBAR AND SACRAL PLEXUSES. The term *lumbo-abdominal neuralgia* has been applied to the neuralgic pains that affect the lower half of the trunk. The condition is analogous to intercostal neuralgia. The chief painful points are (1) an iliac point near the middle of the iliac crest, and (2) a hypogastric point at the lower part of the rectus. Sometimes in the male there is a scrotal point and in the female a labial point. The pain may be mistaken for a girdle pain when the affection is bilateral, but in the neuralgic affection the pain is changeable. Neuralgic pain in the side of the head of the penis is not very uncommon in neurasthenics, who excrete a large excess of uric acid. Such pain is also thought to result from masturbation. In women lumbo-abdominal neuralgia is sometimes due to pelvic disease, habitual constipation, or straining at stool. It is much rarer than myalgic affections of the same region.

Crural Neuralgia, in which the pain involves the anterior aspect of the thigh, is a rare form. It is usually secondary to sciatic neuritis or to a lesion of the lumbar plexus.

In *Plantar Neuralgia* the pain is limited to the plantar region. It is usually associated with paræsthesiæ of the same region, and appears to be due in some cases to slight neuritis. The pain of flat-foot must be borne in mind in this connection.

Metatarsal Neuralgia, or Morton's Affection of the Foot, is a not very uncommon disease of the foot which has usually been classed among the ill-defined hysterical or nervous disorders or among the manifestations of gout. The pain is located at the base of the fourth toe and may extend up the leg. It may be dull, throbbing, or lancinating. Usually it is spasmodic. Sometimes the severity of the pain interferes with walking for a few minutes.

The pain is usually not severe at night. It is increased by pressure over the head of the metatarsal bone. The trouble is often brought on by ill-fitting shoes. It occurs especially in women and frequently in those whose general health or nervous tone is somewhat impaired. The pain seems to be due to a neuritis of phalangeal branches from compression by the head of the fifth metatarsal bone. Pain of similar origin may occur at the base of the second toe. In cases of moderate severity and subacute course the pain may be relieved by avoiding all lateral pressure upon the toes. A specially constructed shoe with a broad sole must be worn and walking may be restricted for a time. In very severe intractable cases the excision of the head of the fourth metatarsal bone is necessary.

Prognosis and Treatment of Paralyses of the Spinal Nerves. It is convenient for reference to group together the chief practical facts relating to the prognosis and treatment of paralyses of the spinal nerves. In what follows the term paralysis is used to include disturbance or loss of the sensory as well as the motor functions of nerves.

Many elements enter into the prognosis of lesions of the spinal nerves, the degree of damage to the nerve-elements, the character of this damage, the presence or absence of infection, the age of the patient, the general health, and the duration of the paralysis. Where the paralysis is due to pressure which has been gradually exerted, as by an exostosis, or tumor, or cicatrix, or aneurism, the outlook depends on the ability to remove this cause of pressure. There is a fair prospect of improvement in function even where pressure sufficient to cause paralysis has lasted a year, if the pressure can be removed. In the paralysis that results from neuritis, the intensity and acuteness of the symptoms and the degree of degeneration as shown by the electrical changes are the chief guides to prognosis. If there is complete loss of power and entire loss of faradic irritability in the muscles at the end of two or three weeks, there will probably be little recovery of power for several months, and many months will certainly elapse before power begins to return if complete degeneration (as shown by the presence of complete R.D.) has occurred, however slowly. Whatever the origin of the neuritis that causes the paralysis, pain is apt to endure after the return of power, and this is especially true of persons in the second half of life or feeble vitality. Neuritis of infectious origin is particularly apt to cause persistent symptoms; the outlook is worse in suppurative neuritis. In traumatic neuritis the prognosis is probably better, as a rule, than in other forms causing the same degree of change in electrical irritability.

In cases of peripheral paralysis of acute development (acute neuritis, compression, partial section, or laceration) no opinion as to the outlook can be given until the lapse of one or two weeks if the paralysis be complete or considerable in degree. If the muscles have lost their irritability to faradism by the end of a week or ten days, considerable atrophy will follow and there will probably be little recovery in power until six or eight months have passed. If complete faradic loss occurs, but only after the lapse of two or three weeks or a month, there will be less atrophy and earlier recovery of power, but there will be paralysis for several months. Where there is merely a moderate diminution of faradic irritability or no loss whatever, recovery of power will begin in the course of a few weeks or months. The possibility of *some* recovery in power is not gone until there is complete loss of irritability to both faradism and galvanism in the muscles, and if there is no return of faradic irritability in the muscles at the end of a year, and their atrophy has been rapid and great, only slight improvement, at best, will occur. Some return of faradic irritability in the paralyzed muscles is usually to be looked for before recovery of power, but at times there is some motor recovery, while faradism is still incapable (in ordinary strength) of exciting contraction.

Occasionally the return of faradic irritability is not followed by any distinct improvement in motor power. Musculo-spiral pressure-paralyses are frequently of very short duration, with little or no change in the electrical irritability of the paretic muscles; recovery sufficient to enable a laboring man to return to his occupation often occurs in ten days or two weeks. A sluggish, labored contraction of the muscles to faradism or galvanism is always to be regarded as evidence of some degeneration in a nerve, but it must be remembered that in muscles (like the gluteus maximus) with coarse fibres there may normally be some sluggishness. When a nerve has been divided, with or without loss of substance, the prognosis depends largely upon the use of appropriate surgical treatment, although there are exceptional cases in which the nerve-ends have reunited spontaneously with recovery of function. The primary nerve-sutures should be employed whenever a nerve is completely or partially severed. By this means the conditions are rendered favorable for the rapid regeneration of the nerve-fibres. Union of the divided ends by first intention rarely occurs, but union in the course of a few days may be expected. A restoration of function occurs in a large majority of cases. Motion usually returns in cases of simple section, in the course of three or four months. Sensibility often returns much earlier, and may indeed be reestablished in the course of a few days, but this is exceptional and sometimes temporary. Slight permanent defects in sensibility are not rare and there may be some stiffness of movement. The return of touch, pain, and temperature sense makes a return of motor power very probable. In some cases the return of motion is very slow, but improvement may occur up to three or four years. The loss of a small amount of nerve-substance (say 1 c.cm.) makes little difference in the prognosis. The success of primary suture is considerably influenced by the age factor, the chances of recovery of function being far better in the young (especially children) than in those over fifty. Warm weather is also distinctly more favorable than cold. There seems to be a difference in the inherent powers of different nerves to functionate anew. Recovery is most rapid in the musculo-spiral, rapid, but less so, in the median, and comparatively slow in the ulnar, in which motion probably never returns before sensation (Bowlby). If primary suture fails, the nerve should be re-examined, freed from scars, and stretched. The secondary suture gives less satisfactory results than the primary suture, but its results are nevertheless surprisingly good. If a secondary suture is done after the lapse of two years, the best that can be hoped for is a partial restoration of function, but where the operation is done in the course of a year recovery is often complete and a considerable restoration of function can generally be counted upon. Motion may return at any time from six months to two years. Greatly atrophied muscles may regain their original bulk in the course of time. Sensation may return much earlier, but such early recovery may be temporary. The prognosis is rendered considerably worse in all cases of nerve-suture where the nerve wound has been infected.

In all cases of motor paralysis from nerve injury of any kind electricity should be employed. The affected muscles should be stroked for at least five minutes three times weekly with the negative electrode of a galvanic battery, the weakest current being used that will produce a fair contraction. There is no doubt that the proper use of galvanism hastens recovery from many cases of pressure-paralysis, and it is likely that it exerts a favorable influence in the recovery of paralysis of other origin. Massage is of much value in preventing the occurrence of contractures and in reducing those that have arisen. The immersion of the paralyzed muscles twice daily in warm water for ten or fifteen minutes also aids in preventing contractures. Careful attention to the general health probably influences, in some degree, the favorable progress of all forms of peripheral paralysis.

CHAPTER XXIX.

DISEASES OF THE MUSCLES.

By GEORGE W. JACOBY, M.D.

SUBACUTE PROGRESSIVE POLYMYOSITIS.

In marked contrast to the attention and study which have been bestowed upon the chronic forms of the affections localized primarily in the muscles, stands the neglect with which the acute and subacute disorders, particularly the inflammatory ones, have been treated. *A priori*, it should be supposed that inflammation of muscles (myositis) would occur as easily and as frequently as inflammation in other tissues, but as a matter fact the occurrence of such muscular inflammation was universally believed to be impossible, until Virchow proved the untenability of such an opinion.

Even to-day many observers do not admit the existence of a primary myositis. This is no doubt due to the rarity of its occurrence, so that while inflammation of muscles due to operative measures, to traumatisms of varied nature, or to propagation of the inflammatory process from neighboring parts is frequently encountered, a purely primary myositis is not often observed. Slight forms are apt to be overlooked and the severe forms are actually of so infrequent occurrence that when they are encountered a disagreement of opinion concerning the diagnosis and character of the affection is not surprising.

Primary acute or subacute polymyositis is a disease so rarely observed, that altogether, including several doubtful ones, only 21 cases have thus far been described. These cases all show the existence of an acute inflammation, rapidly and successfully affecting nearly all of the voluntary muscles, characterized chiefly by pain and swelling, and ending in death in more or less short period of time, or exceptionally in recovery after a prolonged period of illness.

As representative of a typical myositis we may take the inflammation occurring in the course of trichinosis, and although this myositis is a secondary one, due to the inflammatory reaction produced by the trichina, it cannot be a question of doubt that many other noxious influences may produce such results, and that clinically they can be differentiated from one another only with difficulty. Hence, every primary polymyositis, due to whatsoever cause, will necessarily present clinical symptoms closely analogous to those encountered in trichinosis.

Of the etiology of primary polymyositis we know very little. Age and sex seem to be unimportant factors in the production of the disease, although of the published cases two-thirds occurred in males. The affection may occur at all ages. No cases have been observed in childhood; the youngest patient afflicted was seventeen years, and the oldest seventy years of age. Of the cases here referred to, 2 occurred in patients under 20, 5 in the second decade of life, 4 in the third, 2 in the fourth, 3 in the fifth, 3 in the sixth, and in 2 cases the age is not given.

Occupation, or the influence of cold and muscular over-exertion do not seem to be etiologically important. Even of those influences which are active in the production of multiple neuritis, with which this disease may be combined, particularly alcohol, the same may be said. Strümpell has paid particular attention to the possible influence of tuberculosis as a causal factor without arriving at any definite results. In three autopsies a tubercular process of one pulmonary apex was found, but it is probable that this was accidental and not causative.

Symptomatology and Course. The onset of the disease is usually a gradual one, characterized by general disorder. Occasionally the trouble begins acutely without any prodromal symptoms. These symptoms when they are present consist in a feeling of malaise and fatigue; the appetite is disordered, and dizziness and headache are complained of. Gastric symptoms, nausea and vomiting, have been noted in a few cases, but in no case was the advent of the disease heralded by a chill and sudden rise in temperature as is the case in acute infections.

Together with these general symptoms or within a few days, the local symptoms which characterize the disease make their appearance. These at first are purely subjective, and consist in pains in the extremities, back, and loins. These pains as they increase in severity are accompanied by complaints of tension and cramps in the muscles. Objective symptoms soon manifest themselves, and locomotion becomes more and more difficult; finally the patient is obliged to take to bed where a condition of utter helplessness speedily supervenes.

The muscles become affected in a certain sequence, those of the extremities being first involved and at a later period those of the trunk. Mobility is impeded in accordance with the distribution of the muscular affection. Of the extremities, the more distal parts retain their mobility longer than the proximal ones. After the trunk muscles the abdominal muscles and those of the neck become implicated.

About this time the intercostals are attacked, with the effect of rendering respiration markedly diaphragmatic; finally the muscles of deglutition become involved and death is generally due to combined disease of these and the respiratory muscles. In the terminal stage of severe cases the muscles of the head and even those of the tongue and pharynx are affected. Speech thus becomes indistinct and there is difficulty in protruding the tongue or even in raising it.

The subjective symptoms in a developed case, therefore, are chiefly pain and disorder of voluntary motion, but an examination at once demonstrates the presence of other symptoms. Most marked and at once noticed is the painfulness of the affected muscles to pressure or to manipulations of any kind.

Inspection shows that the limbs have lost their marked contours, so that they seem to consist of a single mass, the boundaries of the individual muscles being obliterated. These also are swollen and increased in size, but this swelling is lost amid the general œdematous infiltration of the skin and other soft tissues. To palpation the muscles show a different consistency in different parts and at different times, being hard and distended, soft and doughy, and even simulating fluctuation. This œdematous swelling of the skin, which does not pit upon pressure, and is certainly also of an inflammatory nature, seems to follow the affected muscles as to time and place of its appearance, there always being more œdema over those parts in which the muscles are most affected. Thus also the skin of the proximal parts of the extremities shows more œdema than that of the distal parts, and the hands and feet are usually free. The face, while it may show slight œdema generally, is not thus affected. Later, an approximation of the attachment-ends of the muscles takes place, so that the tendons stand out hard and unyielding, and persistent flexion of the leg upon the thigh and of the forearm upon the upper arm may result. In many cases atrophy of certain muscles sets in during the progress of the case. Thus the vasti, the deltoids, the trapezii, and the small muscles of the hand were markedly atrophied in a case described by me.

The œdematous skin, wherever it is seen, at some stage of the disease shows a marked and peculiar redness which seems to be characteristic of polymyositis. The nearest approach to a description of this redness is to say that it resembles that seen in erysipelas, so that on account of the accompanying œdema it may easily be mistaken for this disease. Sometimes the redness is more erythematous in nature, and it is probable that in some cases the skin affection is still different from that just described, for it has been spoken of as "spotted redness," roseola-like eruption, urticaria-like, etc.

In addition to this eruption, subcutaneous capillary hemorrhages along the borders of the disordered territories are of not unusual occurrence.

It has been claimed that the affection may take an abortive course, simulating purpura. It seems to me to be doubtful whether such is ever the fact, and I am not prepared to accept the case upon which this statement was based as a genuine one. A tendency to profuse perspiration is also noticeable, and is to a certain extent characteristic, if not of polymyositis, at any rate of a severe acute affection of the muscles.

Nervous symptoms of a marked kind, such as we would expect to find on account of the clinical relationship between polymyositis and polyneuritis, are not present.

In consequence of the great tenderness of the muscles, it is difficult to decide whether the nerves are painful to pressure or not. I have paid particular attention to this point, and am sure that the nerve-trunks are not any more sensitive than is normally the case. Disorders of sensation of any decided character are not present in the pure uncomplicated cases. Mechanical excitability of the affected muscles is certainly not increased, perhaps even diminished. Fibrillary twitchings do not occur.

The superficial reflexes are all present. The condition of the deep reflexes will depend upon the greater or less affection of the muscles, so that the tendon reflexes, which in the beginning of the disease are normal, will disappear as the disorder progresses. Hence, in one and the same case we may at different stages of the disease find the tendon reflexes normal, diminished, or absent. There is no valid reason why they should ever be increased.

On account of the pain produced by all manipulations it is difficult to make a careful electrical examination, but it is fairly certain that the electrical reactions correspond to what we would *à priori* assume to be the case, which is that the muscles gradually show diminution and finally abolition of electrical excitability. It is probable that in the early stages of the disease qualitative changes will also be found.

Brain symptoms are not present, and consciousness is retained throughout. The headache, dizziness, and insomnia which are present are due to the same causes which produce such symptoms in other acute febrile diseases. Of the other organs the spleen is markedly implicated, and in cases taking an acute course it will be found distinctly swollen.

As pure complications, but having great import upon the course of the disease, we must regard the bronchitis and pneumonia, which frequently supervene. The paralysis of the soft palate and pharyngeal muscles makes it easy for mucus and particles of food to be aspirated into the larynx and bronchioles. Thus we find that bronchitis and broncho-pneumonia constitute the direct cause of death in the majority of cases. The temperature, as well as the pulse, is increased in all instances, independently of any complication. The *course* of the disease varies greatly, and in considering this we must be careful to separate the acute from the chronic cases, and those which end fatally from the lighter cases, which may end in recovery.

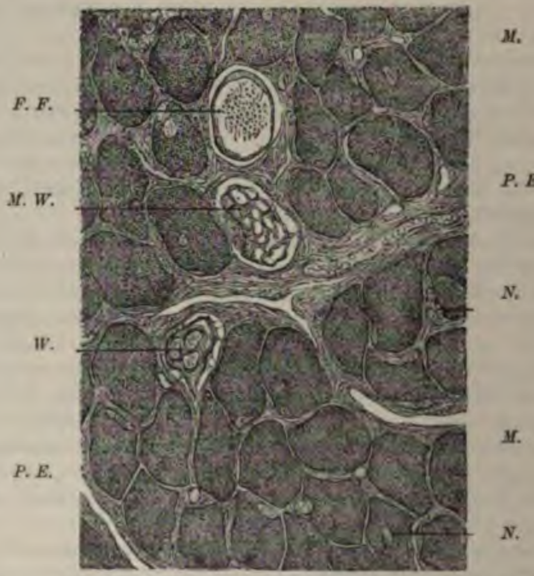
The duration in the fatal cases has averaged from six weeks to three months. The longest period after which death has occurred has been sixteen months. Of the cases which ended in recovery, one lasted only seven days and one for two and a quarter years. In the subacute and more chronic cases the course is not one of constant progress, but remissions occur which are apt to deceive the observer as to the further progress of the disease.

Pathology and Morbid Anatomy. Upon post-mortem examination the diseased muscles seem swollen in toto, and show a distinctly spotted appearance, pale gray spots alternating with dark red places. The muscle is often very friable and easily torn.

Microscopical examination shows that marked changes have taken place in the muscular fibres and also in the interstitial connective tissue. These changes are the anatomical expression of destruction of the fibres. The changes in the interstitial connective tissue are marked, and plainly show the acute inflammatory nature of the process.

The perimysium is augmented, has become myxomatous or fibrous in structure, contains fat globules, and in single parts shows waxy degeneration.

FIG. 279.

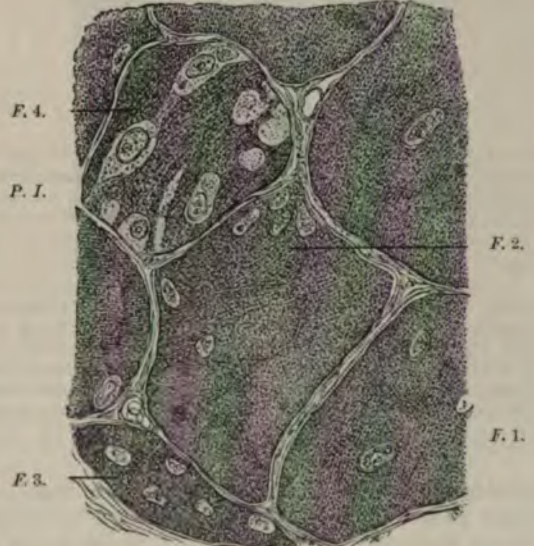


Transverse section. × 200. *M. M.* Muscle fibres of average size in partly transverse, partly slightly oblique sections. *P. E. P. E.* Perimysium externum, with numerous capillaries. *N. N.* Nuclei, some in centre, some at periphery of muscle. *W.* Muscle fibre in waxy degeneration, holding three protoplasmic bodies. *M. W.* Cluster of medullary corpuscles in waxy degeneration. *F. F.* Muscle fibre in waxy, possibly combined with fatty degeneration.

Not all the muscle fibres of a single bundle, but only a limited number, are affected. These affected fibres are either transformed into an apparently homogeneous or into a waxy mass. Partly homogeneous and partly waxy fibres are not uncommon. The nuclei are markedly increased in number, not only at the periphery, but are also found scattered throughout the body of the muscle; indeed, there may be so great an increase of nuclei that the fibres assume the aspect of a so-called giant cell, the regular arrangement of the sarcous elements being lost and the granulations having become irregular. In this case the sarcolemma sheath is still present, and the boundary of each muscle fibre is clearly defined; in other parts can be seen how the muscle fibre breaks up into a number of indifferent corpuscles, losing its distinct boundary and blending with the perimysium, so that no line of demarcation can be drawn between the two. We can clearly see how the original muscle tissue loses its specific structure and becomes transformed into connective tissue. The muscle fibre is not from the start affected in its entirety by the myositic process, but the various parts are attacked progressively. This is shown by the presence of muscular fibres, which in part are unchanged, but in part are transformed into a coarsely granular mass, the sarcolemma still being present. All in all, then, we clearly have a hyperplastic perimyositis, together with a parenchymatous myositis.

The terminations of this myositic process are either, as already indicated, transformation of the contractile matter into connective tissue or fatty and waxy degeneration. The latter termination is undoubtedly the most common. All the changes which the muscles undergo may be clearly followed upon the accompanying cuts.

FIG. 280.

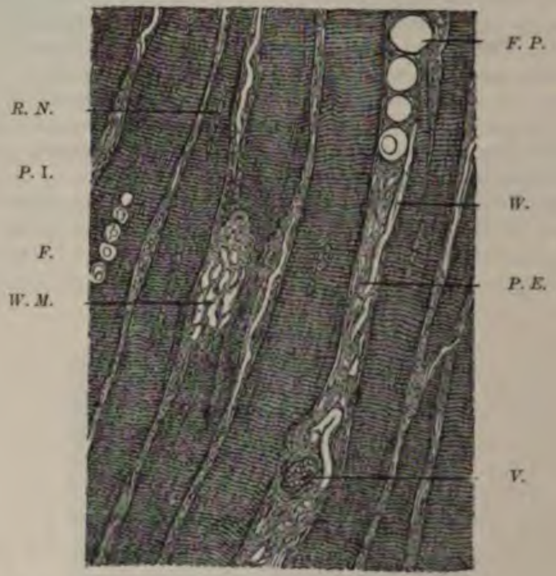


Transverse section. × 800. *F.* 1. Muscle fibre, with central nuclei. *F.* 2. Muscle fibre, with augmented peripheral nuclei, showing indication of breaking up into muscle plates. *F.* 3. Muscle fibre, holding five central nuclei, each one corresponding to a muscle plate, the whole resembling a myeloplax; the sarcous elements enlarged and crowded. *F.* 4. Muscle fibre transformed to a great extent into partly nucleated, partly non-nucleated protoplasm. *P. I.* Perimysium internum almost unchanged.

The nerve fibres in all specimens which I have examined were also the seat of an inflammatory process, but the large number of normal fibres present convinces me that the process in the nerves is entirely secondary to that in the muscles. Brain, spinal cord, and peripheral nerve trunks are normal. Microscopically, also, it has been noted that even those muscles appear diseased which, macroscopically, seem to be normal, thus corroborating the clinical symptoms which point to an involvement of the entire muscular system.

As regards the pathogeny of the disease we know very little; still it seems probable that the trouble is an infectious one, and that, as Strümpell has suggested, the action of this infection is not specifically local (muscular), but that by its agency poisons have been introduced into the circulation and thus brought into contact with all the tissues, of which in this case the muscular ones are alone susceptible to its noxious influence. Whether these cases bear any relationship to the chronic forms of primary progressive muscular atrophy is a question which cannot yet be answered. It seems to me quite possible that some cases of primary dystrophy may be the outcome of light cases of polymyositis. If this is so, then we would not be wrong in looking upon these cases as closely allied, pathogenetically, to cases of primary muscular atrophy.

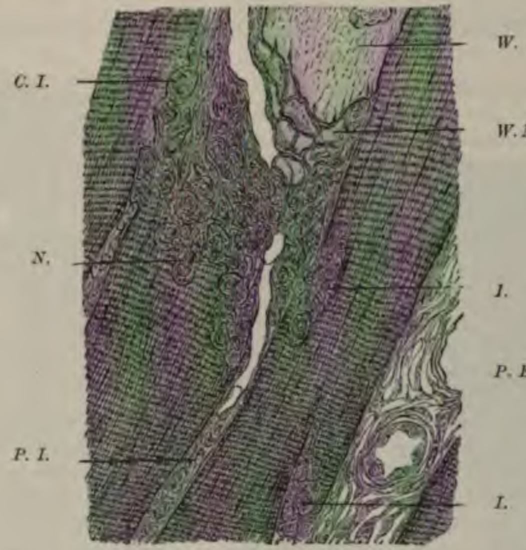
FIG. 281.



Longitudinal section, × 200. *P. E.* Perimysium externum, considerably augmented, composed of coarse bundles of fibrous connective tissue. *V.* Vein, engerged with blood. *F. P.* Fat globules in external perimysium. *P. I.* Perimysium internum, slightly augmented. *R. N.* Rows of nuclei. *F.* Row of fat globules (?) in centre of muscle fibre. *W. M.* Clusters of medullary corpuscles in waxy degeneration. In the vicinity of this cluster the gradual transformation of the muscle tissue into inflammatory corpuscles and the consequent destruction of the muscle tissue is marked. *W.* Peripheral portion of the muscle fibre in marked waxy degeneration.

Diagnosis. The diagnosis, bearing in mind the marked clinical symptoms, should not be a difficult one. The main difficulties will arise in making a differential diagnosis between polymyositis and trichinosis, but the marked gastric and intestinal troubles, the early occurrence of facial œdema and of pains in the region of the ocular and laryngeal muscles, as well as the implication of the muscles of mastication, should direct our attention to trichinosis rather than to polymyositis. The exposure to trichinosis invasion is, of course, most important.

FIG. 282.



Longitudinal section. × 800. *P. E.* Perimysium externum, broadened, composed of coarse bundles of fibrous connective tissue freely vascularized. *P. I.* Perimysium internum, transformed into inflammatory or medullary corpuscles. *I. I.* Groups of inflammatory corpuscles, obviously arisen from previous contractile tissue. *N.* Nucleated inflammatory corpuscles imbedded in muscle tissue. *C. I.* Clusters of inflammatory corpuscles in bay-like excavations of the contractile tissue, in part spindle-shaped, in transition to fibrous connective tissue; the medullary tissue traversed by a large, probably newly-formed capillary bloodvessel.

Against a multiple neuritis and in favor of myositis we must regard the localization of pain to the muscles themselves, the occurrence of marked œdema, and the implication of the skin in the inflammatory process, together with the absence of extended disturbances of sensation.

It must not be forgotten that in some cases an association of the two affections may occur.

Prognosis. The prognosis of the affection is a very dubious one; of the twenty-one published cases, including those in which the diagnosis is doubtful, only nine ended in recovery. Recovery, when it occurs, takes place gradually, and is very much retarded. In only one of the non-fatal cases were the muscles of deglutition and respiration affected, while in nearly all of the fatal ones this was the case. It is thus clear that such a complication is one which renders the prognosis absolutely bad.

Treatment. Treatment of all kinds does not appear to exercise any influence upon the course of the disease. Medication will, therefore, necessarily be restricted to an alleviation of symptoms. Salicylate of sodium, colchicum, and antipyrin may be tried early in the disease and some relief from pain and general malaise obtained. When the pains are most severe morphine seems to be the only remedy which will alleviate the sufferings of the patient.

Prolonged hot baths, or the envelopment of the limbs in hot cloths, are gratefully received by the patient, up to such a time when every movement is avoided on account of the pain thereby produced.

All mechanical remedies, as massage and electricity, are absolutely contraindicated while symptoms of progression are present.

THE PROGRESSIVE MUSCULAR DYSTROPHIES.

The above designation has been given to progressive muscular atrophy not due to disease of the spinal cord or peripheral nerves. The disease is located exclusively in the muscles themselves, and leads to marked atrophy and corresponding impairment of function. To Erb distinctly belongs the credit of creating a clear division between the spinal and myopathic forms of progressive muscular atrophy.

The clinical distinctions between these two forms are mainly that in the myopathic forms young persons, often children, are the subjects, and that several members of the same family are frequently attacked, while in the spinal forms the reverse is the case.

It seems probable that the affection is a congenital one, that is to say, that it is due to the subsequent development of a faulty embryonal disposition of the muscular tissue.

In some cases this faulty disposition shows itself by a gradual disappearance of the muscular fibres, due to a simple defect in their growth, in others there is in addition to this an increase of connective tissue with or without the formation of fat. All of these cases show loss of muscular power corresponding to the disappearance of the muscle fibres; but objectively the cases will vary according to the presence or absence of hyperplasic connective tissue and according to the localization of the atrophy.

Based upon such differences, a number of so-called types have been described. While it is certain that all of the forms actually constitute a unity, it is well for convenience of description to retain certain characteristic types. These are:

1. Pseudo-muscular hypertrophy.
2. Erb's juvenile type.
3. Landouzy-Déjérine type.
4. The peroneal or leg type, which has been classed with the dystrophies, for convenience' sake, but is probably of neurotic origin.

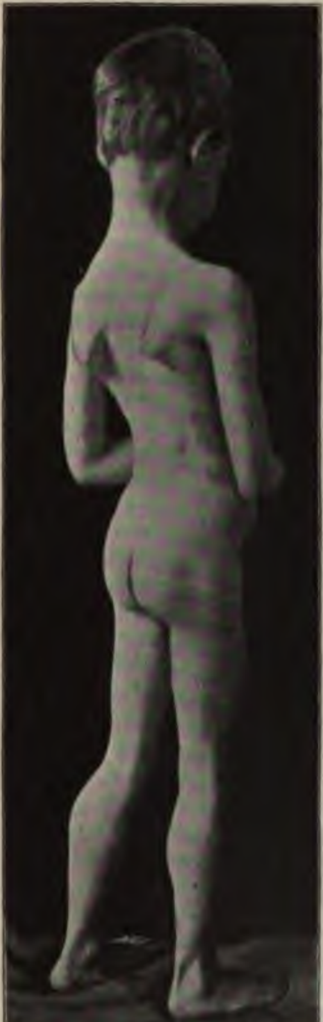
The clinical entity of the various forms is shown by a more or less complete correspondence of the majority of prominent characteristics, by the fact that transitional forms from one type to another are noted, as when pseudo-hypertrophy is found in other forms than the pseudo-hypertrophic type, or when the affection takes the course of the pseudo-hypertrophic form without hypertrophy being present, and by the fact that various types occur in different children of one and the same family. The occurrence of indefinite cases which cannot be classed in any of the known forms also goes far to support this view.

Pseudo-muscular Hypertrophy (atrophia musculorum lipomatosus). The affection in this class of cases begins in childhood, is often present in several members of the same family, and is characterized by progressive disorder of function associated with an increase in size of single parts of the body, due to an interstitial deposit of fat which obscures the existing atrophy of the muscular fibres.

Symptoms. The disease manifests itself gradually, and the first abnormality noted by the parents in the hitherto apparently healthy child is weakness of the legs, particularly manifested in running, jumping, or going upstairs. The child stumbles easily, and falls frequently.

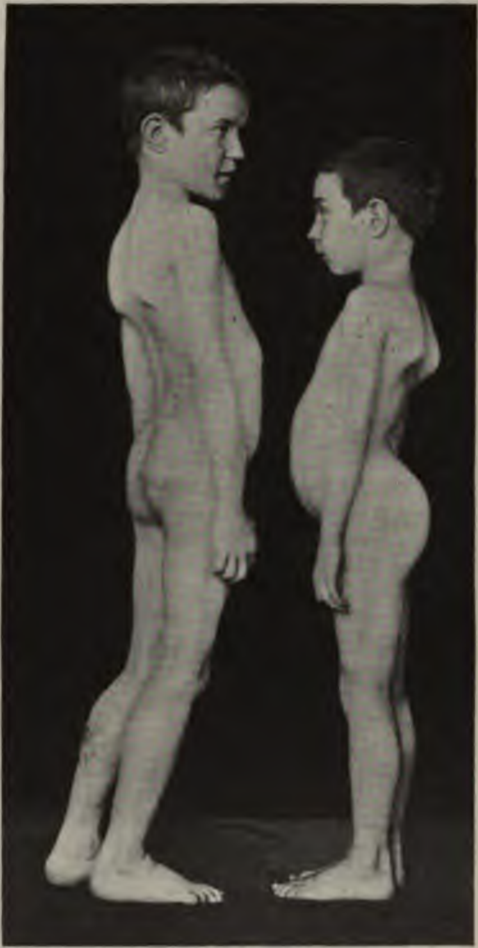
Somewhat later an enlargement of certain muscles is observed, that is to say, the muscles appear large in comparison with other muscles of the part. The muscles of the calf are usually the first to become hypertrophied, the extensors of the knee and the muscles of the back and loins are also affected early. Of the muscles of the upper part of the body, the infra-spinati, are earliest and most markedly enlarged. The supra-spinatus and the deltoid are usually increased in size, but the serratus rarely, and the pectoralis never. In the arm the triceps and biceps may be either enlarged or

FIG. 283.



Typical pseudo-muscular hypertrophy.

FIG. 284.



Pseudo-muscular hypertrophy in brothers. (Infirmary for Nervous Diseases, Philadelphia.)

wasted, but the muscles of the forearm and the small muscles of the hand are, as a rule, unaffected. Together with this enlargement a wasting of certain groups of muscles takes places, and, after a time, the originally hypertrophied muscles also become atrophied.

Weakness of the muscles goes hand-in-hand with their involvement, but it is not always the most wasted muscle which is the weakest. In the legs the weakest muscles are usually the flexors and extensors of the hip, and the extensors of the knee. The calf muscles generally give out before the anterior tibials. Corresponding to the involvement of these muscles of the legs there is limitation of movements which leaves its impress by characteristic symptoms. On account of weakness of the extensors the patients have great difficulty in going upstairs, so that they make use of the guard-rail to pull themselves from one step to another. The gait has a peculiar waddling character, and the power of getting up unaided from the floor becomes lost. The manner in which such patients arise from the floor is, as has been pointed out by Gowers, very characteristic. The child, if not totally unable to rise, does so by supporting himself upon all fours, stretching out his legs, and then with his hands climbing up his own thighs.

Contractions occur late in the affection in those muscles which, through weakness of their opponents, habitually overact. In this way is produced a talipes equinus and permanent flexion of the legs upon the thighs, the thigh upon the hip, and of the forearms upon the arms. Curvature of the spine, antero-posteriorly with the concavity backward (lordosis), is also one of the early symptoms of the disease. This lordosis, which is due to a weakness of the extensors of the hip, may be so marked that a vertical line drawn from the scapula falls far behind the sacrum. Upon sitting; this curvature disappears and is often replaced by a curvature with the concavity forward. Lateral curvature, when present, is due to weakness of the spinal muscles.

The electric excitability of the muscles is quantitatively reduced to both currents in proportion to the wasting of their fibres. Qualitative changes are not present. Fibrillary twitchings do not occur. Sensation is unimpaired and the reflexes remain normal until such a time when the reflex arc is interrupted by the muscular disease.

The central nervous system is not involved.

The course of the disease is a chronic one, but its progress and duration vary greatly. Until power of walking becomes lost the progress of the disease is usually slow, but after that an increase seems to take place. Death occurs in from ten to twenty-five years, and is usually due to some complication, the respiratory organs becoming affected in consequence of the lessened action of the respiratory muscles.

ERB'S JUVENILE OR SCAPULO-HUMERAL FORM. The juvenile form of Erb is a chronic progressive atrophy and weakness of numerous voluntary muscles, beginning in childhood and early youth, somewhat later than in pseudo-hypertrophy, and frequently occurring as a family disease. It is characterized by the early and marked selection of the muscular affection for the upper part of the body. The pectoral, the trapezius, the latissimus, the rhomboids and other shoulder muscles are first affected, and later the muscles of the arm become attacked. Here it is the flexor group of the upper arm which is first involved, then the triceps becomes affected, but the muscles of the forearm and hand are not implicated. To this rule the supinator longus, which is usually affected together with the muscles of the upper arm, forms an exception.

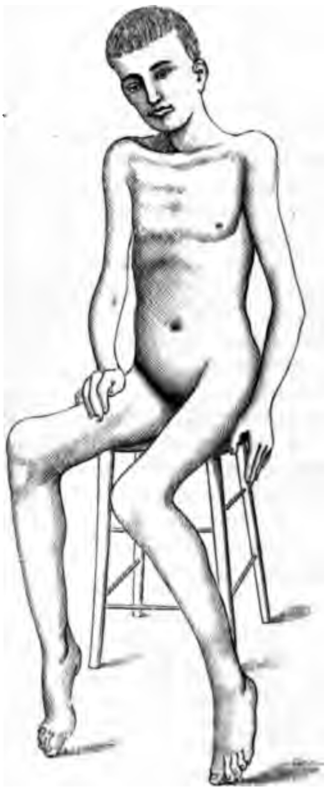
In the lower half of the body the lumbar muscles, part of the abdominal muscles, the glutei, the thigh muscles to a great extent, and, ultimately, the calf muscles and part of the peronei become affected.

While, as a rule, wasting of the muscles is present from the first, such is not always the case. Some muscles, and in all instances always the same muscles, exceptionally show a true or false hypertrophy. These enlarged muscles are the deltoid, the infra-spinatus, the sartorius, and the gastrocnemius. While the changes may for a long time be confined to the muscles here mentioned, ultimately the entire muscular system becomes involved.

The changes produce peculiarities in the appearance of the body which are very characteristic. The abnormal position of the scapula, the lumbar lordosis, and particularly the thinness of the upper arms and thighs, in distinction to the well-developed forearms and the hypertrophic legs, are peculiar to this form of the disease.

The relationship between pseudo-hypertrophy and juvenile atrophy is so close that in many cases the line of demarcation between the two is not clearly defined.

FIG. 285.



Erb's juvenile or scapulo-humeral form of muscular atrophy. (MARIE and GUINON.)

THE LANDOUZY-DÉJÉRINE OR FACIO-SCAPULO-HUMERAL TYPE. This form, which was known to Cruveilhier and was described by Duchenne under the name of infantile progressive muscular atrophy, was in 1884 for the first time correctly recognized as a myopahtic affection by Landouzy and Déjérine.

The muscular affection here begins in early childhood, but may occasionally develop late.

The characteristic feature of this form is that the face is first and markedly attacked. The affection of the face, consisting in a mask-like smoothing and immobility, gives rise to a peculiar appearance known as the "facies myopathique." The orbicularis oris becomes very weak, so that the lips are habitually separated and protruded. This symptom is known as the "tapir mouth." The articulation of labials becomes indistinct and the ability to whistle is lost. The eye muscles and those of mastication and deglutition remain unaffected. The muscles of the rest of the body become affected later and in the manner described in the preceding form.

The electric excitability of the muscles in both of these forms is decreased in direct proportion to the amount of actual atrophy of the muscle fibres. Reaction of degeneration does not occur, and fibrillary twitchings are usually but not always absent. Mechanical excitability of the affected muscle is generally decreased or lost. Sensibility is normal, and symptoms of disease of the brain or spinal cord are not present. Deformities of the body, due to the shortening of the less affected muscles, take place as the disease progresses. All kinds of transitions occur between this form and the preceding one. Cases of juvenile atrophy, with late involvement of the face, are not very rare, so that the main difference between this form and the juvenile one is to be sought in the primary or secondary facial involvement.

Etiology. Of the actual causes which produce pseudo-hypertrophy we know nothing. Boys are affected much more frequently than girls, and as the disease often occurs in several members of one family, it is not unusual to find the boys of the family affected, while the girls are exempt. When heredity can be traced, and this can be done in one-half the cases, the hereditary influence emanates from the mother's side. Indirect influences, such as neuropathic constitution, syphilis, alcoholism, consanguinity, do not appear to have any influence in the causation of the diseases, although in such families occasionally a history of nervous disease, as hysteria, epilepsy, idiocy, etc., is obtainable.

The age at which the disease manifests itself is usually that of early childhood, so that in three-fourths of the cases the symptoms become apparent before the tenth year. Beyond the congenital hereditary tendency common to all forms of dystrophies, we know nothing of the producing causes of the juvenile or Landouzy-Déjérine types. Both sexes suffer about equally, so that, in affected families there is not, as in pseudo-hypertrophy, affection of one sex to the exclusion of the other. The disease may manifest itself very early, but as a rule the onset takes place in late childhood and the beginning of adult life. The facial type seems to begin at an earlier age than the shoulder type.

The action of any direct exciting cause in the development of the disease is unknown, and while the affection has become manifest soon after an attack of acute disease, or exposure to cold, it is unwarrantable to assume any causal connection between the two.

Pathological Anatomy. In all forms of primary dystrophies there is a correspondence of the main anatomical changes. The differences encountered are rather those of extent than of character and may be noted in different muscles of the same individual to the same degree as in muscles of different individuals, and vary as much in different cases of the same form as in cases of different types. Hence it is not possible from a microscopical examination of a specimen of muscle to say from which form it has been taken.

The changes found may be divided into two classes, those of the muscle fibres and those of the connective tissue. The principal changes are those of the muscular fibre, and those which consist of changes in volume; hypertrophy and atrophy must be considered the primary and important ones.

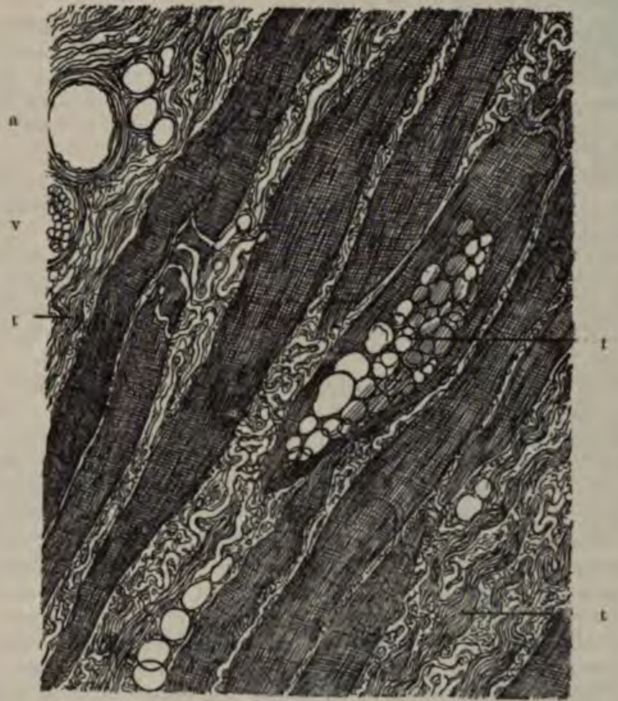
This is at variance with the old view, which is still held by some observers, that the change in the connective tissue is the primary. It is now certain that the muscular fibres are first affected and the connective tissue secondarily, or that possibly they both become affected together.

Hypertrophic fibres may be found in nearly all specimens taken from early stages, but their number varies from a single one here and there to a great many in each transverse section.

These hypertrophic fibres are thought to be characteristic of primary myopathies in contradistinction to the spinal atrophies. The atrophy also varies quantitatively and qualitatively, only single atrophic fibres being found or all muscular fibres having disappeared. The fibres become rounded by losing their sharp corners.

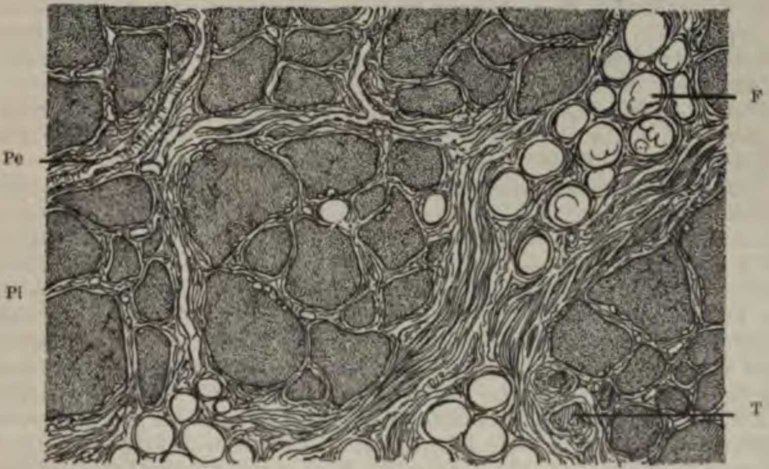
An increase of muscle nuclei is always noticed, those of the periphery being augmented in number, and nuclei appearing in the centre of the muscles. Here and there long rows of nuclei are present. Constant also is the fissuring and fibrillation of the muscles. Vacuoles also are encountered at times, but their formation is not so constant nor so characteristic as the

FIG. 286.



Quadriceps femoris from pseudo-hypertrophy. × 300. Longitudinal section: a, artery; v, vein t t, tendon-like formation of fibrous connective tissue; f, fat globules.

FIG 287.



Quadriceps femoris from pseudo-hypertrophic muscle, × 300. Transverse section: Pe, Pi, perimysium externum and internum T, tendon-like formation; F, fat globules.

changes just noted. The transverse striation becomes indistinct; fatty or waxy degeneration in the fibres is rare.

The connective tissue changes consist in a gradual proliferation, with an increase of nuclei, an increase and thickening of the vessels and a deposit of fat. These changes may vary in different muscles so that all degrees and combinations of muscle fibre, connective tissue and fat may be seen. The increase in connective tissue stands in inverse proportion to the changes in the muscular fibres.

The above changes have been noted chiefly in pieces of muscle excised during life. Autopsies have been made in a number of cases and muscles thus obtained have shown the same changes.

The motor nerves, the anterior nerve-roots, and central nervous system have, with but single exceptions, been found normal. In these exceptional cases, changes of a pathological nature were found in the gray anterior horns or deviations from the normal were observed which were so slight as to be unimportant.

That any of these changes have had a direct bearing upon the production of the dystrophy is doubtful.

Pathologically it would, considering the symmetrical distribution of the atrophy, seem that we were dealing with a disorder of the trophic centres which preside over muscular nutrition, but the changes found all point to the existence of a primary muscular affection. The spinal changes which have been observed in a few cases were so different from those found in true spinal atrophy that for the present, at any rate, they may be disregarded; at the same time, on account of the insufficiency of our methods of investigation, we cannot be absolutely certain that the trophic centres are not involved. Erb, Knoll, Moebius, Liebermeister, and others are adherents to this neurotic theory of production, believing the trouble to be a trophoneurosis; the great majority of investigators, however, consider the affection to be primarily of muscular origin.

Diagnosis. The diagnosis of progressive muscular dystrophy is not difficult if the essential features of the entire group and the special characteristics of the single forms are borne in mind. In pseudo-hypertrophy the waddling gait, the manner in which the children arise from the floor and climb stairs, the enlargement of the calf muscles, often together with contracture, the hypertrophy of other muscles in connection with wasting of neighboring ones, are all symptoms which render a mistake between pseudo-hypertrophy and any other disease impossible.

In all the forms. the affection of several members of a family, the onset of the disease in youth, the distribution of the atrophy to certain parts of the body are almost sufficient to correctly diagnosticate the disease.

Differentially the affections must, above all, be separated from the spinal form of muscular atrophy. If we remember that the latter rarely shows any heredity, that the onset is usually late in life after adult age has been reached, that it usually commences in the small muscles of the hand, progresses to the forearm, upper arm, and body, affecting the lower extremities only very late, that hypertrophy is never present, and fibrillation nearly always, and that the atrophied muscles show reaction of degeneration, then muscular and spinal atrophies will only rarely be confounded. The affection of the facial muscles in the dystrophic form can hardly be mistaken for the bulbar paralysis often met with in the spinal disorder. The absence of pains and sensory disorders, the absence of reaction of degeneration, and the presence of hypertrophy will serve to differentiate the dystrophies from chronic multiple neuritis.

The differentiation of one type from another is rather a question of classification than of diagnosis.

The distinction from the peroneal type will be spoken of later on.

Prognosis. Inasmuch as the disease is progressive in nature, the prognosis must be a serious one. In cases in which the disease develops late, the progress of the affection is slower, and the duration may extend over very many years. As a rule, patients afflicted with the pseudo-hypertrophic forms do not live beyond the middle of the second decade.

In all forms as the affection progresses, more and more muscles become involved until finally, after the patients have become helpless, the respiratory muscles are attacked, the diaphragm becomes involved, and death occurs through asphyxia or some intercurrent disease.

Treatment. As we would, from the developmental nature of the affection, expect, medicinal, and mechanical agents of all kinds have failed to prevent the progress of the disease. At the same time, if anything is to be accomplished therapeutically it must be done in the direction of stimulating the growth of the muscular fibres. For this purpose we may use electricity, massage and gymnastics, and it is fairly certain that while no case has been cured by these agents, the two latter, at any rate decrease the tendency to contractures, and to an extent retard the progress of the disease. The question of preventive treatment is also one which must be mentioned. While it is hardly rational to endeavor in anyway to medically influence the parents of dystrophic children, and thus prevent the appearance of the disease in future offspring, we should, however, counsel against the marriage of even the healthy women belonging to such families, and thus avoid the possibility of transmitting the family taint.

PROGRESSIVE NEUROTIC ATROPHY.

This form of atrophy, which was first recognized by Charcot and Marie in 1886, and shortly afterward described by Tooth under the name of the "peroneal type" has been most carefully studied by Hoffmann in Germany and Sachs in this country.

The affection occupies, so to say, an intermediary position between progressive muscular atrophy of spinal origin and the progressive muscular dystrophies, and shows marked differences from these well-known forms of muscular atrophy.

Etiology. All that is known of the etiology of this form is that most cases are developed upon a family basis, so that several members of a family become affected. Occasionally a history of the affection in the ascendants has been obtained. Males seem to be affected more frequently than females, but these are not by any mean exempt. In some families the male members only are affected. The onset of the affection may take place during infancy, in childhood, and also in later life. The usual period of development is before the twentieth year.

Symptoms and Course. The marked peculiarity in the symptomatology of this form is that the distal parts of the extremities are affected first, and that it usually commences in the lower extremities, while, therefore, the ordinary form is the leg-type or peroneal form, the commencement occasionally takes place in the upper extremities, or all four are affected simultaneously. The small muscles of the foot are usually first attacked, but the early involvement of these muscles is very apt to be overlooked; when, therefore, the affection of the foot is observed at a later period, it is generally erroneously assumed that the disease started in some other part of the body. After the foot muscles the peronei, the extensor communis digitorum, the tibialis anticus, and the calf muscles become affected.

The disease progresses to the muscles of the thighs, particularly to the vastus internus, and usually at the same time the small muscles of the hands, the thenar, hypothenar, interossei, and lumbricales, and, somewhat later, the muscles of the forearm become affected.

The muscles of the upper arm, neck, and body remain unaffected for years, but may eventually become diseased. So, also, the final involvement of the face is not unknown.

As a rule, the atrophy is symmetrical, but occasionally this is not the case, as in a girl seen by the writer, in whom the atrophy involved first the small muscles of one foot, then the muscles of the lower leg upon the same side, and together with this the muscles of the upper leg of the opposite side.

In the lower extremities a paralytic club-foot is early developed, which, especially in children, is a characteristic symptom, and is distinguishable from congenital club-foot only by the electrical reactions. In the upper extremities the wasting of the small hand-muscles causes first flexion of the fingers and then claw-like deformity. The presence of this *main en griffe*, together with double club-foot, in early life, is in itself sufficient for a recognition of the disease. Paralysis and atrophy go hand in hand. Hypertrophy, true or false, does not occur. Muscular spasms are not observed, but slight fibrillary twitchings, tremor, and restlessness in single muscles are not uncommon. Mechanical excitability is reduced in the wasting muscles, and becomes abolished in the wasted ones.

The tendon reflexes also become weaker and weaker, and are finally lost as the muscles upon whose integrity they depend become more and more atrophied.

Of very great import are the changes produced in the electrical excitability of muscles and nerves. This electrical irritability is diminished early and shows complete or partial reaction of degeneration. AnClC precedes CaClC, and the contraction becomes slow and languid. Later a complete loss of excitability to both faradic and galvanic currents occurs.

It should be noted that changes in the electrical excitability of muscles and nerves may be found in certain territories before any signs of atrophy or impairment of voluntary motion are present.

It is also important to recognize the presence of sensory disturbances when they exist. Although many cases show no disorder of cutaneous sensibility, in a number it is either reduced or absent in those territories which are most invaded by the atrophy, and a number of others, while showing no objective sensory disorder, give evidence of subjective ones, such as well-defined pains and paræsthesias. Vasomotor disturbances strictly limited to the atrophic territories are not uncommon, and consist in a marbled cyanosis or reddening of the skin combined with coldness.

Diagnosis. From the spinal forms of progressive muscular atrophy these cases show marked differences; at the same time their commencement in the distal parts of the extremities, the gradual involvement of the more proximal parts, and the final involvement of the body itself, the restlessness of the muscles, which is similar if not identical with fibrillation, the occurrence of reaction of degeneration in the atrophied and paralyzed muscles, the absence of hypertrophy, etc., establish a close resemblance to these spinal forms. So, also, even greater difficulty may be experienced in differentiating this neurotic atrophy from poliomyelitis anterior chronica and from chronic polyneuritis. From chronic poliomyelitis, which is retrogressive in its course, the hereditary and progressive nature will serve to distinguish it. The differentiation from chronic multiple neuritis is the most difficult on account of the close connection between the two; the course of the disease and the constant progression will serve as important diagnostic points; the age of the patient is also of value, for polyneuritis is usually a disease of adult life; in neuritis, furthermore, we expect to find etiological causes which are not present in this form of atrophy. On the other hand, the marked family character of the ailment, and its occurrence at an early age, approach it to the progressive dystrophies, from which the entire absence of hypertrophy and the more marked qualitative changes of electrical excitability will serve to differentiate it.

Pathological Anatomy. There have in all been only three autopsies in cases of this kind, those by Virchow, Friedreich, and Dubreuilt. In these degeneration of the peripheral nerves, fatty and parenchymatous degeneration of the muscles, with simple atrophy of the fibres, together with spinal-cord changes, degeneration of the fibres of Goll, have been found. Whether these changes in the spinal cord are of a primary or secondary nature cannot yet be stated, but judging from a clinical point of view the primary changes are undoubtedly those in the peripheral nerves.

The Prognosis and Treatment of these cases become plain as soon as we recognize the constantly progressive character of the disease. That this progressive course can be arrested or delayed by any known method of treatment is more than doubtful. Whatever methods of treatment are to be tried must be carried out upon the general lines employed in diseases of the muscles and of the peripheral nerves.

MYOTONIA CONGENITA (Thomsen's Disease).

This disease, which is named after the physician who (himself afflicted) first attracted general attention to it by his careful description, is a peculiar disorder of the voluntary movements characterized by motor inhibition, due to a stiffness and rigidity of the muscles, occurring after a period of inactivity. This rigidity passes away in a short time and does not return while the muscles are being used.

HISTORY. In 1876 the disease, as it occurred in himself and in four generations of his family, twenty-three cases in all, was first described by Dr. J. Thomsen. The title employed in his description was "Tonic Spasms in Voluntarily-moved Muscles." The only references to this class of cases prior to 1876 are one by Sir Charles Bell and another by Leyden. Since Thomsen's publication reports of similar cases have been received from nearly all countries. Many of these cases have only certain symptoms in common with the affection described by Thomsen, while others, in addition to the characteristic symptoms, present phenomena which point to the existence of central-nerve disorder. The most complete publication upon the subject, which contains an analysis of all the positive and doubtful cases published prior to 1876, is Erb's monograph on *Myotonia Congenita*. The number of positive cases analyzed in this book is twenty-eight; this number does not include the cases occurring in Thomsen's own family, two of which only have been described, and these without any objective examination. The case described by me is the twenty-ninth, and since then the histories of a few more have been published.

Etiology. The etiology of the affection demonstrates that the most important factor in its production is heredity. In the majority of cases, it is a family disease, and in nearly all the affection was noticed in early childhood as soon as the child was obliged to make systematic use of its muscles. In a number of cases, in addition to the presence of the same affection in other members or in collateral branches of the family, other neuropathic disorders were present. Thus, Weidmann gives the history of a patient of whom one brother was an epileptic, and of whom another had died in consequence of a meningitis. Knud Pontoppidan describes a case in whose family numerous neuropathic disorders were found, and in the family of a patient of Bernhardt a number of consanguineous marriages had occurred.

Both sexes suffer, but males seem to be more frequently affected than females. Of the influence of other causes, particularly psychic emotions, we know little; cases have been described by Seeligmüller, Peters, and Engel, in which the origin of the affection is attributed to fright. Engel's is probably not a genuine case of Thomsen's disease. Climate and country may have some influence upon its production; the affection seems to be more common in Scandinavia and Germany than in France and England, and is exceedingly rare in the United States.

Symptomatology. The disease is characterized by an inhibition and awkwardness of voluntary movements, due to tonic spasm, occurring particularly after a period of rest, and remaining absent as long as the muscles are in use.

This peculiar disorder of motion is already noticeable in children when they first begin to play; they are awkward in their movements and cannot compete with their playmates in such games as require full and rapidly changing control of different muscles.

Every period of rest is followed by fresh spasm, which is stronger the longer the muscles have not been used. After such a period of rest the spasm, in consequence of any muscular exertion, will occur in full force; the muscles then become entirely stiff, and movements can be executed only with the greatest difficulty, if at all.

In a severe case, a slight cause, such as stubbing the toe while walking, will make the patient fall, and he will be unable to rise from the ground until the spasm has subsided.

These spasms always pass off soon, and during the intervals the patients do not differ subjectively from normal individuals.

A patient who, upon getting out of bed in the morning, cannot walk at all on account of the spasm, will, after repeated attempts, succeed in walking a few steps, and will then be able to walk for hours without any trouble; the next period of rest will, however, prepare the way for a fresh spasm.

Various grades of severity are found in different patients. The lower extremities are usually more affected than the upper ones, and, as a rule, all the voluntary muscles of the body, with the exception of those of the face, tongue, and eyeballs, are more or less affected. In a case reported by me these muscles were also affected. Certain influences seem to increase the severity of the symptoms. Above all, we must here place prolonged rest, even standing; cold and damp weather, cold baths, and even great heat, psychical excitement and sudden sensory impressions are important in producing this result. Moderately active exercise serves to ameliorate this tendency to spasm.

In every other way the patients appear normal, no other symptoms of nervous disorders are present, and nutrition is usually perfect.

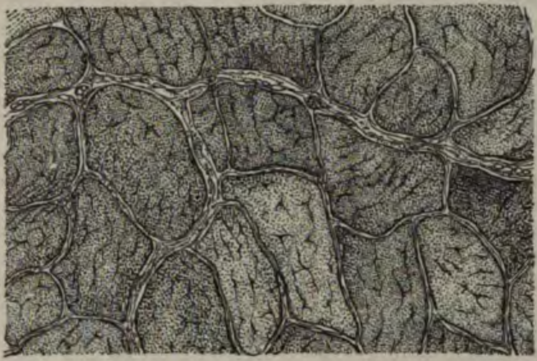
The muscles themselves are well nourished, and are often abnormally large, in most cases presenting a truly athletic development; their strength is, however, always less than their size would lead us to believe.

Fibrillary twitchings and disorders of sensation are not present; the tendon reflexes are usually normal, though sometimes reduced, and may even vary at different times in one and the same individual.

Peculiar changes in the mechanical and electrical excitability of the muscles are found, which have been most carefully studied by Erb, and given the designation myotonic reaction. (MyR.)

The mechanical excitability is increased so that they show overaction to such stimulation. A quick localized blow, as with a percussion hammer, produces a contraction of the irritated fibres, causing a distinct groove in the muscle, which lasts for fifteen seconds or more. Firm pressure causes tonic contraction of the entire muscles with similar persistency.

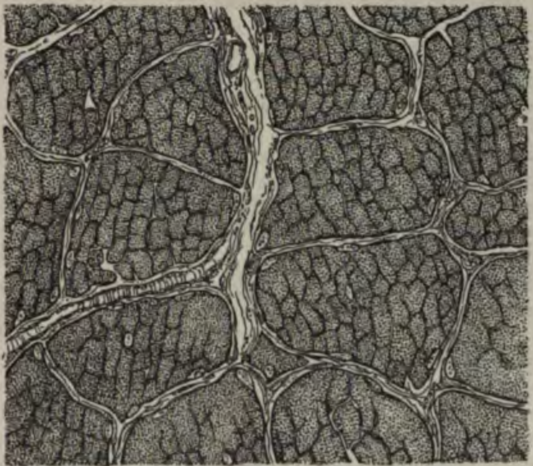
FIG. 288.



Normal muscle. Quadriceps femoris. Transverse section. × 300.

The faradic excitability ot the muscles is also increased and altered, a slow tonic persistent contraction being produced by medium currents. Single opening shocks always produce normal quick contractions.

FIG. 289.



Myotonia congenita. Quadriceps femoris. Transverse section. × 300.

To the galvanic current the muscles show an increased excitability with qualitative change. The cathodal and anodal closure contractions are about equal, or the AnClC is stronger than the CaClC. Marked local furrows are formed under the excitative electrodes. Erb has described, as occurring in his cases, undulating rhythmical contractions, starting at the kathode and passing to the anode, where they ceased.

The mechanical or electrical excitability of the nerves is not changed. In all cases the affection once developed seems to continue during life, though it may at times become so much lighter that it seems to have disappeared.

Pathology. No autopsy has yet been made in this disease, and it is doubtful whether any important results will be gained by an examination of the central nervous system. In a number of cases pieces of the muscles have been either excised or removed by means of the harpoon, intra vitam. The changes thus found by Erb, and corroborated by myself and others, were marked hypertrophy of all fibres and great proliferation of nuclei, with altered appearance of the minute structure. The hypertrophy of the muscle-fibres is so great that they are twice or three times as large as normal fibres. That this hypertrophy is not due to excitation and contraction caused by the process of excisions is shown clearly in the accompanying cut (Fig. 288), showing a transverse section of a normal muscle removed intra vitam from a perfectly healthy individual, from the same locality and prepared in precisely the same manner for microscopical examination as was the piece of muscle removed from the case of Thomsen's disease, a transverse section of which is also shown. (Fig. 289). In addition to the hypertrophy, the fibres differ in shape from the normal, being more circular, with somewhat rounded corners, and not polygonal. The nuclei, greatly augmented in numbers, are not only found along the periphery of the fibres, but also here and there in its interior.

The connective tissue around the muscle bundles and that around the single fibres is distinctly augmented.

The changes seen in the minute structure of the muscle are indistinct transverse striation, irregular non-parallel edges of the fibres, splitting up of fibre into minute fields, with, in many instances, wide gaps between them, homogeneous appearance on transverse section, and formation of vacuoles.

The clinical manifestations of the disease being limited to disordered functions of the voluntary muscles, and the anatomical examinations also showing changes in the minute structure of these muscles, it seems natural that we should argue *post hoc, propter hoc*, and consider the clinical manifestations dependent upon the anatomical disorder. Whether, however, the muscular changes are the primary ones, or whether these are secondary to some disorder in the central nervous system, is a question which cannot as yet be decided. The absolute functional and trophic dependence of the muscles upon the central nervous system, the fact that the myotonic disorder has been noted in diseases of the central nervous system, the hereditary tendency to nervous diseases occurring in some of the families with Thomsen's disease, the occasional etiological influence of fright, and the influence of psychic excitement upon the condition of the sufferer, are facts which make it impossible to assert that the disease is a myopathic one. It is clearly possible that, primarily, the nervous system is at fault, and that we are dealing with a tropho-neurosis of the muscles dependent upon disorder of the central trophic apparatus.

On the other hand, the reaction of the muscles to direct mechanical and electrical excitation, while the reaction from the nerve-trunks remains unchanged, is, to say the least, a powerful argument in favor of the myopathic theory.

Diagnosis. The diagnosis of this affection presents no difficulty on account of the characteristic features of the disease. The family tendency, its manifestations in early childhood, together with the "myotonic reaction" will serve to dispel all doubts. The myotonic reaction of itself is, however, not characteristic of Thomsen's disease, as it is found occasionally in acquired disorders, the precise nature of which is still in doubt. Simulation can, as Erb tersely puts it, always be detected by a few blows with the percussion hammer and a few anodal and cathodal closures with the galvanic current.

Treatment and Prognosis. No treatment seems to exert any influence upon the disease. Patients, in time, learn to avoid certain influences, such as cold, damp air, psychical excitement, etc., and to encourage others, as muscular exercise, and are thus enabled to lead a fairly comfortable life. The disease lasts during the entire life of the patient, and death, when it occurs, is due to some other disease.

ARTHRITIC MUSCULAR ATROPHY.

It is well known that muscles surrounding diseased joints rapidly waste, and that this atrophy bears no relation to the nature nor to the cause of the joint affection. Chronic and acute affections, spontantaneous or traumatic ones, are equally followed by this atrophy. The muscles chiefly affected are the extensors of the diseased joints. The atrophy is of very common occurrence, and presents the following clinical features: The primary joint trouble is, after a more or less short interval, followed by a weakness in the affected extremity, chiefly characterized by an inability to entirely or partially extend the distal portion of the limb. Frequently the patient still complains of pain in the joint.

Upon examination we find that the joint itself is either normal or that it is still implicated to so slight a degree as to be entirely disproportionate to the amount of functional disturbance. The proximal portion of the limb, however, shows marked atrophy of its muscles. If the ankle joint is affected the calf muscles are wasted; if the knee, the quadriceps; if the hip, the glutei; if the wrist, the extensor muscles of the forearm; if the elbow, the triceps, and if the shoulder joint, the deltoid, supra-spinatus, infra-spinatus, and teres minor are affected.

In arthritis of the finger-joints this wasting is marked in the interossei.

Thus, as a rule, almost without exception, the extensors of the joint alone are affected, but occasionally the flexors also are implicated, but always to a slighter degree than their opponent. Even other muscles of the proximal part of the extremity may be affected, but it is very unusual to find any muscles of the distal part atrophied. Characteristic of this kind of atrophy is that it occurs very soon after the injury to the joint, and that it affects the muscles in toto. Thus, in a week after the injury a difference in the circumference of the limb may be detected by measurement, and the wasting, while it may vary as to degree, always affects the muscle throughout its entire length. The atrophy increases during a time, and then becomes stationary and does not begin to improve until the joint trouble has disappeared. Occasionally the atrophy may persist for a long time after disappearance of the joint affections. Atrophy and loss of functions go hand-in-hand.

The electrical excitability of the affected muscles is either normal or quantitatively reduced, so that they respond to strong currents only. Never is there any qualitative change, thus proving that the atrophy is simple in nature and not degenerative.

Mechanical excitability is usually increased in the affected muscles, and an excessive knee-jerk may be obtained if the muscles of the thigh are affected, and a foot-clonus if the ankle-joint is involved.

Sensory symptoms are not present in the pure forms of arthritic atrophy.

Pathology. Microscopical examination shows the atrophic muscles to be paler, more flaccid, and less elastic than is normally the case.

Microscopically the muscular fibers are narrowed, but do not present any other deviation from the normal. Neither in transverse nor longitudinal sections can any degenerated fibres be found; their striation is regular and granular; breaking down of the fibers does not occur. The nuclei of the periphery may be increased and the interstitial connective tissue is augmented. Such fibres in various stages of atrophy are found throughout the entire muscle. Examination of the nervous system has failed to reveal any change in either the central or peripheral parts except in the articular nerve endings in the joint. The changes here found are undoubtedly due to direct extension of the joint affection, and do not in any way explain the atrophy of the muscles.

The question of the manner in which these arthritic atrophies are produced is one which has given rise to a great deal of discussion. The oldest and most simple explanation ascribed these atrophies to functional inactivity of the limb; but this theory is not tenable, inasmuch as the atrophy occurs even if the limb is left movable and the joints are not fixed; furthermore, the rapidity with which the atrophy occurs invalidates this theory, for, while we know that inactivity may lead to a certain wasting of the entire limb, we also know that never does an immobilized limb in which there is no joint affection atrophy in the manner here described.

That the atrophy is due to an insufficient blood supply, that it is the result of a secondary myositis, or that it is caused by an ascending neuritis, are all theories, which, for a time, were advocated by various observers, but whose correctness has been disproved.

The only theory which satisfactorily explains the condition is the reflex hypothesis of Vulpian and Charcot. According to this hypothesis the irritation which is produced in the articular nerve-endings by the arthritic trouble is centripetally propagated to the spinal centres of the motor nerves supplying the wasting muscles, and here produces a molecular change, which is the direct cause of the atrophy.

The correctness of this theory has been experimentally proved by Raymond and Déroche. These investigators, assuming that if the atrophy is actually a reflex one, interruption of the reflex arc must prevent its occurrence, cut through the posterior spinal roots, with the result of preventing the occurrence of the atrophy after an experimentally-produced arthritis. Hoffa has repeated these experiments in a large number of dogs, and was enabled to corroborate the statements of the above-mentioned observers. In a dog in whom the third, fourth, and fifth lumbar, and first sacral posterior nerve-roots of one side had been previously divided, a purulent inflammation of both knee-joints was produced. Upon the side on which the nerve-roots had been divided no atrophy occurred, while upon the other side there was atrophy of the quadriceps so marked that a difference of nearly 1 cm. existed in the measurements of the two limbs. There can hardly be any reasonable doubt that this experiment proves the correctness of the reflex theory.

The reason why only the extensors of a joint are affected in this reflex manner must be sought in the direct anatomical connection which the articular nerves have with the nerves which supply the extensor muscles of the joint.

Diagnosis. The diagnosis will be based upon the preceding joint affection, upon the wide extent of the atrophy, and the reduction in the electrical excitability without any reaction of degeneration. The course of the affection will serve to differentiate it from any of the progressive muscular atrophies.

Prognosis. In acute joint affections the atrophy is usually of short duration, and restoration of function may be speedily hoped for as soon as the joint is restored to its normal condition. If the joint trouble is chronic the wasting will last for a long time, even after cure of the joint affection.

Treatment. The primary point in the treatment of these atrophies is the removal of the joint affections. As long as any disease exists here no increase in bulk of the muscle is to be expected. As soon as the joint is in a normal condition the muscles usually begin to improve of themselves, but this is not always the case, and in many instances the improvment only takes place up to a certain point, and then remains stationary. It is in such cases that local treatment is of the greatest importance. Electricity, galvanism or faradism, of sufficient strength to produce good contractions of the muscles, massage, superficial and deep active movements, with and without opposition, together with hydrotherapeutic procedures, are the remedies which in nearly all cases will enable us to completely restore the muscles to their normal conditions.

CHAPTER XXX.

THE TROPHO-NEUROSES.

By JOSEPH COLLINS, M.D.

Under the title of tropho-neuroses are included a number of diseases in which anomalies of nutrition depending on nervous origin is the one striking feature of the disease. The number of these diseases is gradually increasing. The question of whether or not there exist specific trophic nerves is to-day quite as unsettled as it was a quarter of a century ago. There is any amount of clinical evidence, and that of a convincing kind, to prove the existence of trophic nerves, and there have not been lacking writers and investigators who have claimed the discovery of such nerves, but experimental physiology still fails to reveal any clue of their presence. The truth is that it gives a negative answer. Many trophic disturbances are probably due to vasomotor changes, and it is not possible to separate by any sharply-defined lines the vasomotor and the tropho-neuroses. At the same time it should be distinctly stated that there exists tropho-neuroses in which there are no appreciable vasomotor disturbances, as in many cases of acromegaly and hypertrophies; and, on the other hand, there are any number of vasomotor disturbances which are in no sense trophic. Still there is the closest relationship between vasomotor and trophic disturbances, and it is not improbable that further research into the domain of experimental physiology will reveal that this intimacy is dependent on the relationship existing between the spinal ganglia and the sympathetic system. Recent investigation in this direction tends to postulate the spinal ganglia as responsible for trophic conditions, and the sympathetic ganglia for the vasomotor, and the theory that may be constructed on this supposition explains better than any heretofore the interlationship and partial dependence of one upon the other.

It is not my purpose to consider here the physiology of the vasomotor nerves, no more is it to give in detail the arguments that have been advanced to prove the existence of trophic nerves and centres. These subjects belong properly to text-books on physiology and experimental medicine, and in them may be found any amount of facts and theory bearing on these subjects.

Some of the diseases which are considered in this chapter are of recent recognition, but so assiduously have they been studied that their morbid anatomy and clinical course are quite as well understood as those of much older date.

RAYNAUD'S DISEASES.

Synonyms. Symmetrical Gangrene; Local Asphyxia; Asphyxie Local Symmetrique; Neuropathic Gangrenous Trophoneurosis.

In 1862 Maurice Raynaud described a variety of dry gangrene which he had observed in twenty-eight cases, and which he characterized as a neurosis dependant on an exaggeration of the excito-motor power of the cord presiding over the vasomotor nerves; a kind of gangrene which owns for its cause some error of innervation of the capillary vessels. He showed that in many instances there were three stages in its development: The first, accompanied by icy coldness and pallor, the dead-finger stage, and this he termed local syncope, the affection being usually paroxysmal; the second stage that in which the blood is unfitted to the nutritive requirements of the parts, stagnates in the extremities, he called local asphyxia; the third stage, that of gangrene, when mortification occurs. In 1874 Raynaud contributed a second paper which contained a record of thirty-one cases. Since that time, and particularly since Barlow's translation of Raynaud's article into English, many cases have been reported, although the disease may be said to be extremely rare. The term symmetrical gangrene, which the discoverer himself gave to the disease, is not entirely appropriate, for I have been able to find in the literature no fewer than eleven cases in which the condition was manifest in one extremity only, and in many cases the disease does not go on to gangrene, so although eponyms should be discouraged, the most fitting name for the disease is as given above.

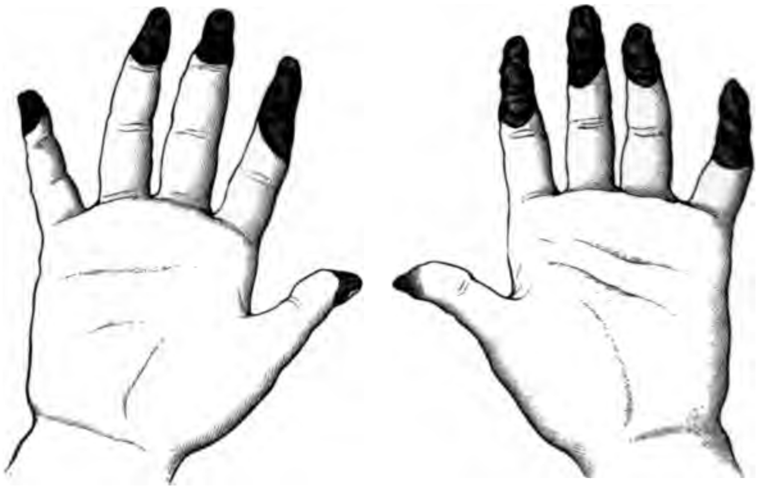
Etiology. Raynaud's disease occurs very frequently in patients who are the possessors of other nervous diseases, such as hysteria, epilepsy, tabes, myelitis, syringomyelia and neurasthenia. It is not uncommonly seen in insane asylums, particularly in patients who have acute mania.

It occurs about twice as often in females as in males. In a hundred cases taken from the literature sixty-one were females and thirty-nine males. It may occur at any age, but is much more frequent during adult life, from twenty to forty. Cases have been reported in children under two years, and early childhood furnishes a goodly proportion of the cases. No cases have been recorded as occurring for the first time after sixty years. Raynaud attributed great etiologic weight to disturbances of menstruation; but statistics since his time have not corroborated him in this. Syphilis was a direct etiological factor in one case; this was proven to be a fact by the prompt disappearance of the symptoms under antiluetic treatment (Barlow: *Lancet*, July 6, 1889), and in a few others it is possible to get an inherited or acquired syphilitic history. A neuropathic diathesis is to be made out in a large proportion of the cases. Anæmia and all conditions of impoverished blood, whether due to malaria, excesses, or intercurrent diseases, are strong predisposing factors. Cases of Raynaud's disease are sometimes seen associated with urticaria, scleroderma and telangiectasis. The exciting causes are fright, exposure to cold, frost bite, acute fatigue and trauma, especially to the sympathetic system, through blows or injuries over the solar plexus and splanchnic ganglia. The disease has come on in a few cases after the acute infectious diseases, including influenza, in one case after dog bite, and in another after a poisonous dose of chloral. By some writers the original cause of the disease is considered to be of a toxic nature.

Symptoms. The local symptoms generally precede any subjective symptoms, and the patient remarks that the fingers or toes, or, more rarely, the nose, after or without having been exposed to the cold, looks pale and glossy, and presents the characteristics of a local syncope or regional ischæmia. With this there is a feeling of tingling and itchiness, a sensation as if the parts had been stung by nettles, and a feeling of numbness and loss of dexterity and tactile sensibility. This condition may persist for some time; gradually, or paroxysmally, the parts affected become more blanched and the local syncope more exaggerated. The end or the entire finger may become apparently completely exsanguinated, waxy, and colorless, the so-called *digiti mortui*. To the touch they are cold and pulseless, although the pulse at the worst may be demonstrable. A needle plunged through the skin into the tissues does not cause a drop of blood. With this stage there may be some constitutional symptoms, such as in the beginning a chill, nausea, anorexia, constipation, a general feeling of hypothermia, lack of energy, pain in various parts of the body, and a sense of tingling in other extremities which are not the seat of local syncope. If the symptoms persist, the skin either preserves its glassy, waxen look or it becomes shrivelled and puckered. This stage, after lasting from a few minutes to several hours or days, may pass off without leaving a trace, or it may pass into the second stage, that of local asphyxia.

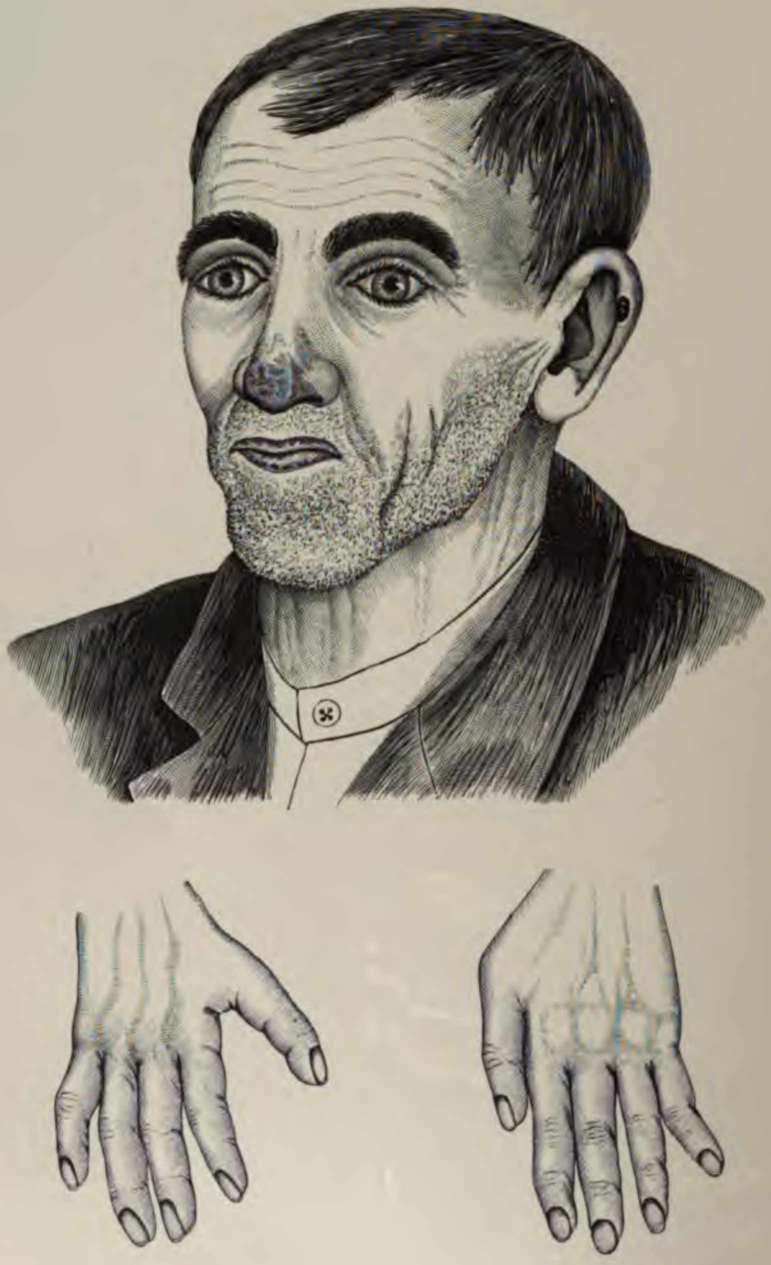
In the stage of local asphyxia or regional cyanosis, the tips of the fingers, the toes, or any part affected begins to assume a bluish-black appearance. The nails especially look as if they had been dipped in ink. If the affected parts are pressed upon the anæmia produced takes a long time to disappear. Occasionally in this stage of regional cyanosis, the fingers are swollen, of a vivid-red color, extremely hot, covered with a profuse perspiration, the capillaries and all the vessels fully distended, and anæmia caused by pressure disappears rapidly. In both forms the integrity of the circulation is interfered with, and the results are the same. Simultaneously with the appearance of this stage the patient begins to complain of pain generally of a neuralgic character, and generally in proportion to the cyanosis so is the pain. The fingers of both hands or the toes may be involved at the same time, but frequently the appearance in one hand slightly antedates that of the other. After continuing in these parts for a time it is not uncommon to find the ears and even the tip of the nose beginning to be similarly affected. Leading up to the third stage, the epidermis over the affected parts may show small blisters, and in some cases these contain a small quantity of blackish blood. After several hours or longer the skin over the black portion of the fingers, especially over the bulbs, separates from the adjacent parts, the derma becomes extensively destroyed and small ulcers can be seen eroding the deeper tissues, or the gangrene takes a deeper hold, and the terminal phalanges or even the entire finger becomes shrivelled and mummified (Fig. 290). If the gangrenous process is limited to the formation of small necrotic areas, these heal slowly after the attack has passed off, and the only evidence re-

FIG. 290.



Gangrene of fingers in Raynaud's disease. (DEHIO.)

PLATE VII.



Local asphyxia of hands, nose and ears, and gangrenous patch in left ear. (Henry.)

maining is a slight scar in the pulpy part of the finger. A number of these scars may tell of many previous attacks. If the gangrenous process is of the severer form, the skin of the end of the fingers becomes black, dry, and shrivelled, while adjoining, in the healthy tissue, may be seen forming the line of demarcation from which a reactive process is to be set up which separates the dead part, and from which the reparative process develops. The process may be so severe that spontaneous amputation of extremities, such as the feet, may occur. The process of separation and cicatrization is a very slow one and frequently extends over many months. In the majority of cases, however, the gangrene does not go on to this extent.

The disease may show itself in other parts of the body than the fingers, toes, ears, and nose, but in these it is most common. (See Plate VII.) It is not, however, uncommon to find patches over the heels, deltoid muscles, calves, maleoli, nates, cheeks, and lips, and on the abdomen especially on each side of the umbilicus. When it involves any of these parts it does not ordinarily go on to complete gangrene. The tongue, the penis, and the vulva are unusual seats of the lesion.

The constitutional symptoms of this disease vary, and are not altogether constant. The absence of fever is characteristic of its entire course, and during the stage of local asphyxia the temperature of the skin of parts affected may be very much lowered. The most interesting symptom, as was emphasized by Barlow, is intermittent hæmoglobinuria. This may occur during an attack, or it may seemingly take the place of an attack. The causes of hæmoglobinuria occurring apart from Raynaud's disease are not well understood; but unlike the latter disease it occurs preponderatingly in males, and particularly in those who sometimes in their lives have suffered from malarial infection. And, furthermore, the symptoms attending hæmoglobinuria *sui generis* are much more severe than those of Raynaud's disease. Psychical disturbances sometimes occur at the beginning or during the course of the attack, and consist of a feeling of malaise, irritability and depression, transient attacks of aphasia (Weiss), loss of motor power (Raynaud), unconsciousness (Englisch), convulsions (Southey), all of which may be attributed to ischæmia resulting from spasm of the arteries of the representative parts of the brain, analogous to that occurring in the arteries of the fingers and extremities. Disturbances of vision during a paroxysm is not uncommon. It may be a simple dimness or obscuration dependent upon a narrowing of the calibre of the central artery of the retina and its branches, and which can be demonstrated ophthalmoscopically, or it may be associated with profound hebetude, bluntness of hearing, noises in the ear, perversion of taste, and other symptoms that point to vascular depravity in the brain. Occasionally the disturbance of vision may be due to pupillary conditions, such as iridoplegia (Hutchinson), and these cases point to an involvement of the sympathetic.

Fortunately these severer symptoms do not occur in the majority of cases. Trophic disturbances, such as atrophy of the muscles of the hands, proliferation of the epidermis, urticaria, chilblains, changes in the nails, and even scleroderma may occur. Very rarely synovitis and evidences of involvement of one or more of the joints are seen. In those cases in which neuritis and lepra nervorum are present there will be striking defects of sensibility and motion, and changes in electrical irritability.

The duration of the disease is a variable one. The first and second stages last from a few hours to several days, and the third stage, depending if it goes on to mummification, or if limited to the formation of local necrotic spots in the form of ulcers, is a variable one. If the former occurs, three or four months will elapse before recovery sets in, and in the latter case the same number of weeks may suffice. The first attack may be the only one, but in the great proportion of cases the disease returns after a variable intermission, during which time other vasomotor phenomena, such as urticaria and angeioneurotic œdema, may show themselves.

In children the disease runs a very rapid course and frequently there is nothing paroxysmal about the attack; the symptoms progress uninterruptedly and the child may die within two days from the time when the disease first showed itself.

The pathology of the disease is obscure, and very little can be said with absolute certainty. Most observers of the disease since the time of Raynaud have corroborated his opinion that the local syncope is produced by a contraction or spasm of the vessels, the arteries and veins, in the parts affected. The local asphyxia occurs when the spasms cease in the veins and capillaries, but still continues in the arteries, such is the condition when the second stage is characterized by a black appearance of the parts. When they are of a vivid-red color, as has previously been described, it is probable that there is a paresis of the vaso-constrictors, or more probable an irritation of the vasodilators, which retards the circulation through the parts. Attempts have been made to associate the disease with certain pathological conditions of the arteries resulting from syphilis, Bright's disease, etc., but if these conditions are present they can only be regarded as intercurrent or accidental. That the disease does occur with certain diseases of the spinal cord, such as tabes, syringomyelia, and myelitis, is beyond cavil. In a few cases neuritis has been found in the nerves distributed to the affected parts (Pitres and Vaillard: *Archives de Physiologie*, 1885), (Wigglesworth: *Trans. Path. Soc.*, 1887, London, vol. xxxvii.), (Affleck: *Brit. Med. Journ.*, 1888, vol. ii. p. 1269), but it is probable that these changes were secondary. The same may be said of those cases where endarteritis and endophlebitis have been found, as in the cases reported by Dehio and by Goldschmidt. Raynaud's disease may attack people whose vessels are not healthy, but in such individuals spasm of the bloodvessels and not degeneration of their walls is at the bottom of the disease; back of all this is some derangement of the sympathetic nervous system and that part of the central nervous system from which the sympathetic has its origin. It may be due to affection of the local vasomotor system, as in cases where exposure to cold is directly the cause of the attack. It may be due to affection of the sympathetic ganglia, as in a case reported by Collier (*Manchester Med. Chronicle*, 1889), where irritation of the great abdominal sympathetic from an ancient and recent peritonitis was clearly the cause. Again, it may be due to lesion of the vasomotor centres or conducting paths in the spinal cord, syringomyelia, tabes, etc., or of the vasomotor centres and areas in the medulla and brain; and finally it may be called into being through physical or psychical reflexes.

Diagnosis. When the affection is well developed it is scarcely possible to mistake it for any other disease. The symmetry of development, the distribution, the sensory, motor, and trophic changes coming on in regular order, and the absolute lack of dependence of these symptoms on constitutional disease, are characteristic. Other forms of gangrene can be differentiated by their etiology, and it is only necessary to mention, senility, ergot, constriction, which have not only these etiological peculiarities, but cause gangrene having very different appearance and distribution. Erythromelalgia may be confounded with Raynaud's disease when local erythema occurs in the second stage, but it may be differentiated by its mode of onset and the fact that erythromelalgia never goes on to gangrene. Diabetes and nephritis can be quickly ruled out by an examination of the urine. It should not be forgotten, however, that both of these conditions, particularly the latter, may complicate Raynaud's disease.

Prognosis. Generally speaking the prognosis may be said to be good. The proportion of cases that terminate fatally is not easy to calculate. Numerous cases in which death occurred are on record, but as a rule the cause of death was some complicating or intercurrent disease, such as phthisis. The prognosis is bad when the disease develops in individuals who are already afflicted with some incurable disease, such as tabes or syringomyelia. Except in children an attack probably never eventuates in death. When the disease goes on to the formation of gangrene *en masse* the general health may become so depraved that the body becomes an easy victim of infectious disease, particularly tubercle, which in turn shortens the patient's life.

Treatment. The most important factors in the treatment of this disease are to increase the patient's nutrition, to counteract the neuropathic diathesis from which so many of these patients suffer, and to render him immune to causes that are known to excite attacks, such as exposure to cold, mental shocks, and the like. Measures that contribute to the patient's general health, and which are consistent with bodily and mental quietude, such as change of climate, regular and systematic exercise, and avoidance of fatigue, should be taken. During an attack, if the pain is very severe, it may be necessary to give an anodyne, such as morphine, which, however, should never be injected into the seat of the pain, for any irritation to those parts tends to increase the liability to gangrene and amount of destruction if the gangrenous stage does occur. The affected parts should be wrapped in cotton and placed in a position most favorable for the circulation, the temperature of the part maintained by a moderate degree of artificial heat, either dry heat or occasional lukewarm hand and foot baths. The administration of nitroglycerin and nitrite of amyl has been warmly recommended by some, but the consensus of opinion is that use of either one is usually disappointing. Electricity has been used extensively since Raynaud first recommended it, but its therapeutic value in this affection is very small. Peter, of Paris, recommends the use of the galvanic current, the positive pole applied over the cervical enlargement and the negative pole in a basin of warm salt water. Each of the four extremities are bathed in turn for five minutes, while the number of elements used is increased from 4 to 8, 10, and even 16 for each member, the intensity of the current being from 2 to 3 milliampères. It has also been recommended to apply one pole of the galvanic current to the cervical region and the other to the lumbar and send the current through the spine, and likewise a mild galvanic current to the sympathetic in its various portions, but the truth is that this method of treatment is of very little use. When gangrene sets in it should be treated according to the principles of modern surgery.

ANGIONEUROTIC ŒDEMA.

SYNONYMS. Acute Circumscribed Œdema; Acute Idiopathic Œdema; Periodic Swelling; Urticaria Tuberosa, or Giant Swelling; Acute Noninflammatory Œdema.

Angioneurotic œdema is a disease characterized by the appearance of circumscribed swellings on different parts of the body, by preference the face, throat, and extremities, without apparent cause or premonition, and noninflammatory in nature. Reference to the disease can be found in medical literature dating back as far as 1827, but it is only since 1882, when Quincke and his pupil, Dinkelacker, gave a critical description of the disease, that it has been thought anything more than an intercurrent symptom. The term angioneurotic œdema, under which the disease is most commonly described in this country, is not, considering our ignorance of the pathology of the disease, an entirely justifiable one, as it takes for granted certain factors in the genesis of the disease which are not demonstrable. The term acute circumscribed non-inflammatory œdema is the term which best expresses the clinical characteristics of the disease.

Etiology. The most important predisposing causes are heredity, disordered health, overwork, and exhaustion. In a large proportion of the cases that have been reported there is evidence of a strong neuropathic taint, manifesting itself as hysteria, hystero-epilepsy, neurasthenia, or some evidence of degeneration. Direct heredity is one of the most important and interesting elements in the production of the disease. It has been pointed out with remarkable accuracy by Strubing (*Zeitschr. f. klin. Med.*, ix. p. 389), Quincke (*Monatschrift f. prakt. Dermatol.*, 1882, i.), Falcone (*Rivista Veneta, Veneza*, 1887, vii.), Osler (*American Journal of the Medical Sciences*), and others. In the latter's case the author was able to trace the hereditary influence through five generations, in which time no less than twenty individuals were afflicted with the disease. In the fourth generation eight people were affected. In cases that are directly hereditary, male and female seem to furnish about an equal number of cases.

The sinister influence of psychical and physical exhaustion can be traced in some cases, although the greater number of these patients considered themselves in good bodily health. In some cases the disease develops while the individual is doing exhausting bodily and mental labor, as was the case in a physician under the writer's observation.

The period of early adult life furnishes the greatest number of cases, the average age being about about twenty-seven years. Childhood is by no means exempt. Dinkelacker has reported a case in a child three months old, in whom the disposition to the disease was inherited. It rarely occurs for the first time in individuals upward of sixty years, although it does occasionally, as is shown by a case reported by Goltz (*Deutsch. med. Wochenschrift*, 1880, No. 17). An examination of ninety cases, to determine which sex is most frequently affected, shows that it occurs about twice as often in females as in males in Germany and in France, while in this country and in England the occurrence in the sexes is about equal.

Among the exciting causes that may be mentioned as having been found in several cases are exposure to cold, gastric irritation, the onset of puberty, the climacteric, masturbation during the unstable time of adolescence, trauma, fright, and the influence of certain toxic substances, particularly tobacco, malaria, and alcohol. The causative influence of the last three mentioned is frequently seen when they have been intercepted for a time and then renewed. Such, for instance, as excesses in alcohol and tobacco after a period of cessation, and return to a malarial district after a period of prolonged absence. Of the exciting causes exposure to cold is one of the most potent, and is shown in cases reported by Starr, Widonitz, Kirsch, Jamieson, and myself. In one of Starr's patients the hands swelled every time she put them in cold water, and in winter exposure of the face and hands when out of doors, or of the buttocks in a cold water-closet, was sure to be followed by an appearance of the swelling.

The time of the appearance of the swellings has been in some cases very regular; such, for instance, as in a case reported by Matas (*New Orleans Med. Journ.*, Oct. 1887); but in these cases the periodicity is to be attributed to malarial poisoning. In the great number of cases the swellings may appear at any time during the twenty-four hours; the time when attacks are most liable to show themselves, however, is between 2 and 6 A. M., a period when the tide of life is at its lowest ebb and the parts least resistant.

Attacks are more frequent in summer and winter than during the other two seasons. In the summer, for the reason that it is during this period that sudden cooling is apt to take place.

It has been previously mentioned that this disease is seen in those who are handicapped through some inherited or acquired neurotic taint, but it must be mentioned that it occurs in those from whom no neurotic history can be obtained, who have no bad habits, and who are in comfortable circumstances in life. In fact, it may be said that in a considerable number of cases the closest search will not reveal an attributable cause.

Symptoms. The manifestations of the disease generally present themselves without warning and most commonly on the face, the lips, the tongue, pharynx, forehead, and genital organs. In some cases the patient may complain a short time before the appearance of the swelling of a feeling of malaise or depression, associated with ill-defined gastro-intestinal symptoms. The œdema reaches its full development in from one-half to two hours, and gives the patient very little discomfort, except by its mere presence. There is a feeling of stiffness and unwieldiness and a sensation of tension, but no subjective sensation of inflammatory swelling, such as a sense of throbbing and pulsating pain. The swelling varies in color, is clearly circumscribed, and is closely differentiated from the surrounding surface. Ordinarily the color is whitish, with a little rose tinge, but in some cases the skin over the swelling is of a dull roseate hue, with a whitish shading near the centre of the swelling. The swelling does not pit on pressure, or, if it does slightly, the indentation is quickly effaced. The amount of swelling varies in each case; sometimes it is so slight that it is scarcely noticeable, while in other cases the patient is quite unrecognizable when the swellings occur on the face. It lasts from a few hours to a few days, but in the vast majority of the cases no trace of the swellings can be found after twenty-four hours. It may occur in separate parts of the body simultaneously, but frequently its disappearance from one part of the body is the signal for its manifestation in another, which may have no relation to the part previously affected; for instance, it will jump from the forearm to the eyelids, or from the lips to the crest of the ilium, or from the cheeks to the stomach—that is, to parts having no apparent anatomical or physiological connection. As a rule, it does not show itself in more than two or three localities at one visitation, and frequently in only one. After its disappearance from an area, a heavy wooden sort of a feeling is complained of, although the responses of the different sensations are unimpaired.

The subjective sensations are a sense of scalding or burning as the swelling is showing itself, and due probably to the tension under which the skin is suddenly placed. Occasionally there is a feeling of itchiness, not only in the parts that are swollen, but in the neighboring skin, and if the skin is scratched an artificial urticaria is the result. Aside from these if the swelling does not encroach upon any organ, such as the eye, the stomach, the penis, and testicles, or if it does not block up the conductivity of a passage, such as the mouth, pharynx, the larynx, or intestine, as it sometimes does, there will be scarcely any other subjective symptoms. There are two symptoms or conditions which sometimes occur, but which must be regarded as complications, viz.: hæmoglobinuria and albuminuria, and effusion into the joints.

The mucous membrane of the larynx, the pharynx, stomach, and intestines are sometimes affected, and when any of these parts is the seat of the swelling the symptoms may become very distressing, and even inimical to life. When the swelling appears in the larynx the symptoms are those of obstruction, and are in proportion to the amount of encroachment that it makes. This is often so great that distressing symptoms of suffocation are produced, and in some instances death has taken place in consequence of the œdema, as in two of the cases reported by Osler. In some cases the symptoms are so urgent that it is necessary to make scarification of the membrane or to do tracheotomy. When the swelling is in the glottis or the pharynx difficulty in swallowing and a sense of constriction are proportionate to the amount of œdema. In a few instances the swelling has come on so abruptly and reached such an extent that the awful sensation of choking to death develops within a very short period. Only one case has been reported where the œdema of the glottis was so great that death resulted. (Krieger: *Meditzinskvie Obozrenie*, 1889, No. 17).

In about one-third of the cases gastro-intestinal symptoms are prominent. They are, first, a feeling of uneasiness and tension, as if something undigestible had been swallowed; with this there is loss of appetite and sense of weight in the epigastrium, which is soon followed by a feeling of distention in the stomach, nausea, and constipation. The symptoms grow more distressing, and sharp colicky pains often attend with profuse vomiting, and great thirst follows. The pain is often so severe as to demand the administration of morphine. The character of the vomit depends at first upon the contents of the stomach, but later it is watery, contains a good deal of stringy mucus, and is profuse in quantity. After the paroxysm, which may last from a few minutes to several hours, there is frequently diarrhœa of a colliquative nature with an apparent retraction of the abdomen, and a general feeling of languor and prostration. Often during or following such an attack the patient passes a large quantity of water, which, however, contains nothing abnormal save an increased quantity of earthy phosphates.

A few authors have reported cases in which the œdema seemed to manifest itself in the lungs, although this has never been proven by post-mortem examination. If it be granted that it does occur in the lungs, the symptoms resulting will not differ materially from those of ordinary pulmonary œdema except in the suddenness of its onset and the urgency of the symptoms and, frequently, the abrupt mode of departure.

The primary point of manifestation and the area of distribution of the swellings were studied by me in a series of 71 cases, personal and taken from the literature. In these the swelling showed itself primarilly: in the face in 29 cases, on the extremities in 22, in the larynx in 5, on the genitals, penis, vulva, and scrotum in 3, on the body in 6, on the gums and palate in 1, in the stomach in 3, on the neck and behind the ear in 1 each. Of the cases in which the swelling appeared primarily on the face, 3 were restricted to the forehead, 3 occurred first on the eyelids, 7 on the lips, while the remainder were distributed on various parts of the face. On the extremities the hands are by far most frequently affected, and the swelling here attains sometimes an enormous degree. The crest of the ilium, the buttocks, the front of the abdomen, and over the shins, may be the seat of the swellings.

Although it attacks some regions by preference, it rarely confines itself to one locality; migration is one of its marked characteristics, and rarely is it confined to one locality. The occurrence of swelling in one spot seems to predispose that place for future attacks. Parts of the body which have previously been injured, or which have been the seat of protracted pain, seem in some instances to be a favorite seat for the swellings.

The interval between attacks is a variable one: in some cases a few days, in others as many months. The general health between the attacks is, as a rule, good. In one case recently under the writer's observation, there was some tachycardia, palpitation, and a pulse of very low tension; but the cause of the disease in this man was undoubtedly tobacco, it being his habit to consume from thirty to fifty cigars daily; so the cardiac symptoms were attributed to the smoking. For a day or two after an attack there may be a feeling of tiredness and languor, especially if the digestive mucous membranes have been involved, but this soon passes away, and the patient remains well until the appearance of the next attack.

Diagnosis and Prognosis. The diagnosis of this disease is, as a rule, not difficult, particularly if arrived at by a process of exclusion. The fact that the swelling is distinctly circumscribed, that it is not tender to the touch, that it does not pit on pressure, that it has none of the characteristics of an inflammatory swelling, that it is frequently associated with urticaria, and the mode of its onset and disappearance are characteristic and are not easily mistaken for other forms of œdema. Kussner (*Berlin. klin. Wochenschr.*, 1889, No. 16) has described angioneurotic œdema as occurring with a rheumatic attack, but it is probable that such attacks are but intercurrent non-inflammatory exudates into the joint, which complicate this disease, as was mentioned previously. It may be confounded with the blue œdema of hysteria, as described by Sydenham, or the white œdematous swellings occurring with the same disease, as described by Charcot. In hysteria, however, and especially if the hysterical attack is sufficiently profound to have œdema as one of its attendants, there will always be found some of the well-known stigmata of hysteria, which will be sufficient to make the diagnosis. It may be said with truth that the œdema occurring with hysteria is an angioneurotic œdema, but it has a different symptomatology and suggests different treatment than the symptom-complex which we have described. Although almost all the cases of angioneurotic œdema are developed in neurotic subjects, in my experience it is most uncommon to find any evidences of hysteria. Malarial œdema, menstrual œdema, and a disease of the Antipodes, known as Australian blight, are in all probability forms influenced by the etiological factor of the disease in question. It is barely possible to confound it with erythema nodosum, but the absence of tenderness and the pale color will make the diagnosis. The integrity of the internal organs and the peripheral vessels will exclude the possibility of attributing the œdema to some primary cause. The duration of the disease varies from a period sufficient for one attack to a lifetime. It is probable that a respectable proportion of the cases cease spontaneously as the patient grows older. In some cases, after an absence of several years, the symptoms manifest themselves. In some cases it continues to recur with varying intervals during the patient's entire life, which may not, however, be perceptibly shortened by the occurrence of these attacks. In a general way it may be said that when the disease does not appear in the mucous membranes it does not in any way interfere with the life of the patient. When it does affect the mucous membranes, especially of the larynx and pharynx, there is danger to life from suffocation. The prognosis as regards recovery is fairly good, particularly if there be no hereditary element in the etiology. The prognosis is best in those cases which can be proven to be dependent on some toxic element, such as tobacco, alcohol, and malaria, and in those associated with some diathesis, such as the lithæmic.

Pathology. The nature of the lesion is unquestionably that of a non-inflammatory œdema, and clinical and experimental evidences tend to show that the essential disease is a vasomotor neurosis rather than a tropho-neurosis. The vasomotor system frequently is called upon to manifest psychical states, either by an excessive dilatation of the vessels or by a contraction, causing blushing or pallor. That is, there is no doubt of the occurrence of of neurotic ischæmia and hyperæmia. There is no dearth of clinical evidence to prove that œdema may occur through purely nervous influence. Weir Mitchell has recorded several instances of the occurrence of œdema associated with injury of a nerve, and it is not uncommon to see a non-inflammatory swelling develop after stretching of a nerve for a therapeutic purpose. Cases of trigeminal neuralgia and of cranial neuralgia, which are accompanied by œdematous swelling, and transitory œdema associated with some diseases of the spinal cord, such as tabes, although not the rule, are still not uncommon. The cases that develop œdema under hypnotism would seem to be most convincing to prove that psychical and nervous influences are sufficient to cause such swellings. Many of these cases have been reported. A case related by Paschalis (*Gazeta lekarska*, 1890, No. 30) shows clearly the psychical influence. A young woman consulted a fortune-teller, who prophesied misfortune for her client if she went to a certain town. She went, however, but soon after her arrival had severe pain in the arm and forearm, which became œdematous and remained so until she left the town. Experimentally it has been proved that irritation of the peripheral end of the divided lingual nerve will cause œdema of the tongue, and Horsley and Boyce have corroborated Ranvier's statement that after ligation of the iliac vein on one side, œdema does not occur in the limb of that side until after the sciatic nerve has been cut. That the transudation of living fluids through living membranes is not a mere physical phenomenon has been proven by Tigerstedt and Santesson (*Mittheilung vom Physiolog. Lab. des Carol. Med.-Chir. Instit.* in Stockholm, 1886).

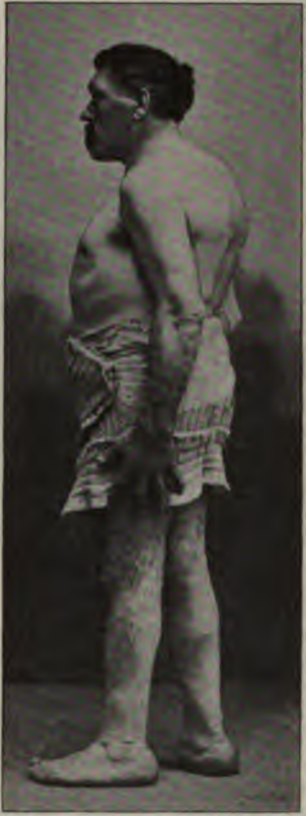
The fact that a neurotic œdema can occur seems to be beyond cavil. The seat of the œdema is probably most often in the connective tissue of the derma beneath the papilla and in the subdermal tissue; very rarely the œdema confines itself to the more superficial parts. The nerves affected are the vasomotor, and the sequence of events is probably a local inhibition of the vaso-constrictors or a reflex stimulation of the vaso-dilators; the result is a retardation of the current and a resulting serous exudation.

The disease in its development has a close relation to other vasomotor neuroses, such as morbid blushing and flushing, and exophthalmic goitre, to many of the arthopathies not yet well understood and particularly to urticaria, which is so commonly an accompaniment of the appearance of the œdema.

Treatment. Unless the cause can be discovered, removed, or counteracted, therapeutic measures are of but little avail either in mitigating the length or the severity of the attacks. If the cause can be discovered, its inhibition and placing the patient on a vasomotor tonic and stimulant will be followed by relief. The verity of this statement is well shown by the patient spoken of previously in whom the disease was the result of the excessive use of tobacco. As soon as the tobacco was interdicted and the patient placed on mineral acids and strychnine he began to recover. As a rule, the greatest success will be obtained by adopting such measures as give tone and stability to the nervous system. As an all-round vasomotor and general tonic to the nervous system strychnine is the best. It should be given in large doses and until its physological effects are manifest, particularly on the spinal cord. The next most important drug in the treatment of the disease is atropine; it should be given likewise in large doses, and its administration continued in small doses during the intervals between attacks. Tonics, invigorating baths, exercise, massage, and the prevention of cold and trauma are the most important factors in the treatment of this disease. If there be an excess of uric acid in the blood, as manifested by a disproportionate relation to the urea in the urine, this condition demands regulating. In these cases the administration of colchicum is often followed by gratifying results. The frequency of the attacks will be diminished by attention to the bowels, digestion, and menstruation. For the paroxysmal hæmoglobinuria and albuminuria that sometimes occur, quietness and low diet should be enjoined. The treatment at the time of an attack will depend somewhat on the part of the body where the disease is manifest. If the dermal surface of the body is the seat of the swelling, the most satisfactory treatment is to keep the patient quiet, in an equable temperature, and apply dry heat to the swelling; if there is much uneasiness a mild anodyne may be given. Compression by means of a bandage or a Gamgee dressing is sometimes of benefit. When the disease manifests itself in the mucous membranes, the treatment is entirely symptomatic. When the gastro-intestinal symptoms are prominent, morphine should be given. When the swelling occurs in the pharynx or the larynx, the physician should be ready to operate at any moment.

Measures devoted to bettering the general health of the patient will be followed by the best results.

FIG. 291.



Case of acromegaly. (OSBORNE.)

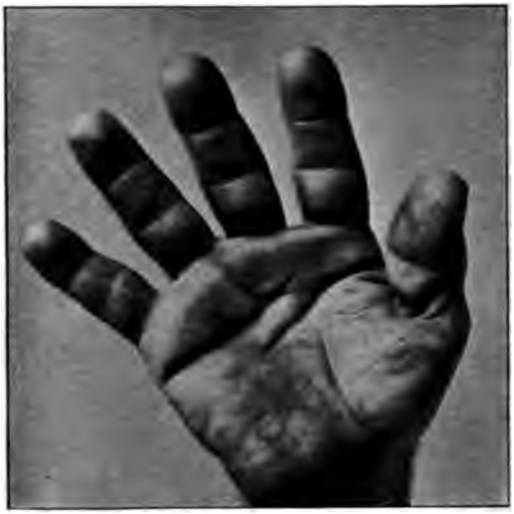
ACROMEGALY.

In 1886 P. Marie, of Paris, described a chronic disease characterized by great increase of the extremities of the body, the hands, the feet, and the face and head, to which he gave the name acromegaly. Since that time the disease has been recognized the world over, and upward of 125 cases have since been reported, in many of which careful post-mortem examinations have been made. Although the most striking changes in acromegaly are to be seen in the extremities, the disease is by no means limited to them; it involves in its progress almost every part of the body. Like a great many other diseases that are seemingly of recent date, acromegaly has been noticed and described for many decades past. A disease which was probably acromegaly was reported in two cases by Friedreich under the title of hyperostosis of the entire skeleton; another by Fritsche and Klebs under the title of gigantism; another by Lombroso as general hypertrophy, macrosmia. Some of the cases that have been published under the heading of myxœdema have been cases of acromegaly, particularly one reported by Henrot (*Notes de Clinique Médicale*, Rheims, 1877.) Souza-Leite thinks that there can be no question but that a case reported by Saucerotte-Noel in 1772 was a typical case of this disease.

The name acromegaly literally means large extremities, and many writers believe that this term is too limited to embrace a disease in which enlargement of the extremities is but one of many striking features. Von Recklinghausen has suggested the name pachyacrie, and he has received the support of Mosler, and more recently Arnold (*Virchow's Archiv*, vol. 135), who says that as the extremities are not only enlarged in length, but from the enormous growth of bone and accompanying changes in the soft tissue the volume is greatly increased, the term pachyacrie would literally and exactly describe the condition of these parts.

Etiology. The disease affects males and females about equally, the tendency being in favor of males. It occurs in every race; cases of it having been reported in the negro, the Indian, and the Mongolian. Although it may develop at any age, it is most frequently discovered between the ages of twenty and forty years. It is probable that the disease begins to develop most frequently shortly after the age of puberty, but unless its course is rapid it is rarely considered other than excessive natural growth at first. It may occur in the first year of life, Moncorvo (*Rev. Mensuelle des Maladies des l'Enfance*, December, 1892), of Rio Janeiro, has recently reported such a case, and it may develop after sixty years. So far three congenital cases have been reported, although Marie has stated that congenital and hereditary influences can be eliminated. The previous health of the patient has seemingly no bearing on the development of the disease. In most of the patients the general health has been good. In a few a history of rheumatism, malaria, or some of the more common acute diseases have been obtained, but they have positively no significance. Among the exciting causes that have been mentioned by more than one writer are mental worry and depression, fright, exposure to cold, injuries, alcoholism, rheumatism and gout.

FIG. 292.



Hand in acromegaly. (OSBORNE.)

It occurs in connection with syringomyelia, with locomotor ataxia, with elephantiasis, hypertrophic osteo-arthropathy, gigantism, and some psychoses. Various theories have been suggested to explain the occurrence of acromegaly, some of which have already been disproven, notably one by Freund, who suggested that the disease was one of development showing itself first about the time of puberty and consisting of a return to a primitive type, with large hands and feet, projecting lower jaw, receding forehead, etc. Klebs suggested that the thymus gland, which he had found in some cases that had come to autopsy, and which Erb thought was indicated by the presence of retrosternal dulness, might be a starting-point of a vascular budding and proliferation with the formation of angioblasts and resulting angiomatosis, which afterward played an important part in the production of the characteristic signs of the disease. It is now conceded that the theory has no anatomical basis, for the comparatively large number of cases that have come to autopsy during the past two or three years do not in any way bear it out. Virchow suggested that in acromegaly we have described but half a disease, the latter and degenerative half; that in the beginning these cases are often accompanied by an increase of muscular power, and that some of them are hereditary. Von Recklinghausen propagated a nervous theory to explain the origin of acromegaly: the disease was essentially an angioneurosis, the primary manifestations being in the bloodvessels, especially the veins, and the result of defective innervation. Many of the symptoms of the disease, such as the excessive perspiration, the polyuria, pigmentation of the skin, and disturbances of menstruation, lend support to this theory. In fact, it may be said that the supposition that the disease is a tropho-neurosis dependent upon or associated with disease of the pituitary gland, and less frequently the thyroid, is constantly being corroborated by autopsies that are carefully performed, in the greater number of which some diseased change has been found.

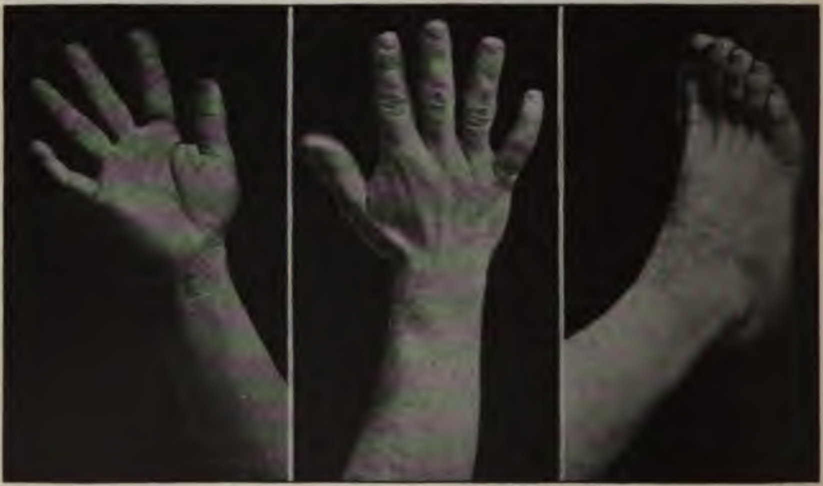
The function of the pituitary gland is not well known, but recent researches, particularly those of Andriezen, Boyce, and others, have thrown much light on this subject. The former investigator has proven that the subneural gland in larval amphioxus is the analogue of the pituitary gland in higher animals and in man. He believes that the ensemble of evidence proves that the pituitary gland is not a simple structure having one simple function, but a complex organ composed of three parts: (*a*) An anterior secreting glandular organ, (*b*) a water-vascular tube lined with ciliated epithelium and connecting the buccal cavity with the ventricles and the rest of the neural cavities, and (*c*) a posterior sensitive nervous lobe. The last two are well developed and functionate in ancestral vertebrata, but become obliterated and atrophied in structure and function in all forms above larval acraniates and ammocetes. In man the posterior lobe represents little beyond a neuroglia remnant of what was once a functional portion of nerve-tissue in ancestral vertebrata. The glandular secreting portion (the anterior lobe is the type of a secreting structure of epithelial cells arranged in lobules and acini with many ducts opening into one principal duct. Its secretion is carried with the water vascular stream through the central nervous system, and the action of that secretion must be either a trophic one on the nervous tissues or it must have a destructive effect to neutralize waste products resulting from the activity of nerve-tissues.

The secretion of the pituitary is needed just as much after the closure of the pituitary duct and the cessation of the water vascular system, for the oxygen which was then provided for the nervous system by the water vascular system is now provided by the blood vascular. Hence the pituitary continues its secretion after the duct is obliterated and the gland is changed into a ductless gland, the difference being that the secretion is an internal one and absorbed by the lymphatics.

The pituitary gland reaches its highest functional development before man is reached in the scale of evolution, but there can be no question that it plays an important part in the economy analogous to that of other ductless glands, such as the thyroid. Clinically and anatomically it has been proven to be diseased in a large number of acromegalics, and the conviction is growing that it is the most important etiological factor.

Symptoms. The symptoms of the disease come on very gradually, and in most cases it is impossible for the patient to tell the time when the disease first showed itself. Very frequently it is recognized first by the physician who sees the patient for some other trouble, or accidentally. The early symptoms are a feeling of general weakness, apathy, frontal headache, which may become very severe, and some dragging pain and paræsthesia in the extremities. In women irregularity of menstruation and in men diminution of sexual appetite and potency are early symptoms. The patient may notice that his desire for food and drink is increased and that he is gaining in weight. In some cases vertigo and disinclination to make any great mental or bodily exertion are early symptoms. The patient may notice that psychically he is not so so alert as formerly, and his friends notice that he is introspective and depressed. Vasomotor symptoms, such as excessive perspiration, increase of urine, and pigmentation of the skin, and a tolerance of cold weather may all be early symptoms. Concomitant with these or following them the patient or some one with whom he comes in contact (his bootmaker, glovemaker, or tailor), notices that there is an enlargement of the fingers and the hands, and simultaneously, or later, a similar increase in the feet and face, while the stature is gradually becoming less. (Fig. 293).

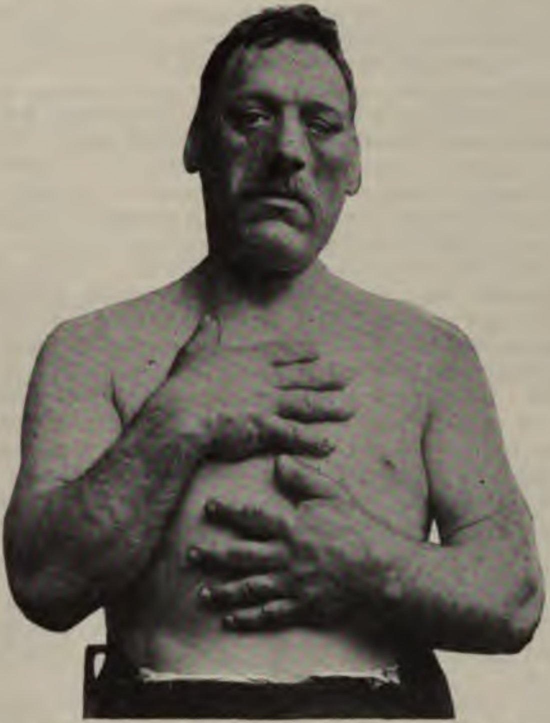
FIG. 293.



Hands and feet in early stage of acromegaly.

If one examines such a patient he will find that the patient has a peculiar posture both in walking and standing; the neck is bent forward, while the head is tilted slightly backward in order to bring the eyes up to a level; the shoulders are rounded and stooping, while the chest is prominent; the gait is non-elastic and heavy. The patient's posture is accentuated by the markedly prognathous lower jaw, which is projecting and heavy. The lips are large, particularly the lower one; the mouth has lost its expression; the naso-labial creases have become fissures; the nose is enlarged, proboscis-like, frequently slightly pigmented, and with wide nasal apertures; the eyes are deeply set and overhung by thickened supraorbital arches on which the eyebrows are coarse and unkempt; the lower part of the forehead is bulging, and this gives a retreating appearance to the upper part; the malar bones and zygoma stand out prominently; the cheeks are flattened; the ears are enlarged; the hair is coarse and dry; the beard is scant; and the skin of the face is dry and frequently pigmented. The expression of the eyes is dull, unanimated, a patient grieving sort of look. (Fig. 294.) The hands are enlarged, but the proportion between the hands and fingers is preserved; the fingers are sausage-shape and the hands resemble a spade. The nails are small, flat, and longitudinally striated. The wrists are enlarged and thickened, which gives the resemblance of the hands to a battledore. The soft parts of the hands are markedly increased, especially along the ulnar side, and the lines in the palms are greatly deepened. All the tissues of the hands are excessively developed, the bones, the muscles, the cellulo-adipose tissue, and the skin. Similar changes are to be seen in the feet. The large toe may be increased out of all proportion to the rest of the foot, and frequently very striking is a pad of thickened skin and cellulo-adipose tissue along the fibular edge of the foot. The foot has lost its arch and its grace, and the increase in the os calcis makes it project backward like a snowshoe.

FIG. 294.



Case of acromegaly, showing facial expression. (DERCUM.)

The spinal column shows a cervico-dorsal kyphosis; the thorax projects anteriorly, and, with the enlarged sternum, gives the appearance of a hump on the chest. The kyphosis and the enlargement of the thoracic cage give rise to a deformity which has been aptly compared by Marie to the figure of Punch. There is frequently a slight degree of scoliosis, and always, if the course of the disease is sufficiently long, a compensating lordosis in the lower dorsal and upper lumbar region. (Fig. 291.)

The mammary glands are flabby, soft, and small; the abdomen is voluminous; and the genital organs in both men and women are frequently the seat of marked changes. In women the labia majora, the nymphæ, and the other parts of the vulva are very greatly increased; the vagina is long and capacious and the uterus presents the changes common to senile involution. In man the genitals are atrophied or in some cases hypertrophied. On the skin of the face and back and chest are frequently found small molluscous growths.

So much can be determined by looking at the patient. If the examination be made with some detail it will be found that none of the special senses with the exception of sight are very much disordered. Smell and taste have been reported defective in but one or two cases. In a few cases the patient complains of tinnitus and gradually increasing deafness, and in these cases a thickened and degenerated membrana tympani has been found. Exophthalmos is a common symptom, and is due both to actual enlargement of the eyeballs and bony growth in the orbital cavities. Narrowing of the visual fields and hemianopsia are reported in a great number of cases. Ophthalmoscopically varying degrees of optic neuritis and atrophy are found. Some optic neuritis, the result of pressure, may be demonstrated very early in the course of the disease, while vision is still normal. Nystagmus and paralysis of the ocular muscles are more common. Disturbance of sensation is rare. The skin is hard and hypertrophied over the extremities, while on the body and face, and especially the arms, there is a considerable deposit of brownish pigment. The muscles, especially of the arms and legs, are in the beginning of good tone and development depending upon the patient's occupation. With a few exceptions, they are never greatly hypertrophied except in the hands and feet. Electrical irritability of nerves and muscles may be slightly diminished, but is, as a rule, normal. After the disease has lasted for a variable time the muscles become soft and flabby, and, occasionally, the unfortunate patient, unmindful of the weakness which goes hand-in-hand with the muscular atrophy, looks upon the thinning of his legs and arms as a good omen. When the patient takes a long inspiration it is noticed that the expansion is almost wholly confined to the inferior portion of the thorax and the abdomen. Percussion of the thorax reveals nothing abnormal, except in some cases a limited area of retrosternal dulness, which Erb supposed was due to the presence of the thymus gland. This supposition has been disproven, and the dulness may be attributed to the increase in thickness of the manubrium.

The visceral organs are, as a rule, normal. The voice is markedly changed in both men and women. It is low pitched, resonant, very voluminous, and of a peculiarly disagreeable intonation. The larynx is greatly augmented in volume, and Marie explains the increased resonance by the dilatation of the sinuses of the face. Speech is slow, guttural, somewhat embarrassed, and seems to stick in the mouth. This is due to the enlargement of the tongue, which may go on to an enormous degree. The tongue is deeply fissured, the alveolar processes enlarged, and the palate thickened.

The urine is generally markedly increased in quantity, occasionally contains albumin, and less frequently sugar and peptones. The appetite is increased, and constipation and hemorrhoids are common attendants. Perspiration is commonly excessive, and often of a most disagreeable odor. Electrical irritability of the muscles and nerves are normal, as are the reflexes, except, of course, a quantitative diminution when the muscles are atrophied.

Mentally the patient is often irritable, depressed, forgetful, and introspective, and presents some impairment of the intellectual faculties; occasionally somnolency is very marked.

The course of the disease is a progressive one, and invariably ends in death, either from exhaustion, commonly the result of intra-cranial pressure, from suicide, or some intercurrent disease which finds an easy victim in the acromegalic. The duration of the disease cannot be stated with accuracy, but is probably from ten to twenty years.

Differential Diagnosis. Acromegaly must be differentiated from myxœdema, hypertrophic osteo-arthropathy, osteitis deformans, leontiasis ossea, elephantiasis, adiposis dolorosa, gigantism, and local hypertrophies. The first two mentioned are of most importance, and the points of difference are contrasted in the following columns:

Acromegaly.	*Myxœdema.*
1. Occurs most commonly in early adult life.	1. A disease of mature life, 40 to 50.
2. In males and females equally.	2. Five times as frequent in females as in males.
3. Enlargement of the bones characteristic.	3. No enlargement of the bones.
4. Marked prognathism of jaw and flattening of cheeks	4. Face full-moon shape.
5. Skin is brownish-yellow; hair coarse and unwieldy; the nails are short and striated.	5. Skin pale, waxy, shiny, and boggy; hair falls out; nails not affected.
6. Fingers are symmetrical and sausage-shaped.	6. Fingers clubbed at the end.
7. The administration of thyroid extract is of the smallest benefit.	7. Thyroid treatment of the greatest benefit.

Acromegaly.	*Hypertrophic Osteo-arthropathy.*
1. Not associated with pulmonary trouble.	1. Always secondary to some pulmonary, cardiac, or otitic disease.
2. Enlargement of the hands and fingers universal and symmetrical.	2. Symmetry of hands and fingers not preserved; joints and ends of fingers principally enlarged.
3. Nails too small for fingers.	3. Nails too large for fingers, and characteristic beak-shape.
4. Cervico-dorsal kyphosis.	4. Dorso-lumbar kyphosis.
5. Increase of bony and soft tissue.	5. Increase in bony tissue alone, especially at the articular ends of long bones.
6. Lower jaw markedly prognathous.	6. Lower jaw not particularly prognathous.
7. Eye symptoms common.	7. Eye symptoms rare.
8. Mentally depressed and introspective.	8. Mentally hopeful.

Arthritis deformans, leontiasis, and elephantiasis will rarely be confounded with acromegaly if the latter is carefully studied. In leontiasis ossium there is a growth of true bony structure in the shape of tumors of the cranium and face, and generally an absence of hypertrophy of the extremities. In arthritis deformans the changes are principally in the joints of the extremities, the face is rarely affected. Elephantiasis consists of cystic and tubular enlargement of the lymphatics, first of the cutaneous structure, then of the more deeply-seated tissues, attended by thickening and induration of the skin and connective tissue, and dilatation and multiplication of the blood-vessels, with wasting of the muscles and absence of changes in bony structures. In osteitis deformans the increase in bone tissue is most marked in the cranial bones. The bones of the face are but slightly affected. The long bones are principally involved, and often show great curvature and deformity. The hands are not commonly enlarged, and the lower part of the face is pointed. The differential diagnosis of gigantism, adiposis dolorosa, and erythromelalgia will readily suggest itself. It should be kept in mind that acromegaly is frequently seen in giants, such cases having been reported by Dana, Bramwell, and Taruffi.

Pathology. The most constant organ to show disease in acromegaly is the pituitary gland. In a large proportion of the autopsies that have been made this organ has been found to be the seat of tumor, cyst, hypertrophy, or in some way diseased. Very much less frequently the thyroid gland is diseased; cystic, enlarged, or atrophied. Very rarely is the thymus found diseased. The characteristic pathological changes are to be seen in the bones, particularly in the extremities of the long bones. The growth of the bones is a true hypertrophy, the increase taking place from the periosteum. The long bones may apparently lengthen from a laying on of bony structure at their extremities, while the shaft is not at all affected. The bones of the cranium are thickened, and new bone develops in the diplöe and in the outer and inner plates. The frontal and sphenoidal sinuses are greatly dilated, the sella turcica is generally deepened and enlarged. All the bones entering into the formation of the parts already described as enlarged, show the hypertrophy. The blood-vessels are frequently increased in calibre, and their walls thinned, and this is especially noticeable in the veins. An increase of neuroglia cells of the brain and hypertrophy and sclerosis of the sympathetic ganglia are occasionally found. The skin is hypertrophied, particularly over those parts where the enlargement has been the greatest, the cellulo-adipose tissue is increased, and the muscles are generally atrophied.

The bodies of the vertebræ are generally enlarged, especially in the anterior portion, and the intervetebral cartilages are thickened. The sternum, the costal cartilages, the clavicle, the scapulæ, and pelvis all show varying degrees of hypertrophy. The visceral organs are, as a rule, normal, or show only the lesions of intercurrent diseases.

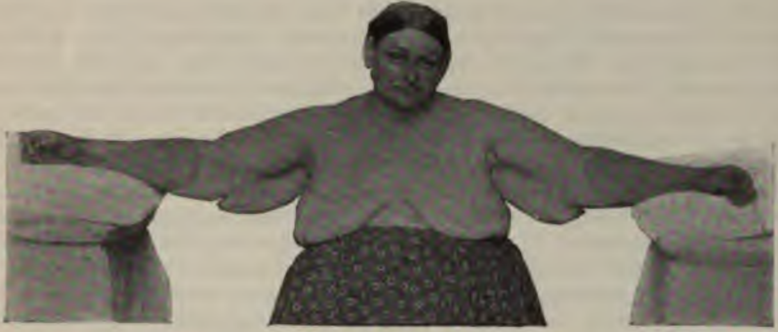
Treatment. The treatment is entirely symptomatic; measures devoted to keeping up the general strength of the patient should be taken. The headache and pains are best combated by the use of antipyrin, phenacetin, and arsenic. The thyroid extract has been used empirically. In a case now under my observation the patient has been taking it for upward of a year without the slightest degree of benefit except that it has exercised a cosmetic action on the skin, which has become softer and more pliable. The patient has also lost flesh. It may be remarked in this connection that the thyroid extract seems to have a particular power to reduce obesity, and by so doing it may at first seem to exercise a beneficent action on the disease, but even this is temporary and fleeting.

ADIPOSIS DOLOROSA.

Under the name adiposis dolorosa, Dercum, of Philadelphia, has described a peculiar dystrophy occurring in adult persons characterized by the deposition and formation of fat in various parts of the body, first in the form of bunches or nodules, later uncircumscribed, and attended with pain and frequently diminished cutaneous sensibility and excessive muscular weakness. No other cases than those cited by the first writer have yet been put on record, but cases have been observed by other clinicians, Dr. F. P. Henry having described a case under one title of myxœdematoid distrophy. Dr. Frederick Peterson and Dr. B. C. Loveland have put at my disposal the notes of six cases which they have had opportunity to carefully study. With the exception of one case seen by Peterson, all the patients have been in women from forty to sixty years old. Another suggestive factor in their histories is that in none of them, except in one case observed by the writer, can a specific or alcoholic history be ruled out. In the family history a neuropathic predisposition is evident in all the cases. Further than this nothing can be said of the etiology.

The patient first notices an enlargement of some part of the body, the ankles or legs, the arms or shoulders, or the trunk. This enlargement is apt to show itself in the shape of nodules or a more or less limited enlargement; these may for some time limit themselves to the original areas, but it generally increases gradually in size, and eventually the deposit makes its appearance elsewhere and may become very extensive. Regions of the body may exist which remain permanently uninvolved. The onset of these swellings is coincident with such nervous symptoms as paræsthesiæ, sense of coldness in the part affected, dull, aching pain, and as the fatty accumulation increases and becomes more universally distributed there is a considerable loss of muscular power and lessened cutaneous sensibility, which may go on to complete anæsthesia, especially in parts where the subcutaneous accumulation is greatest. The pain in these cases is a very variable quantity; in some cases it has been paroxysmal, of a burning, scalding character, while in others it is present only when the parts are subjected to pressure. The appearance of the enlargements is in no way characteristic. If it shows itself first about the ankles the parts will appear puffy, but pressure shows that it is not in the least œdematous; the skin is white, soft, flexible, not thickened, and except that it is very dry, feels quite normal. When the bunches of fat develop first on the upper extremities or body they are more or less well defined, and as they continue to increase in size they form huge pendulous masses. (See Fig. 295.) To the touch they are somewhat elastic, comparatively firm, yet withal have a pultaceous feeling, which Dercum says resembles the feel of a large varicocele, except that it is more resistant. These swellings may reach an enormous size, so that combined with the muscular weakness and pain, which movement frequently causes, they serve to make locomotion very difficult and eventually impossible. The fatty accumulations have not been noticed in the hands, face, or feet, and frequently the contrast between the feet which preserve their normal outline and contour and the legs, when the latter are involved, is most striking.

FIG. 295.



Adiposis Dolorosa. (DERCUM.)

Symptoms that were present in some of the cases observed, but not in all, were headache, occasional attacks of herpes zoster, attacks of hæmatemesis and epistaxis, early appearance of the menopause, and in one case the recurrence of the uterine flow many years after cessation of menstruation.

Trophic symptoms, such as pigmentation of some parts of the skin, atrophy, and reaction of degeneration in the thenar and hypothenar muscles, and absence of perspiration, occurred in two cases. A lessening of the patellar and triceps reflex has been observed, but this was probably due to mechanical interference. In four cases there was progressive mental weakness, and in one of these cases complete dementia resulted. Two cases have come to autopsy, but the nervous system was not investigated microscopically in either one. In both cases the most remarkable deviation from normal, aside from the fatty accumulations, was a change in the thyroid gland; in one case it was enlarged, in the other diminished in size, but in both cases it was infiltrated by calcarcous matter. Otherwise the autopsies showed nothing but the lesions of the disease from which the patient died, commonly bronchitis, pulmonary œdema, and degenerate heart.

Dercum examined microscopically portions of the fatty accumulations which he obtained in two of his cases by means of a Duchenne trocar. The examination revealed connective tissue and fat cells present in varying degrees. The former was decidedly embryonal in type, the cells being large and fusiform, and their nuclei correspondingly large and prominent. The fat cells for the most part were associated with these connective-tissue cells, and occasionally individual fat cells were seen in which fatty metamorphosis had not been complete. In the more recent cases the embryonal connective-tissue cells were considerable, while in the older areas a fully formed adult fatty tissue seemed to be present.

What the nature of the disease is it is impossible to say. Dercum originally described his first case by the title "A Subcutaneous Connective-tissue Dystrophy of the Arms and Back associated with Symptoms resembling Myxœdema." The fact that the thyroid gland has been found diseased in the two cases in which a post-mortem has been made is important in view of what we know is the dependence of myxœdema upon disease of this organ. Dercum, however, is of the opinion that as these cases lack the peculiar physiognomy, the spade-like hands, the infiltrated skin, the peculiar slowing of speech, and a host of other symptoms found in true myxœdema, that we are not dealing with aberrant forms of this disease. In his opinion we have to do with a connective-tissue dystrophy, a fatty metamorphosis of various stages of completeness, occurring in separate regions, or at best unevenly distributed, and associated with symptoms suggestive of a fugitive and irregular irritation of nerve-trunks—possibly a neuritis. That this, however, does not embrace the whole truth is evidenced by such symptoms as the diminished sweating, the headache, and contraction of the visual fields. To Peterson it seems that the pathological condition underlying this disease is a rudimentary polyneuritis, with a hyperplasia of connective tissue and a fatty infiltration of connective-tissue cells, and with this view the writer is in fullest accord. The fact that syphilis or alcoholism is a prominent factor in all of the patients' previous histories would lend color to this view, and it is quite probable that microscopical examination of the nerves in future cases will show the presence of some change in their peripheral nerves.

The disease has to be differentiated from myxœdema, acromegaly, general obesity, and elephantiasis; but in view of the fact that it is because this disease does not tally with the clinical picture of any of these diseases that led to its being set apart for separate description the diagnosis will not be difficult. It is diagnosed from lipomatosis perimuscularis circumscripta by the fact that the latter is painless.

The disease does not tend to spontaneous cessation or to recovery. All of the cases have been of a progressive nature. The indications for treatment are to improve the nutrition by means of change of climate, water therapy, electricity, measures that contribute to excessive oxidation, the administration of strychnine and massage. In the light of what has been said in a previous connection of the efficacy of thyroid therapy to reduce simple obesity, and of its really marvellous properties in many cases of myxœdema, this is a form of treatment that should be given a thorough trial.

SCLERODERMA.

Scleroderma is a disease resulting in a diffuse or circumscribed induration and atrophy of the skin and subcutaneous tissue. It has been variously described as "hide-bound" disease, scleroma adultorum, sclerosis, chorionitis, elephantiasis sclerosis, etc. Clinically, two forms of the disease are distinguished: (1) Diffuse symmetrical scleroderma, and (2) circumscribed scleroderma, more commonly known as morphœa or Addison's keloid. Formerly, morphœa was considered a disease apart from scleroderma, but at the present time almost all writers on the subject are in accord that it is a circumscribed variety of scleroderma. There are others, however, who deny this.

The most characteristic change in scleroderma is first an infiltration giving rise to a hard swelling in the beginning, which is frequently preceded by vasomotor disturbances, and later the absorption of the infiltrated tissue and the occurrence of a scar-like atrophy. That the disease is a very rare one is shown by the statement of Croker, who says that in his wide experience with skin disease he has treated but five cases.

Etiology. Nothing positively is known of its actual causation. The theory that it is a vasomotor neurosis receives most support. The etiological factors of importance seem to be sex and previous infectious disease. About three-fourths of all the patients are females. It may occur at any age, from one year to seventy, but the majority of patients are in the second, the fourth, and the sixth decennium. It occurs after infectious diseases, such as erysipelas, scarlatina, pneumonia, malaria, tuberculosis, and rheumatism. The time elapsing between the activity of any of these diseases and the manifestations of scleroderma is a variable one, but usually it is not so long but that a relationship can be traced. Other factors that seem to have a causal relationship are first of all exposure to cold and extreme temperatures, trauma, anomalies of constitution, such as anæmia and scrofulosis. Psychical influences, grief, anxiety, and worry are often present. An infectious origin, as was suggested by Hoppe-Seyler, cannot be conceded. It occurs almost always in neuropathic individuals and occasionally in those who are suffering from some nervous disease, such as syringomyelia, chronic myelitis, Raynaud's disease, and disease of the brain.

Symptoms. The disease is frequently first noticed after exposure to cold and wet or fatiguing and exhausting influences. Pain in the joints and extremities may precede for some time the feeling of stiffness in the skin. This feeling of stiffness is most commonly felt at the back of the neck, the shoulders and arms, the face and scalp. The lower extremities are affected very rarely. The onset of the stiffness is insidious and unattended with constitutional manifestations. Its progress may be either slow or rapid. When sclerodermatous induration has reached its height the skin of the affected region is somewhat increased in volume and of leather-like thickness, and as it involves the skin over the joints these become fixed as the skin becomes rigid. The line of demarcation between the healthy and the involved skin is not a closely defined one either to the eye or to the touch, for the one merges gradually into the other. Attempts to pinch up the skin or to produce pressure on pitting are not successful, but when the finger is quickly and firmly drawn across the surface a whitish line with a pink border, which disappears slowly, is left behind. When the skin of the face is affected all trace of expression is obliterated and the regular features take upon them the white, set straightness of a death mask. The mouth can scarcely be opened, the alæ of the nose are bound, and the eyes nearly closed or drawn wide open. When the skin over the chest is involved, the latter is flattened, the breasts compressed so as to be quite effaced, and respiration is often seriously impeded. Involvement of the skin of other parts of the body produces more or less fixation depending upon the parts affected. The head may be drawn up and almost immovable from affection of the skin at the back of the neck, the upper extremities may be fixed and incapable of flexion or extension, and the process may become so extensive that the patient is in a state of more or less complete rigidity.

The appearance of the skin differs in different patients and in different stages of the disease. It may be so white that it resembles marble, or it may be mottled, pigmented, striated, and the natural lines obliterated. The presence of subcutaneous nodules similar to those sometimes found in rheumatism and in chorea occasionally pushes the skin up and gives it an uneven appearance, particularly over the bones. Paræsthesia, pruritus, hyperæsthesia in the parts affected are not infrequent, although, as a rule, there is no defect of sensibility. The secretions of the skin are not notably diminished. In rare instances the more exposed mucous membranes, such as those of the vagina, the mouth, pharynx, etc., are affected.

After the disease is fully developed recovery may take place without leaving any trace, although, as a rule, it may be said that it shows no tendency to spontaneous cure; or atrophy of the parts may progress. Although this atrophy is symmetrical it often causes deformities. An extremity may be reduced almost to skeleton size, the joints fixed, and even wasting of the bones may occur. To the deformity occurring in the hand due to flexion and extension of the phalanges and fingers, Ball has given the name sclerodactylie. The lips may become as thin as ribbon and unyielding, the teeth fall from atrophy of the gums, and the eyes may be uncovered from contraction of the orbicularis.

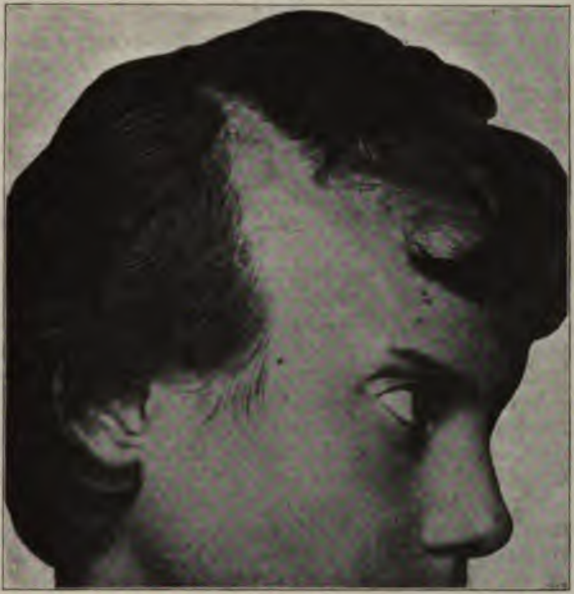
The course of the disease may extend over several years. As has been said, during the stage of hypertrophy recovery may occur, but after the atrophic state is fully developed recovery never results. Although the disease does not itself cause death, it predisposes to acute disease, such as rheumatism and acute inflammations of the respiratory tract, and these, because of the emaciation and depression of vitality of the patient, are very apt to lead to a fatal termination. When Raynaud's phenomenon complicates this disease, which it does not infrequently, the prognosis is bad.

At the present day there is little doubt that morphœa (μορψη, form or blotch) is a circumscribed variety of scleroderma. It presents itself in the form of patches, bands, or streaks. These patches vary in size from the end of the finger to the palm of the hand. They present themselves gradually without attracting the patient's attention, and as they often develop on parts of the body that are not easily seen, such as the back of the neck, the patch frequently obtains its full development before the patient is cognizant of its presence. In color they are dead-white or yellowish, bordered by a pinkish zone, due to minute dilated bloodvessels, which may be made out by careful scrutiny. The patches are usually limited to one side of the body and often confined to the distribution of one or more nerves, as in herpes zoster, and on account of this parallelism Hutchinson has suggested the name of herpetiform morphœa.

They are especially apt to occur on the breasts and on the face over the distribution of the fifth nerve, particularly its supraorbital branch (Fig. 296). Unlike the diffuse form, it is more common on the lower than the upper extremities. On the lower extremities the surface to which the terminal twigs of the short saphenous nerve are distributed is a favorite seat of morphœa. The affected skin feels like parchment or leather depending upon the amount of infiltration, but it is not so hide-bound as in symmetrical scleroderma. The subjective sensations complained of do not differ materially except in intensity from those of the more diffuse form. When this form of scleroderma occurs in bands it is usually single and raised up into a sort of a ridge, or if adherent to the adjacent tissues it is sunk into a sulcus. It may stretch itself the whole length of an extremity or it may extend across the forehead or nose, and, from a superficial examination, it is easily mistaken for a scar. The course of this form of the disease is usually slow, but in some cases a patch may be evolved in a few days. As in the diffuse form, it may disappear and the skin assume its natural appearance, or it may go on to contracture.

In morphœa the potency of neurotic influences in causing the disease are more apparent than in the diffuse form, but aside from this the etiology is not materially different. In some cases pressure or local irritation, as from a garter, seems to determine the location of the affection.

FIG. 296.



Morphœa. Traumatic origin. (CANTRELL.)

Pathology. Anatomically the disease is characterized by a proliferation of connective tissue and by vascular changes which eventuate in a fibrous peri-, meso-, and endarteritis. The changes in the bloodvessels precede those in the skin and are causative of the latter. That this disorder of the bloodvessels is probably a vasomotor one, and that the central nervous system as well as the sympathetic is involved is shown by its association with diseases of these parts, by the distribution of the circumscribed form, and, particularly, by treatment which is most successful when the measures are directed to the vasomotor system. The thickening of the nerve-sheaths and fatty degeneration of nerves found in parts affected with scleroderma are secondary to the vascular changes. The result of thickening of the vessels is to interrupt the circulation, and this causes the swelling and hyperplasia of the first stage of the disease. If this impairment of circulation continues long enough it leads to depravity of nutrition and atrophic changes. In the circumscribed form the changes in the bloodvessels are less severe and is greatest at the centre of the patch, while at the periphery or border of the patch there is a dilatation of the capillaries, and to this can be attributed the pinkish zone often seen at the border of the blotch. The morbid anatomy of the sclerodermatous process shows that the epidermis is of normal thickness; the striatum corneum is seen to be composed of a few layers of flattened cells; the papillæ and appendages of the skin are normal, but their connective tissue envelope shows extensive small cell infiltration; a similar condition is to be seen in the subpapillary bloodvessels. The upper and lower layers of the corium show a considerable connective tissue, the individual fibrillæ of which are distinctly divided into sections.

The diagnosis of scleroderma, either of the circumscribed form or the symmetrical, offers no difficulties if the disease is fully developed. The striking swelling and hardness of the skin, its hide-bound immovability, the peculiar color of the diseased areas in the first as well as in the last stage, and the fixation of parts affected, are pathognomonic. The condition from which the circumscribed form of morphœa is most difficult to differentiate is hemiatrophia facialis. Both diseases are tropho-neuroses and often affect similar areas, and undoubtedly many of the cases reported under the former heading are quite as properly described under the latter. The two diseases are, however, distinct. In the former all the tissues are affected primarily; in the latter atrophy of the deeper tissue is secondary to pressure. In the former it is very probable that the pathological cause of the disease is defect of development or disease of the fifth nerve.

Treatment of these cases is not considered so hopeless now as formerly. The object of treatment should be to invigorate the patient, to pay especial attention to the vascular system, and to obviate factors that are known to aggravate the disease. Careful attention to diet, exercise, and hygiene, combined with the administration of iodides and arsenic and cod-liver oil, will best meet the first indication. Electricity by means of the constant current, salt-water baths, followed by an application of a 2 per cent. salicylic vaseline, will meet the second, and a salubrious and equable climate for the diffuse form, and a careful avoidance of exposure and chilling in the circumscribed, will best obviate the causes that aggravate the disease.

Lustgarten has referred to one case of general scleroderma under his observation which was cured by the use of the thyroid extract. Sachs has also seen a case of the diffuse form of a severe type and of many years' duration in which the thyroid extract has done so much good that the patient is able to do her housework, sew, etc. Much has been claimed for massage in the treatment of the generalized form, and undoubtedly in some cases it does good, particularly if combined with hydrotherapy in the most approved forms, but great care is necessary not to allow the massage to act as an irritant; if it does it becomes an agent for harm, instead of good.

PROGRESSIVE FACIAL HEMIATROPHY.

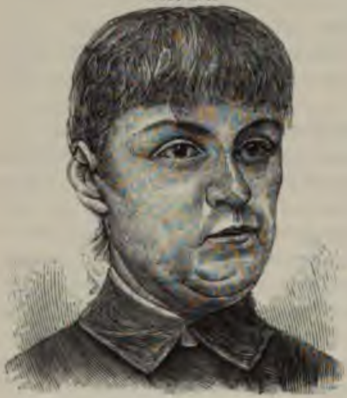
As was stated above, the disease that so closely resembles morphœa of the face that many physicians, particularly dermatologists, believe them to be the same disease, is hemiatrophia facialis. Since this disease was first described by Parry, in 1825, it has been known by such names as facial trophoneurosis, prosopodysmorphie, neurotic atrophy of the face, aplasie lamineuse progressive, and atrophie du tissu conjonctif. The name, progressive facial hemiatrophy, carries a clear conception of the disease from a clinical standpoint. The disease is, as its name implies, a progressive wasting of one side of the face, in which all the structures participate in a varying degree; the muscles least, and the skin, cellulo-adipose tissue, and bone most of all. Although the disease has been extensively written on during the past half century, since Romberg, in 1846, gave an explicit account of its symptomatology, the number of cases reported is still very few and the disease correspondingly rare. In an extensive search of the literature I have found records of but 126 cases.

Etiology. The disease occurs in neuropathic individuals by preference, and more frequently in females than in males. Youth is the favorite period for its development, although cases have been reported in young children and after sixty (Borgherini). Direct heredity has been determined in two cases. In Seeligmuller's case the mother of a nine-year-old child with hemiatrophy was similarly affected. Local trauma, such as a squeeze of the face and head between two doors (Skyrme), a blow on the side of the face (Fromhold-Treu, Maragliano, Schuchardt), has been attributed in a number of instances. Exposure to cold and irritation of the side of the face—such, for instance, as an abscess behind the ear (Preobrashenski)—has been thought to have some etiological influence. In one instance the disease developed immediately after incision and probing of a phlegmonous dacrocystitis (Borgherini), and in another after an inflammation of the submaxillary gland, which had been associated with angina (Baerwald). In a few cases one of the infectious diseases—influenza, erysipelas, scarlatina, and typhoid —has preceded the onset of symptoms. It occurs frequently in people who have some degenerative nervous disease, such as hysteria (Parry, Nothnagel), epilepsy (Meyer, Brunner), tabes (Jolly), insanity (Mendel), multiple sclerosis (Jolly), and syringomyelia (Schlesinger, Déjérine, and Miraillia). It has occurred in connection with multiple exostosis of the head and face (Karewski), with localized and diffuse scleroderma in other parts of the body (Nixon, Muratow, Rosenthal, Newmark), with pityriasis rubra (Graebe), and with Addison's disease (Schulz). It may occur with disease of the fifth nerve.

Symptoms. After any of the above etiological factors, or without any apparent cause, the patient notices one or more whitish spots on the side of the face, generally on the lower jaw or near the orbit, which gradually become larger, or, if more than one, they coalesce and become yellow or yellowish-white in color. The skin over these patches or areas sinks and forms pits or trough-like depressions, due to the disappearance of the subcutaneous fat. The skin may become pigmented, not only in the areas where the atrophy is marked, but on other parts of the face. This pigmentation is in small spots or patches, and varies in color from a grayish-yellow to brown or blue. The atrophy involves the skin, both in its dermal and epidermal layers, its appendages, the cellulo-adipose tissue, and bone, and the muscles to a very slight degree. The hair of the beard, of the head, and the eyelashes may lose its color and fall out in patches, or the areas of alopecia may be quite symmetrical. The sebaceous glands are atrophied and their secretion diminished and eventually checked. The secretion of perspiration is normal, frequently increased. After a variable time the entire half of the face becomes involved, and a deep fissure like a sabre-cut in the centre of the forehead and chin separates it from the healthy side. This fissure or depression has been considered one of the landmarks of the disease, but Borgherini has recently reported a case where the disease involved both sides of the face, and there was no difference between the two sides. When the disease is well developed, shedding of the epidermis and preservation of the contractility of the muscular fibres of the skin give the latter a roughened, puckered appearance. The atrophy may involve the tongue, the hard and soft palate, the gums, and the uvula. In the face it may become so extensive that the diseased side looks like an appendage to the normal side.

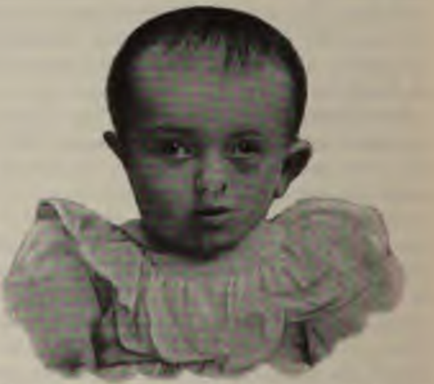
As a rule, there are no marked disturbances of sensibility. A slight degree of hyperæsthesia has been reported by Tanturri and by Vulpian, but excessive sensibility is more common. Neuralgic pains preceding the appearance of the disease and during its early course are by no means uncommon, although formerly so considered. Itching of the skin is sometimes a distressing symptom, and occurs when the disease is associated with patches of scleroderma in other parts of the body. The tension of the atrophying skin may give a sensation as if a rubber mask was stretched across the face. An uncommon symptom that has been observed by Axmann and Heuter preceding the development of the disease, and by Sachs, Muratow, and Fromhold-Treu during the disease, is a spasm of the buccinator and masseter, rarely of the temporal muscles. (Fig. 297.) These twitchings may be clonic alone, or clonic followed by a tonic condition, which lasts for a few minutes, and during which time the patient cannot separate the jaws. Sometimes it extends to the tongue and interferes with its functions. When these twitchings occur they are generally the cause of a good deal of pain, and are made worse by exposure and by excitement. As the disease progresses, disappearance of the fat in which the eyeball is imbedded gives the patient a painful appearance, due to enopthalmos. The special senses show deviation from the normal only in rare instances, taste and hearing being impaired somewhat more frequently than sight. Occasionally a unilateral myosis or pin-point pupil shows the influence of the sympathetic, and is present with other symptoms referable to that part of the nervous system, such as difference in surface temperature of the two sides of the face (Sachs, Seeligmuller), pallor or flushing of the face, hemihyperidrosis, or absence of perspiration on one side of the face, and contraction of the nostril. With the exception of those cases in which the sympathetic seems to be involved the bloodvessels retain their normal tone and calibre. In the advanced stages of the disease the stretched skin presses upon the vessels and interferes with their response to vasomotor influences, and the result is a continual pallor, inability to blush, and loss of vascular response to electrical stimulation. Extremely rare symptoms are neuroparalytic ophthalmia and difficulty in swallowing. Marie and Marinesco have described a case of hemiatrophy of the face and upper limb, with facial paralysis of the same side, which apparently does not entirely correspond with the description given above, insomuch as the skin was not changed in thickness, consistency, or color. They regard the phenomena which their patient presented as due to changes in the domain of the great sympathetic.

FIG. 297.



Hemiatrophia facialis. (SACHS.)

FIG. 298.



Facial hemiatrophy in an infant under the care of Dr. Dercum. (Jefferson Medical College Hospital.)

The course of the disease is generally rapid in the beginning up to a certain stage; then its progress may apparently cease for a long time, to be followed sooner or later by a period of exacerbation. The prognosis as regards recovery is unfavorable; the first case of such a desirable termination is not yet on record. It must be said, however, that the disease does not tend to shorten life, and so far cases have not come to autopsy except in two instances. The findings in both these cases are most suggestive to a proper interpretation of the pathology of the disease. The case of which Mendel was fortunate enough to obtain an autopsy had been under observation by Romberg, Remak, and Virchow, and is well known in the literature, and an analysis of the symptoms of this case had been used to fortify the theory that the disease was a neurotrophic one. The patient had during twenty-five years presented the symptoms of typical facial hemiatrophy complicated by atrophy of the left upper extremity. Erysipelas during a confinement had immediately preceded the development of the disease originally. A minute examination of the entire nervous system revealed a proliferating interstitial neuritis of the left fifth nerve, most marked in its second branch. The seventh nerve was entirely normal, while the left musculo-spiral had undergone changes similar to those in the left fifth nerve. Centrally, an atrophy of the descending root of the fifth nerve and a partial atrophy of the substantia ferruginea was all that was of importance. In Homen's case a tumor of the dura mater, which pressed upon the Gasserian ganglion and the branches of the trigeminal nerve caused a hemiatrophy of the face and tongue, accompanied by anæsthesia of the region affected and paralysis of the oculo-motor nerve. On autopsy it was found that the tumor caused a degeneration of the nerve, especially of its sensory portion, the part corresponding to the posterior-root nerves and in part of the third nerve, and microscopical examination revealed the phenomena of degeneration in all the branches of the fifth nerve and some of the seventh, third, fourth, and sixth nerves. These two cases are not sufficient to warrant us in saying that the pathology of the disease is lesion of the fifth nerve, but in view of the fact that the only cases in which autopsies have been made have shown lesion of this nerve, and moreover, when they corroborate the most plausible theory that has been propounded, they are very suggestive.

Experimental evidence to prove that hemiatrophy may result after injury to the fifth nerve is not wanting. Girard· has seen hemiatrophia facialis, atrophy of the muscles of mastication, thinning of the hair, atrophy of the bones and face, and partial atrophy of the tongue follow section of the posterior root of the trigeminus at the base of the skull. He concludes, as most investigators before him have concluded, that the trophic fibres of the trigeminus are in the posterior root. He believes, further, that the seventh nerve plays no rôle in the development of hemiatrophy. Schiff, however, has proven that experimental atrophy involves not alone the muscles of mastication, but also the other face muscles. These facts, taken together with distribution of the atrophy and the various arguments that can be adduced to show that the disease is neurotic, go far to corroborate the view that the affection is a tropho-neurosis dependent upon lesion of the fifth nerve. There is much clinical evidence to suggest that the disease can be due to other causes, particularly affection of the cervical sympathetic. Seeligmuller has described a case in which the wasting followed injury to the cervical sympathetic of the same side, and Popoff thinks his case can be explained in no other way. But the theory suggested, a compression of the arteries going to the bones by the cutaneous and subcutaneous wasting, to explain the mechanism of these cases, is not in accord with the teachings of physiology. The vasomotor symptoms that sometimes occur with the disease can be explained by affection of the fibres of the sympathetic that run in the fifth nerve.

The disease can only be confounded with congenital asymmetry, morphœa, and possibly facial paralysis. In congenital asymmetry there are other conspicuous somatic and mental defects which will quickly make the diagnosis. Its relationship to morphœa has already been spoken of. Only the most careless observer could mistake it for an ancient case of facial paralysis.

Treatment. As the disease is incurable, preventive treatment, if possible, in the shape of removing the cause, is of greatest importance. Basing his suggestion on the fact that section or excision of the fifth nerve for the relief of neuralgia is not followed by any atrophic change, and upon the hypothesis that hemifacial atrophy depends, not so much upon a failure of trophic nerve stimulus as upon a perversion of that stimulus, Dercum has suggested early resection of the branches of the trifacial. There are no records to show that this plan has ever been tried. The use of galvanic electricity helps to stay the progress of the disease in some instances.

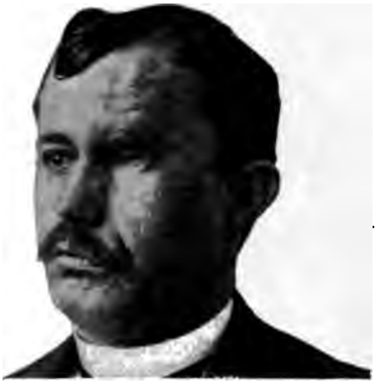
To overcome the cosmetic defect when the disease has reached a standstill the patient may have a plate constructed by a dentist which can be attached to the teeth of the upper jaw on the affected side and with a slightly convex surface against the mucous membrane of the cheek. This can be worn with comfort, and detracts remarkably from the unsightliness of the deformity.

FACIAL HEMIHYPERTROPHY

Scattered throughout medical literature are a number of cases reported in which the only departure from normal is the enlargement of one-half of the body or a segment of one-half. These cases are in some instances associated with other diseases, but, as a rule, the hypertrophy is the only pathological condition. The parts of the body that are most frequently the seat of unilateral hypertrophy are the extremities, the cephalic, and digital.

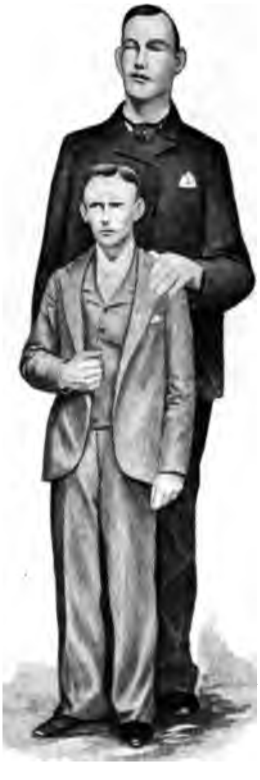
Unilateral hypertrophy limited to the head and face occurs less frequently than hypertrophy of one-half of the body. It is very much rarer than its analogue and opposite, progressive facial hemiatrophy. The majority of cases on record are of congenital origin, although cases have been reported by Schieck, Berger, Dana, and Montgomery and Thomson in which the overgrowth began during the first years of life, always before puberty. In some of these cases irritation of the fifth nerve would seem to be the initial process in the disease. In Berger's case the overgrowth followed an inveterate neuralgia of this nerve. In Montgomery's case it was thought possible to attribute the onset of the growth to an irritation of the branches of the fifth by an abscess of the cheek, which was present shortly before the progressive unilateral enlargement of the face was first noticed. In Dana's case the disease was associated with gigantism. Of 15 cases selected from the literature, 9 were males and 6 were females. Both sides of the face are involved with equal frequency, the proportion being slightly in favor of the right side. The enlargement may involve the entire side of the face and head, or it may be manifest principally in the eyebrow, the zygoma, the cheek, or the angle of the jaw; generally, however, it confines itself very closely to the distribution of the fifth nerve or one of its branches. If the condition is not congenital, the first signs of the disease will pass unnoticed, as a slight degree of facial asymmetry is not uncommon in individuals who are considered normal. Ordinarily the enlargement begins first in the alveolar eminence of the superior maxillary, in the supra-orbital arch or in the malar process. It involves the skin and subcutaneous structure as well as the bone, and, although limited to the side of the face, it may involve the ear, the temple, lips, lids, buccal cavity, the mucous membrane of the mouth and cheek, the teeth, gums, tongue and tonsils. When the hypertrophy is fully developed the patient presents a striking appearance, and at first glance the condition may be taken for hemiatrophia facialis on account of the disproportion between the two sides. The expression of the face is changed, the mouth is distorted, the fissures of the face are deepened on the affected side. The skin, although sometimes unaffected, generally participates in the thickening, the color may be normal, although in some cases it is pigmented ; very rarely does it betray any evidences of hyperæmia. In one case reported the skin was rough, coarse, thickened, and in some places even tumefied, and somewhat darker than on the opposite side. The hair occasionally participates in the evidences of overnutrition, and the lanugo as well as the bristling hairs become coarser on one side of the head and face than on the other. The surface temperature is equal on both sides; this in contrast to cases of hemihypertrophy of the body, in which considerable deviation of temperature of the sides has been noticed.

FIG. 299.



Hemihypertrophy of face. (MONTGOMERY.)

FIG. 300.



Facial hemihypertrophy in a giant. (DANA.)

Occasionally glandular secretion, perspiration, and saliva, are increased ; this has been observed most frequently in congenital cases. There are no disturbances of sensibility except in those cases in which the skin is very much thickened ; tactile sense may be somewhat blunted (Lewin). In a case reported by Friedreich there was diminution of the sense of taste and sight, but whether or not there were ophthalmoscopic findings to explain the disturbance of vision is not stated. In cases where the eyes have been examined carefully no defects have been found. In a few cases that have come to autopsy an examination of the affected tissues hat been made, but no investigation of the nervous system. The morbid phenomena in the bones is essentially an hyperostosis with enlargement of the foramina and broadening and widening of the natural fissures and crevices. In some cases this hyperostosis is uniformly distributed ; in other cases it has a tendency to develop in masses which may cause pressure on important structures, and so produce symptoms. In Thomson's case pressure on the brain caused epilepsy and consecutive mental deterioration. The muscular tissue, instead of being enlarged, is atrophied and the thickness of the soft parts is due to an increase of cellulo-adipose tissue. The skin is sometimes involved, but the epithelium is normal.

In instances where portions of the tissue has been excised for microscopical examination during life, the bloodvessels have shown round-cell infiltration. The pathology of the disease is based on theory. It is supposed that we have to deal with a neurotic hypertrophy, a hypertrophy that is secondary to irritation of certain trophic fibres contained in the fifth nerve which causes an exaltation of function and results in an overgrowth due to an excessive proliferation of the vulgar cells of the parts. The exact limitation of the hypertrophy to the distribution of the fifth nerve or one of its branches, and the remarkable contrast between it and hemiatrophia facialis, in which the fifth nerve is diseased, speak in support of this supposition. It cannot well be explained on vascular grounds alone. Although in many cases hyperæmia is found, it is probably secondary to the hypertrophy, instead of productive of it.

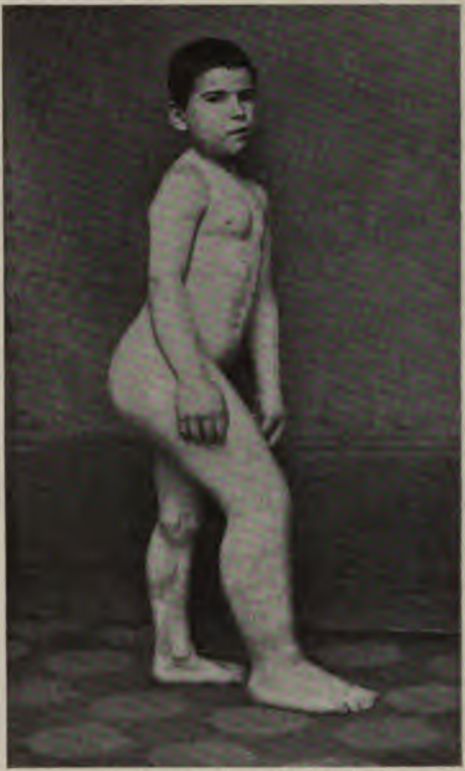
The course of the disease is as a rule a progressive one, but after adult age is reached its progress is very slow. In one case only did it tend to shorten life by causing pressure on the brain. No form of treatment has been suggested that is worthy of serious consideration.

HEMIHYPERTROPHY AND LOCAL HYPERTROPHIES.

Hypertrophy of one extremity or of one side of the body is very rare. Hypertrophy of the fingers and toes is more common. The former is frequently, but not always, congenital; the latter may occur with diseases in which enlargement of the body is the most prominent symptom, such as acromegaly and gigantism.

Richardiere, who has described two cases of congenital hypertrophy of the hand, considers that there are two forms of such hypertrophy. A true hypertrophy, in which all the parts participate, the soft parts, the tendons and bones are augmented in volume. In the variety which he terms false hypertrophy, there is an abnormal development of cellular tissue and subcutaneous fat. The discrepancy in size between the two sides of the body or between two extremities may be very distinct at birth, and the relative proportion between the enlarged and the normal size is preserved during the years that the child continues to grow. This is well seen in a case reported by Moebius about ten years ago. In referring again to the case in a recent communication, he says the difference in the two sides has remained practically the same during the past eleven years. The hypertrophy of one-half of the body may be so great at birth as to constitute almost a monstrosity. This was so in a case reported by Demme, in which, in addition to the enlargement of one-half of the body, the tongue was increased to more than twice its natural size, and was never withdrawn into the mouth; eventually it suffocated the little patient. The hypertrophied extremity may be increased in length as well as in breadth and volume. The structural participation differs in different cases. In some instances the muscles of the limb are hypertrophied, and there is a corresponding increase in strength. This was so in a patient reported by Osler. In most cases, however, the muscles are very deficient, and the result is more or less disability of the member and possibly some deformity. The cellulo-adipose tissue shows the greatest change. The bones are not involved in the majority of cases; they may be involved in one part of the body, but not in another, although the latter may be greatly increased in size. This was so in Demme's case. in which the left half of the body was involved, but the bony increase was confined to the skull. In cases where the muscles do not seem to be especially involved, microscopical examination of them shows increased diameter of the muscular fibres, absence of cross striping, granular degeneration, decrease of inter-fibral tissue, increase in nuclei of the sarcolemma and development of embryonic cells in the connective tissue between the muscle bundles. Neither the nerve endings nor the peripheral nerves seem to be affected. In one case it was thought that there was an increase in ganglionic cells of the anterior cornua of the affected side, and one hemisphere of the brain was larger than the other. A remarkable fact that has been observed in several cases is a unilateral increase of surface temperature. This has been noted by Trelat and Monod, by Ollier and by Redard. The last mentioned author thinks that if all cases are examined carefully in reference to this point a difference in the two sides will be found. Careful observation by other clinicians discredits this statement. Sensibility of the enlarged extremities is as a rule undisturbed, likewise the vascular supply as manifest by the pulse of the part and by the color. Very rarely there is pigmentation of the skin or evidences of continued hyperæmia. Hyperidrosis has been observed in some cases, but it is not the rule. In some instances the superficial glands are enlarged and spots like cysts appear beneath the skin, but there is nothing to suggest elephantiasis.

FIG. 301.



Hemihypertrophy. (MOEBIUS.)

Crossed lateral hypertrophy has been reported only in a few instances. In Friedreich's case the left side of the face and left arm and the right lower extremity were enlarged, and in this case the vascular symptoms were marked.

Local hypertrophy, unless compensatory, is also a rare condition. It is most often seen affecting an appendage of the body, such as the penis, the breasts, the external ear, etc., or the carpal or pedal digits. Compensatory hypertrophy is not uncommonly seen in one testicle or one kidney after the removal of its fellow. The lymphatic glands also exhibit the phenomena of compensatory hypertrophy. Local hypertrophy may be congenital or it may develop at any time up to the age of maturity. It is seen almost entirely in those who are neurotic through inheritance, or in those who are suffering from some affection of the nervous system. In the case reported by Springer the two large toes developed an enormous symmetrical enlargement while the patient was suffering from myelitis. In Kanthack's case of acromegaly the second toe was hypertrophied out of all proportion to the enlarged foot. Hysteria in some of its manifestations is at the bottom of many cases, especially where the breasts are involved. Hypertrophy of the nails, onychogryphosis, is associated with neuritis, neuralgia, myelitis, injuries to nerves, and in some cases of muscular atrophy. Enlargement of an ear is generally seen with some manifestations of disordered sympathetic on the same side. Hypertrophy of the cephalic extremity developing late in life has been noticed by Starr. Although the hypertrophy seen in cretins is neurotic, we do not discuss it here. It is treated under cretinism. Hypertrophy of adipose tissue shows itself either in the shape of obesity, circumscribed or diffuse, or adiposis dolorosa, and it is with these that such cases as the one reported by Mitchell (*American Journal of the Medical Sciences*, 1885, p. 162) is to be classified. Hypertrophy of individual muscles or groups of muscles are almost without exception traceable to overuse or overstimulation.

The only suggestion that has been made in the way of treatment for any of these cases is pressure on the vascular supply when they develop prenatally, or injection of an astringent into the parts, such as iodine when they are congenital. There is no record that any such measures have ever been of any service.

CHAPTER XXXI.

TROPHIC DISEASES ASSOCIATED WITH PATHOLOGICAL CHANGES IN THE THYROID GLAND.

By CHARLES W. BURR, M.D.

MYXŒDEMA.

Definition. A constitutional disease dependent upon atrophy of the thyroid gland and characterized by a myxœdematous condition of the subdermal tissues and progressive mental failure.

History. The first description of the disease is given in a paper by Sir William Gull, "Upon a Cretinoid State Supervening in Adult Women," published in 1873. Four years later Ord published an article describing the condition both clinically and pathologically, and proposed the name myxœdema (mucous œdema). The most exhaustive study of the subject is the report of the committee appointed by the Clinical Society of London, published in 1888.

Etiology. Climate seems to exert no influence, at least, as the disease becomes better known more cases and in more scattered regions are being reported. It is much more frequent in women than in men, probably in the proportion of ten to one. It may be transmitted through the mother, and several persons in one generation may be attacked. It is to a large degree a disease of middle life. It has occurred after acute articular rheumatism, erysipelas, and persistent hemorrhages. Hochler, Pospieloff, and others have seen it in syphilitics, and in the case of the first-named cure followed specific treatment.

The direct and immediate cause is atrophy of the thyroid gland. As to the causes of the atrophy, we are still largely in the dark. It is by no means impossible that in some cases the thyroiditis secondary to the acute infectious fevers may pass on to the condition of sclerosis, and thus be a cause. The disease has been produced artificially by removal of the gland both in animals and man. We will speak more fully of this under the pathology.

Symptoms. The onset is ordinarily slow, and the whole course of the disease occupies from ten to fifteen years. There are a few cases, however, like the following, described by Osler, to which, as he says, the term acute myxœdema might be applied: "A young man, aged twenty years, presented a gradual enlargement of the face, particularly of the lips, cheeks, and nose, without actual œdema. The backs of the hands were also swollen, but did not pit. The condition came on with enlargement of the thyroid, and, after persisting for between three and four months, is now subsiding."

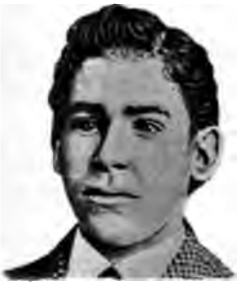
The symptoms are referable to the skin, the mental functions, and the thyroid gland.

The coarse, broadened, moon-shaped face is characteristic. The nose is wide and thick, and the mouth larger than normal. The lips are thickened.

The eyelids are swollen sometimes so greatly that they cannot be opened. The complexion is yellowish white, with oftentimes a reddish patch on either cheek. The expression is immobile and stupid. Like the face, the bulk of the whole body is increased.

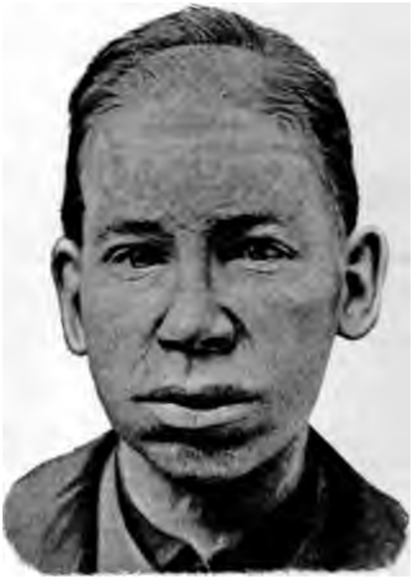
The yellow-white skin, on superficial examination, looks œdematous, but it does not pit on pressure. On the contrary, it is firm and elastic. This false œdema is less marked on the trunk than on the extremities and face, and is not influenced by gravitation. Perspiration and the sebaceous secretions are scanty or absent. The skin is dry but not tense, and scaly particles of epithelium brush off. The hair is dry and brittle, the eyebrows and lashes fall out, and baldness comes on. The hands and feet are swollen, and the fingers move with difficulty. The nails are brittle, striated, and either atrophied or thickened. In the supra-clavicular and axillary regions local swellings occur resembling false lipomata. The mucous membranes are swollen and dry. Toward the end there may occur a secondary nephritis which complicates the picture of the disease.

FIG. 302.



Photograph taken when the disease was commencing. (HUN, American Journ. of the Medical Sciences, July, 1888.)

FIG. 303.



Photograph of same case taken when disease was fully developed.

The mental changes were noted by Gull. In his first paper he says: "The mind, which previously had been active and inquisitive, assumed a gentle, placid indifference corresponding to the muscular languor, yet the intellect was unimpaired." Slowness in mental processes, in apprehension, thought, and action, is almost constant, being absent in only three of the cases studied by the committee. Nevertheless patients are liable to outbursts of fretfulness and ill humor. Absolute insanity, acute or chronic mania, dementia or melancholia with delusions or hallucinations occur in about one-half of the cases. Delirium of suspicion and exaltation is not infrequent. Shame on account of the appearance may precipitate mental trouble. Thus Wilks records a case in which a young woman living in a country town was so dis-tressed by the village boys shouting after her, "pig-faced woman," that she refused to go out at all, began to find insult when none was intended, and finally became so threatening and violent that she had to be placed in an asylum. The peculiar slowness and deliberation in all movements are also due to the mental condition, and not to any paralytic weakness. Memory is impaired. Speech is slow and difficult. Out of the one hundred and nine cases analyzed by the committee of the Clinical Society, the thyroid was found atrophied in twenty-two cases, not altered in size in twenty-three, and with a history of previous enlargement in three. In life it is frequently difficult, especially if the neck be thickened, as happens so often, to tell anything about the presence of atrophy, so that such figures as the above have only a relative value. Ord reports one case and Sollier two in which exophthalmic goitre preceded the myxœdema.

To the above main symptoms are added always secondary ones. The bodily temperature is below the normal—97° to 97.5°. The only objective sensory symptom is delay in recognizing sensory stimuli, but various paræsthesias, feeling of coldness, pricking, and numbness are common. Ringing in the ears, vertigo, and dull headache are frequent. Albuminuria and rarely glycosuria may be present. The urine is apt to be large in amount and of low specific gravity, and a few casts may be found. Palsies, contracture, and tremor are very rare. The knee-jerk is present, but decreased. The heart, lungs, and abdominal organs usually functionate normally. Sleep is usually good; indeed, there may be excessive somnolence; but, occasionally, one or the other form of night horror is complained of. Smell, taste, and hearing are deficient. Anæmia is common. Kräpelin found in three cases abnormal size of the red corpuscles, a high specific gravity of the blood, and a lowered amount of fibrin. Remissions are not infrequent, the patients being always better in summer. Death may result from the disease itself, or, as is more frequent, from pulmonary phthisis, or renal or cerebral complications.

Differential Diagnosis. The most important point is to differentiate the mucous œdema from the common œdema due to Bright's disease, a matter the more important since casts, few in number, it is true, may be present in the urine in the first condition. The paper of M. Allen Starr is the best upon the subject. Mucous œdema is uninfluenced by gravitation; the upper eyelid is affected as much, or it may be more than the lower. It is more intense over the masseter muscles and in the supra-clavicular spaces than elsewhere. The genitals are not especially affected as in nephritis. There is no pitting on pressure, and, indeed, the fluid below the skin cannot be displaced. In chronic nephritis the skin may be the same dirty, yellowish-white, the mental and gastric symptoms may be similar, but in nephritis we do not have the reddish patches upon the cheeks, the dry scaly skin, and the total absence of perspiration.

Obesity, scleroderma, Arabian elephantiasis, and acromegaly, though some of them, at any rate, will probably be proven to stand in close relation to affections of the thyroid, can scarcely be mistaken for myxœdema, and need only be mentioned. (See also page 897.)

There is a condition described by Dercum under the name of adiposis dolorosa, characterized by great and widespread hyperplasia of the subcutaneous fat, with local deposits in certain parts of the body, accompanied by pain, muscular degeneration, and diminished perspiration, which, while not bearing any great resemblance to myxœdema, should be mentioned here, since in two of the three cases reported (one by Henry) the thyroids were indurated and calcareous. (See page 898.)

Prognosis. The prognosis formerly was absolutely bad. Under new methods of treatment the disease certainly can be immensely ameliorated—

indeed, the symptoms caused to disappear. Whether, however, cases will remain cured over a series of years is yet to be determined.

Morbid Anatomy and Pathology. Let us first consider the effects of surgical removal of the thyroid producing *operative myxœdema* (cachexia strumipriva). Reverdin pointed out that in certain cases in which removal was done for goitre the patient became cretinous. Horsley proved that complete removal of the gland in monkeys was succeeded by a myxœdematous condition. Kocher, of Bonn, showed that the same condition followed in a small proportion of cases in man. Complete removal is necessary, since if but a small portion be left it seems to functionate well. In certain cases supernumerary glands take the place of the main one. The condition usually begins to develop about four months after operation. There is mental and physical lassitude, slowly oncoming changes in the skin, etc., till finally all the symptoms appear. The extent of symptoms varies inversely with the age of the patient. Thus, if the operation be done in early childhood myxœdematous cretinism results, while in adult life we have only the symptoms of myxœdema.

In cases of the natural disease that have come to post-mortem the thyroid gland has always been atrophied, sometimes more in one lobe than the other. Macroscopically, the organ is bluish-white, hard and firm. Microscopically, there is in the early stage an infiltration of the walls of the vesicles by embryonic tissue and a proliferation of epithelium. There are at the end stages the evidences of a chronic thyroiditis. Secondary changes in other organs are frequent. The subcutaneous fat is usually abundant, though there may be emaciation. There is an increase of connective tissue, not in the skin only, but throughout the entire body. The tubes of the sweat and sebaceous glands are blocked by swollen epithelium. Perineuritis is sometimes present in the skin. Ord found on chemical examination a large excess of mucin in the skin, and other observers have found it even in the blood. In Gründler's case of operative myxœdema, on the contrary, and in several spontaneous cases no such excess was found. The amount present probably depends upon the stage at which death occurs. The cerebral hypophysis is not infrequently enlarged, but may be normal in size. In this connection it is of interest to note that in the experiments of Hofmeister on rabbits, enlargement of the hypophysis appeared. Save for fibrous changes in the cervical sympathetic, no serious changes have been found in the nervous system.

While it must be accepted as proven that the thyroid gland is the seat of the essential lesion in myxœdema, yet that is only one step in the pathology. When we attempt to go further to find what it is that influences the entire organism so markedly we are beset with difficulties owing to our small knowledge of the functions of the gland. According to Horsley, it is a blood-forming organ. Experimentally he showed that during the anæmia resulting from its removal the blood of the thyroid vein contains 7 per cent. more corpuscles than the corresponding artery. He also found that mucin was increased in the skin, the salivary glands and the blood, and hence holds that the gland regulates the formation of it. We do not know, however, whether mucin is itself a poison or whether it is merely an evidence of degeneration. According to Schiff, the normal thyroid secretes a substance which influences the nutrition of the nervous system. The absence of this substance produces the trophic changes found in myxœdema. That the blood is toxic is proven by the fact that, though harmless in healthy animals, it hastens the cachexia in those in which the thyroid has been removed, as has been shown in the experiments of Rogowitsch, Fano, and others. Putnam thinks that certain of the phenomena of the disease, for example, the localized swellings in the neck and the changes in the ovaries of rabbits can be less easily explained as results of irritation and degeneration than as evidences of a sort of variation in structure, analogous, perhaps, to that which follows castration.

Treatment. Schiff found that if a thyroid gland was implanted in the peritoneum of rabbits thyroidectomy was harmless. Murray, in 1891, introduced treatment by hypodermatic injections of thyroid juice in women, with good results. Since then many brilliant results have been obtained by the use of the gland in many ways. The fresh sheep's thyroid, finely minced, may be eaten raw or slightly warmed, a half or quarter of a gland being a dose. For hypodermatic use the following solution is recommeded by Murray: One drachm each of expressed juice, glycerin, and one-half of one per cent. watery solution of carbolic acid. The dose is from five to fifteen minims, injection two or three times weekly. The dried extract in doses of five grains by the mouth seems to answer every purpose. The anæmia is not relieved by treatment, indeed, it may become more aggravated. Gray sums up the effect of thyroid treatment as follows: Elevation of temperature, increased appetite, with more complete absorption of nitrogenous foods; loss of weight, with nitrogen excreted in excess of that taken in the food; growth of skeleton in the very young; marked improvement in body nutrition generally; increased activity of mucous membranes, skin, and kidneys. Removal to a warm climate is sometimes necessary on account of the extreme suffering produced by cold. Warm or Turkish baths are useful. Pilocarpine hypodermatically employed often does good.

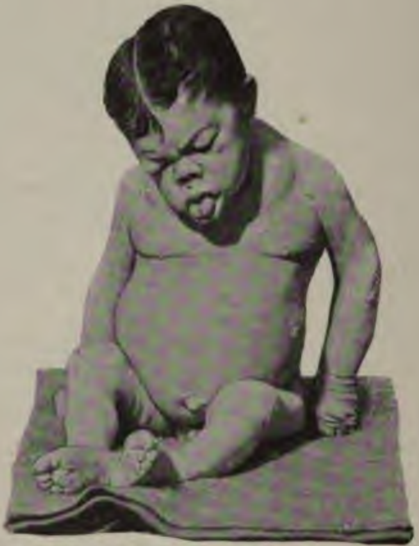
CRETINISM.

The etymology of the word is undetermined. It has been variously claimed to be derived from the Latin, creta, chalk, in allusion to the chalky color of the skin; from christianus, because cretins are good-natured, and from cretina, stupid. Cretins are sometimes improperly called Capots or Cagots.

Etiology. The disease is found in all countries. It is both endemic and sporadic, and we will concern ourselves principally with the latter type. In the countries in which it is endemic it is not found over large areas of territory, but here and there in limited tracts. It is especially frequent in mountainous regions. The Alps, the Pyrenees, the Vosges, and the Jura are favorite seats. In North America the endemic form is confined to certain parts of Vermont, Massachusetts, and California. Drinking glacier water and water rich in lime salts is an alleged cause. It is very doubtful, since in certain countries in which there are large deposits of limestone cretins are rare, and in others in which the water is perfectly soft they are common. Altitude, atmospheric moisture, and all climatic and geologic factors have been thought to be causative. Heredity is important. Judson Bury lays great stress on interbreeding. The disease may begin in early uterine life, in which case the sufferer rarely survives birth, or late in uterine life, or after birth. The frequency of the sporadic form increases with the knowledge of the disease; from Philadelphia alone, for instance, cases have been reported by Dercum, Mills, Sinkler, and Lloyd.

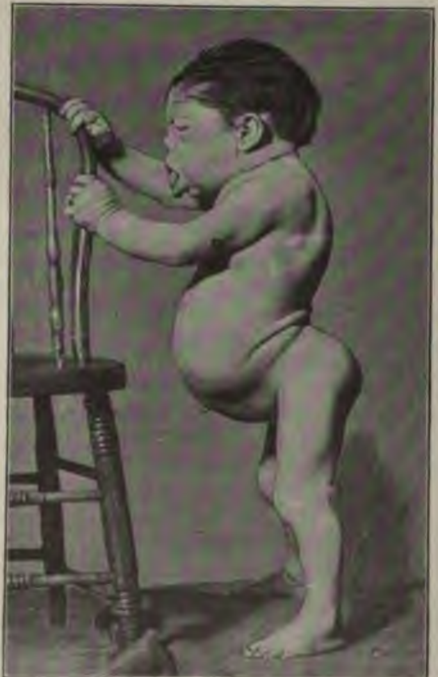
Symptoms. In the sporadic type the symptoms usually begin about the fifth year. They are characterized by mental degeneration and physical retardation. Growth is stunted, the stature rarely reaching beyond five feet. The different parts of the body develop disproportionately, causing deformities. The limbs are crooked, the ends of the long bones enlarged, the trunk too long, and the chest large and flattened. There is lumbar lordosis, throwing the abdomen far forward. The head is brachycephalic, the vertex and occiput flattened. The hands are large, flat, spade-like, the gait awkward, bear-like. The hair is a dirty brown, coarse, rough, and thick. The body hair does not grow at all or is scanty. The face is large and square, the nose-

FIG. 304.



A cretin, aged about thirty-five years. (Philadelphia Hospital.)

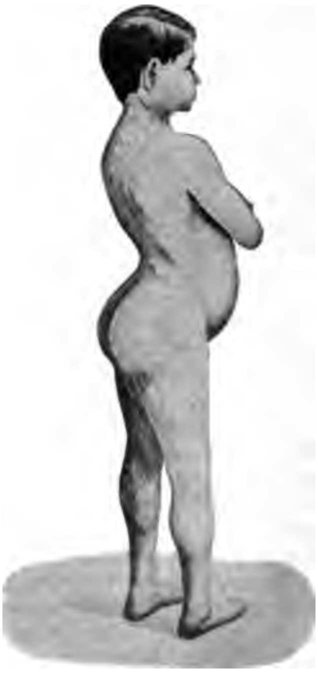
FIG. 305.



Cretin; same case as shown in Fig. 304.

bridge much flattened and wide, keeping the eyes far apart, and the nostrils dilated. The eyelids are swollen, and sometimes cannot be opened. The tongue is large and flabby, protrudes between the enormously thickened lips. The thick, viscid saliva dribbles constantly. The milk teeth decay early, and the permanent set may never appear. The skin is a dirty yellowish-brown, coarse and wrinkled, or, if myxœdema be present, it is pale. Subcutaneous fat is abundant, and there are apt to be local deposits, especially in the supraclavicular fossæ. The ears are malformed and stick out from the side of the head. The muscles are soft. The neck is bull-like, and goitre may or may not be present. The eyesight is usually good, though there may be strabismus. The other special senses and common sensation are poorly developed. Puberty is delayed, sometimes till the twenty-fifth year, and menstruation either is scanty or soon ceases. Respiration and pulse are slow, and the temperature subnormal. The circulation is poor, the extremities cold and blue. Red patches occur on the cheeks. The urine is thick, bad smelling, and quickly becomes ammoniacal. Speech is thick, the voice rough. Sleep is heavy. They are liable to curious attacks, in which they will sit for hours, the eyes open and fixed, the body motionless, scarcely breathing, and showing almost no signs of life. It would almost seem, as Maffei says, "that the soul had entirely left the body." The amount of mental deterioration varies much. Sometimes it is possible to educate them to fulfil many of the ordinary duties of life. One, for instance, who had but little schooling, keeps a news-stand, supports himself, and knows well the value of money. From this there are all grades down to absolute vacuity, in which the patient, though not paralyzed, cannot walk, nor talk, nor understand, nor even feed himself, being merely vegetative. Though ordinarily quiet, gentle, even affectionate, they sometimes are irritable and easily angered. All movements, all actions, mental or physical, are slow, not only in the initiation, but throughout the act. Many are deaf and dumb. According to the report of the Sardinian Commission, only one-third have normal hearing. The disease usually progresses for twelve or fourteen years, and then remains stationary.

FIG. 306.



A case of sporadic cretinism. (DERCUM.)

Morbid Anatomy. Cretinism, whether sporadic or endemic is closely allied to if not identical with myxœdema. We will here, therefore, consider only the lesions found and refer the reader for the study of the pathology to myxœdema.

Autopsies have been few. The first sporadic case was reported by Curling. The most important lesions are those of the thyroid gland and the osseous system. In cases beginning early in uterine life the thyroid is always absent. In those commencing later it usually atrophies slowly; sometimes, however, it increases in size and may develop into a large goitre. But little is known of the microscopic structure of the gland. Virchow's description of the skull of an endemic cretin has become classic. The spheno-basilar bone was prematurely ossified, preventing antero-posterior growth of the skull. This would cause, in order to compensate, expansion of the cranial vault, and hence widely open fontanelles. The condition has not, however, proven constant. Lombroso reports a case in which the basilar process and the occipital condyles were absent. The bones of the skull may be greatly thickened. All the long bones (developing in cartilage), except the clavicle, are shortened. The brain may present various convolutional anomalies, but none are constant. In Barlow's case the most interesting abnormality was abnormal fissuration in the temporo-sphenoidal lobe. The cerebellum was more covered by the cerebrum than normal. Many cases of so-called fœtal rickets are, according to Barlow and Bury, really cases of fœtal cretinism. Post-mortem there is much subcutaneous fat. The limbs are dwarfed. The long bones are firm, smooth, and unduly compact. In the skull the membrane-formed bones are well developed, those formed in cartilage are stunted. Microscopically the bone condition is the reverse of that found in rickets. The thyroid is not atrophied in all cases. Eberth has found similar conditions in malformed calves.

Treatment. The thyroid treatment should be used in the same way as in myxœdema, and it is probable that in every institution for the feeble-minded and in every insane asylum there are few or more cases that could be greatly benefited.

EXOPHTHALMIC GOITRE.

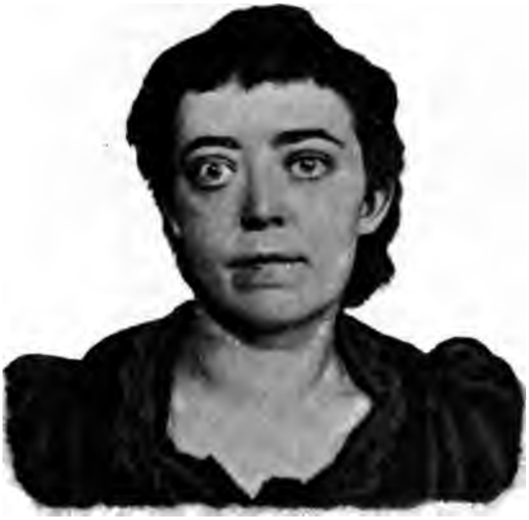
Synonyms: Graves's Disease; Basedow's Disease; Struma Exophthalmica; Cardio-thyroid Exophthalmus.

History. The Italian Flajani was probably the first to describe the condition (in 1802). The English physician Parry speaks of the most important symptoms in his work published in 1825, but it was not until Graves's description of the disease, in 1835, that the interrelation of the three great symptoms was recognized. Basedow discovered the disease, independently of Graves, in Germany in 1840.

Etiology. No specific cause is known. Direct inheritance plays a very subordinate part. Occasionally, however, several persons in one generation are affected, as in the family reported by Oesterreicher, in which eight out of ten children suffered. Indirect inheritance through insane, epileptic, or neurotic parents is frequent. It is far more frequent in women than in men. Of thirty cases only one was a man. In Eshner's table forty-two are males and one hundred and eighty-five females, a proportion of about one to four. Ross says it affects females twice as often as males. It may occur at any period of life, except early infancy and extreme old age. The thirtieth year is the time of greatest frequency. Hawkes reports a case in a girl of six years, and Gowers has seen it appear as late as fifty-three. The causes of cystic goitre are without causative influence. There is no evidence that preceding organic heart disease predisposes to the affection. All factors which tend to reduce strength, exposure, anæmia, chronic discharges, abortion are, of course, in a way causes. The most frequent and most important immediate cause is emotion—a sudden moral shock or prolonged grief. Thus a man when told of his brother's sudden death began immediately to complain of palpitation and soon presented the typical symptoms.

Symptoms. Rarely the onset is rapid, almost sudden, and the course very acute. Such usually die, though sometimes recovery takes place or the condition becomes chronic. Osler quotes a case of J. H. Lloyd's. A woman, aged thirty-nine years, who had been considered perfectly healthy, but whose friends had noticed for some time that her eyes looked rather prominent, was suddenly seized with intense vomiting and diarrhœa, rapid action of the heart, and great throbbing of the arteries. The eyes were prominent and staring, the thyroid much enlarged and soft. The patient died on the third day. Another case, scarcely comparable to the foregoing, but of interest as showing all the objective signs without any subjective symptoms until they suddenly developed after a slight shock, was that of a woman who came to the hospital solely on account of the disfigurement produced by exophthalmos, and who began to complain bitterly of palpitation and tremor immediately after being exhibited in clinic. Most frequently the development of the disease extends over many months. Of the three cardinal symptoms, cardiac overaction, exophthalmos, and goitre, the first named usually appears earliest. The course of events is about as follows: At the beginning the patient complains of attacks of palpitation and cardiac distress after exertion or emotion, with some dyspnœa. Examination may reveal nothing but a rapid pulse. As time passes the attacks become more frequent, are more severe, and last longer until finally the pulse-rate becomes persistently high, often 100 or 120 per minute, rarely 200. There is increased force of the heart's action as well as increased frequency. The impulse is strong and widely diffused, the entire chest sometimes throbbing. Early the apex beat is in the normal position, but later it is displaced downward, and to the left, and the signs of dilatation appear. Hypertrophy is rarer. The heart sounds are loud. Graves in one case heard them four feet distant from the body. Systolic murmurs heard plainest at the base, but sometimes audible at the apex and transmitted to the axilla, are common. They are not always functional. It is probable that not infrequently in the fully developed disease there is a true mitral regurgitation; the mitral orifice having been mechanically dilated by the dilating ventricle, an apical thrill may be felt. The carotids and even the abdominal aorta are dilated and pulsate strongly. Venous and capillary pulse sometimes is present. Systolic murmurs are heard in the larger arteries. There is greater or less dyspnœa.

FIG. 307.



Exophthalmic goitre.

Enlargement of the thyroid and exophthalmos usually appear together, or at about the same time, and after the cardiac symptoms. The enlargement is slow and painless and affects the whole organ, one lobe much more than the other, or very rarely the isthmus only. At first the swelling is soft, but its density depends much upon the strength of the cardiac action. Is never so great as that which is seen in common cystic bronchocele, and varies from time to time. The arteries, and, indeed, the whole gland pulsate strongly. A thrill is felt on palpation, and on auscultation there is heard a distinct, sometimes, loud systolic murmur. According to Guttman there is a bruit in the goitre synchronous with the pulse, even though the heart sounds are normal. It is probably due to aneurismal dilatation of the arteries. A venous hum in the neck is frequent. The superficial veins may be much dilated. Striking, as the thyroid symptoms often are, there are not a few cases in which at no time in the course of the disease is there any recognizable involvement of the gland.

Exophthalmos rarely is the first symptom. It may be very slight, causing only a staring appearance, or so great, as is alleged in one case, as to dislocate the ball completely from the orbit. Frequently it is sufficient to allow the scleral coat to be seen above and below the cornea, and sometimes the insertions of the recti muscles are exposed. The amount of exophthalmos varies from time to time. Usually vision is unaffected, but occasionally the antero-posterior diameter of the globe is increased, producing myopia. The pupils are normal. Ophthalmoscopic examination is usually negative except that arterial and venous pulse is sometimes present. Œdema of the disk, and rarely atrophy, due most probably to the mechanical stretching of the nerve, are met with. When the exophthalmos is great the lids are prevented mechanically from closing, and the dry conjunctivæ become inflamed. Opacity of the cornea, and even ulceration and destruction of it, may ensue. Sometimes there is œdema of the lids, even though the exophthalmos is slight. The so-called von Graefe's sign is very important. In health, on looking downward, the upper lid follows the ball; ordinarily in this affection, the lid either does not move at all, or descends haltingly, not acting in unison with the ball. Other movements of the eyelids, as in winking or in going to sleep, or in voluntarily closing the eyes, are not interfered with. The symptom is not to be mistaken for the inability to close the eye because of the extreme exophthalmos. Stelwag's sign, the widening of the palpebral fissure, on account of the retraction of the upper lid, is really only one of the factors of von Graefe's sign. Retraction of the lower lid is spoken of by Hill, Griffith, and others. Joffroy found in three cases a condition which bears much analogy to von Graefe's sign. If the patient, looking at the ground was told to look at the ceiling, as the eyeballs rolled up the frontal muscle did not contract as in health. Voluntary contraction of the muscle was perfect.

The general condition of the patient is much affected. She is apt to be irritable or depressed, hysterical, and neurasthenic. Anæmia and emaciation are frequent. There is apt to be moderate or even quite high fever, but the temperature may be normal throughout. Subjective sense of heat, hot flashes, profuse perspiration, sometimes very acid and rarely local, are often complained of. The "tâche cérébrale" sometimes can be obtained.

Tremor is almost a constant symptom. It may be fine and slight, or coarse; indeed, choreiform. The hands are often alone affected, but it may be more extensive, involving in one case, for example, the tongue. While it is present during rest it may be increased by voluntary muscular effort or emotion.

Cardiac overaction, exophthalmos, and goitre, together with tremor, constitute the essence of the disease; but there are always present, to a greater or less degree, other symptoms which may have their origin, so varied are they, in almost every organ. One of the most important complications is insanity. The type found varies much, ranging from the simple depression and irritability spoken of above to extreme melancholia or acute mania. As a rule, the mental symptoms are periodic, or Graves' disease may recur, with attacks of periodic mania. In general paralysis there may be exophthalmos only, or the entire triad of symptoms. The coexistence of mental disturbance is far too frequent to be merely coincidental, and as Spitzka well says, "It is an interesting problem for the future to solve why enlargement of the thyroid should in two disorders, such as exophthalmos and cretinism, be associated with mental disorder."

Owing probably to the dilatation of the small bloodvessels and the excessive perspiration, there is a marked diminution of the electrical resistance of the skin, which, while normally amounting to from 4000 to 6000 ohms, may be reduced to 3000 ohms. Local redness of the skin, and even a general erythema is not very uncommon, and vitiligo sometimes occurs. Urticaria may be troublesome. In one case the patient was awakened several times to find painful wheals scattered over legs, arms, and trunk. They would last a day or two, and then pass quickly away. Bronzing similar to that of Addison's disease, but almost never affecting the mucous membranes, has been occasionally observed. In a case of my own it seemed to deepen or grow less from week to week. There is sometimes marked œdema. Gowers speaks of a case in which myxœdematous swelling of the eyelid and pigmentation of the skin followed permanent recovery.

Polyuria is common. Glycosuria is not very frequent. Transient albuminuria is probably present at some period in every case.

The alimentary tract offers many interesting symptoms. There may be simple anorexia or nausea at the sight of food and vomiting, or, on the contrary, the appetite may be ravenous. Sometimes there are attacks of acute severe diarrhœa, in which many large, painless, watery stools, with undigested food, are passed daily. The attack may be so sudden and severe as to resemble sporadic cholera. The condition has been regarded as an intestinal sweating. The biliary secretion may be increased and an icterus of several months' duration develop.

Menstruation is either very irregular or suppressed, and its absence may coincide with a severe and persistent leucorrhœa. It is stated that among men there may be at the beginning a marked increase of sexual desire, followed later by impotence.

There are many complications referable to the nervous system. Various palsies of the cranial nerves, especially the motor nerves of the eye, the third, fourth, and sixth are met with. Ballet speaks of a case in which complete external ophthalmoplegia was associated with double facial palsy, and Gowers quotes from Bristow one in which it was associated with right hemianæsthesia, including the special senses, epileptic fits, palsy, with rigidity of the right side, hemorrhages from the ears, and persistent pyrexia. Post-mortem examination revealed nothing.

Paraplegia, either spastic or flaccid, chronic muscular atrophy, paralysis of the sphincters, even locomotor ataxia, and acute myelitis are occasional complications.

At times there are marked local trophic changes, the hair may be lost from the brows and lids, the mammæ atrophy, the skin even becomes gangrenous in places.

Epilepsy, neuralgia and migraine are occasional complications.

Course and Prognosis. Fortunately no one patient suffers from the entire long catalogue of symptoms. Cardiac excitement is probably never absent, though its intensity may vary greatly. The disease is in most cases essentially chronic, and the exact date of onset is hard to determine, since the affection may exist for a greater or less time without the knowledge of the patient. Throwing out the very acute cases mentioned above, death is rare within six months, and the disease may last five or even ten years. Recovery results in about 25 per cent. of the cases, according to Gowers, and it is probable that this estimate is far within the truth if ill-developed cases be included. Remissions are not infrequent, and complete intermissions occur sometimes. The symptoms may disappear independently of each other. Thus, not infrequently, while the heart continues much affected, exophthalmos and goitre may, under treatment, become much less marked.

The prognosis in any given case is difficult. While in general it is true that the more severe the symptoms the worse the outlook, it is also true that the worst symptom may subside and the subordinate ones persist. Cases seen early, and which have developed rather rapidly, are more favorable than those in which the onset was very slow and unnoticed. Pregnancy, curiously, sometimes exerts a beneficial influence. The disease is more fatal in men than in women.

Death usually results from the heart. Palpitation and dyspnœa increase, œdema and diarrhœa set in, and great general exhaustion ends the scene. Tuberculosis is sometimes the immediate cause, or vascular cerebral trouble.

Diagnosis. In well-developed cases there is no difficulty in making a diagnosis. If the onset be very acute mistakes can easily be made. Thus, if the fever take a typhoid course, as happens sometimes, and the legitimate symptoms are slight, the affection may for a time be mistaken for typhoid fever. Again, if the symptoms be largely thoracic, and rapid emaciation and profuse sweating be present, acute phthisis may be thought of. The difficulty is further increased here by the fact that in the latter disease there may be at first no stethoscopic signs. When hysteria is added to chlorosis, the palpitation, the hæmic murmur, the digestive troubles, the nervousness, all combine to give a picture much resembling beginning Graves's disease.

The cases in which the symptoms are almost purely cardiac in the early stages are often difficult to diagnose. Either organic heart disease is feared, or the trouble is made light of as being merely nervous. Every case of persistently rapid heart without definite cause should be carefully examined for ocular and thyroid symptoms.

Ordinary cystic goitre is usually easily differentiated. There are neither heart nor eye symptoms, the enlargement is greater, and endemic influences may be present. Occasionally, however, the goitre, from pressure on the sympathetic and vagus, may cause rapid pulse, one-sided exophthalmos, and spasmodic mydriasis. Carotid aneurism needs only to be remembered to avoid error.

The morbid anatomy of Graves's disease is chaotic, its patholgy unknown. Post-mortem, various changes in many organs have been found, changes so numerous as not to give light, but rather to confuse.

The heart is dilated and the walls may be thickened and degenerated. Slight valvular endocarditis is common; when severe it is probably not due to the cause of Graves's disease, but coincident with it. Examination of the cardiac ganglion has been negative. There is dilatation and thickening of the walls, and even atheroma of the arteries to a greater or less degree throughout the body.

The thyroid may be simply hypertrophied. Sometimes there are marked colloid changes throughout the organ or in small areas, forming cysts surrounded by fibrous envelopes. In still other cases there is great proliferation of the connective tissue comprising the vesicles and producing a true sclerosis. The bloodvessels are dilated and thickened. Möbius, Spencer, and others have found a persistent thymus the seat of changes similar to those found in the thyroid. The orbit is often normal. Occasionally the orbital fat is increased in quantity. There are often evidences of an active congestion of the retro-bulbar vessels probably quite sufficient to have exerted some influence in the causation of exophthalmos. The ocular muscles are often the seat of fatty degeneration, and in not a few instances Müller's muscle has been absent. Elongation of the optic nerve is, of course, present when there is exophthalmos.

No important lesions in the abdominal viscera have been noted except that in three cases Savage found disease of the supra-renal capsules. In one these organs were small, diffluent, and broke easily. Savage and Howse have seen enlargement of Peyer's patches. Nephritis, probably secondary to heart disease, occurs sometimes. There may be a chronic enteritis following prolonged diarrhœa.

In earlier days Recklinghausen and others found lesions in the sympathetic, but recent observations with more improved methods have not verified them. Increase of the connective tissue in the cervical ganglion, atrophy of the nerve cells and the like have certainly been found, but these lesions are not constant; in the examination of Déjerine and others they were absent, and when present may be secondary.

In the spinal cord there has been found an overgrowth of the neurogliar tissue and dilated and thickened vessels. In some cases at least those were simply the changes incident to age. Drummond reports poliomyelitis. Usually the medulla is normal, though sometimes it shows the evidences of congestion, and even recent hemorrhages. Mendel reports one case in which the solitary bundles were unequally developed, and one restiform body was atrophied. Marie and Marinesco had a case associated with tabes in which there was atrophy of the solitary bundle and of the ascending root of the tri-facial. On the other hand, Oppenheim reports the same lesion in a tabetic who suffered from tachycardia.

Certainly many of the lesions described have no causal relation to the disease, and no theory has yet been evolved which satisfactorily explains all the major symptoms. Theories there have been in plenty. The opinion of Stokes and Graves, that the disease is one of the heart itself, has long since fallen into oblivion. The view that the symptoms are caused by pressure upon the vessels and nerves of the neck needs only to be mentioned to be dismissed. Disease of the cervical sympathetic, a view which has had much vogue, will explain some but not all the symptoms. Rather, we are driven to the central nervous system for the seat of disease, and it will probably be proven to be in the medulla. Filehne, and after him Durdufi, divided the anterior part of the restiform bodies and obtained exophthalmos, in some cases enlargement of the thyroid also, and in one the three major symptoms. The absence of any very gross lesions in the cases examined, and the probability that the congestions and hemorrhages sometimes present are merely terminal conditions, together with the fact that cases of long-standing may be recovered from, which is against organic disease, make it conceivable that the cause may be some poison acting upon the medulla without causing a lesion gross enough to be recognizable by our present methods of examination. The view that some poison is the primary cause seems to be the most widely-accepted doctrine at the present time. Möbius held that the enlarged thyroid produced a toxic blood state. Johnston believes that there is a "hyperthyroidation" of the organism, while others, Joffroy among them, claim that there is an alteration of the secretion. Durdufi has found in the normal thyroid a substance analogous to cocaine, and the latter may cause increased pulse-rate, exophthalmos, and trembling, and certainly does cause retraction of the lid and a condition similar to von Graefe's sign. Boinet and Gilbert have found certain ptomaines in the urine which, however, do not produce the disease when injected. In conclusion, we may say tentatively that the evidence is quite strong that the cause of the disease is a poison originating in the thyroid and acting mainly upon certain structures in the medulla.

Treatment. Rest is all important. In a severe case the patient should be kept absolutely in bed. Many drugs have been used. All seem to do good sometimes. No one can be depended upon before trial in any given case. Digitalis and strophanthus sometimes give excellent results, sometimes are useless, sometimes do harm. Belladonna, recommended strongly by Gowers, probably is the most useful of all drugs. It should be given in ascending doses until the patient can bear no more. The sheep's thyroid gland and its extract are useless. General hydrotherapy is to be recommended. An ice-bag over the heart, or friction with ice, at first only for a few minutes, and increasing the time each day, will often reduce the pulse very much and give the patient lasting relief. The galvanic current applied to the region of the cervical sympathetic seems sometimes to exert a quieting influence on the heart's action. Either the galvanic or the faradic current may be passed with good effect through the thyroid, or applied at the back of the neck and over the heart. The thyroid has been excised frequently, sometimes with good result, often with none. If anæmia be present iron must, of course, be given. The diet should be carefully regulated. Jaboulay has recently introduced a new operation, exothyroipexia, in cases in which the enlargement of the thyroid is vascular. An incision is made in the median line, the gland detached from the trachea and exposed in the wound, covered by an antiseptic dressing. It rapidly decreases to the normal volume, and the wound is closed. The result is claimed to be excellent. There are certain cases associated with nasal hypertrophy which have, it is alleged, been cured by cauterizing the nasal mucous membrane.

CHAPTER XXXII.

SYMPTOMATIC DISORDERS.

By JAMES C. WILSON, M.D.

HEADACHE.

Definition. Headache is a term used to designate diffuse pain referred to various regions of the head. It may be paroxysmal or continuous.

SYNONYMS. Cephalalgia; Cephalæa.

Of these terms, the former was applied by the ancients to slight, limited, or transitory headaches; the latter to severe, deep-seated, and chronic pains in the head.

Headache is a symptomatic neurosis, and cannot be regarded as in itself constituting a definite disease. The propriety of the separate consideration of headache in a systematic treatise like the present may justly be called into question. Headache is, however, in many cases a symptom of such importance and prominence that it overshadows all others and lends to the clinical picture its most characteristic feature, sometimes at first sight its only obvious feature. Headache is, moreover, a symptom frequently significant when other morbid phenomena are obscure; it thus acquires in doubtful cases diagnostic value, often of a high degree. Again, it is a symptom of many diverse conditions, and therefore of far-reaching and manifold importance. For these reasons it seems proper, even though involving repetition, to consider it separately and at some length.

Etiology. Headache is a very common nervous symptom. It occurs in varying degrees of intensity as a manifestation of the most diverse morbid states. It arises at all periods of life, but is most common between the ages of puberty and middle life—that is, between the fifteenth and forty-fifth years. Early childhood and advanced life enjoy a comparative exemption, which is to be ascribed partly to the fact that these periods are relatively free from many of the underlying pathological conditions and partly to the fact that at the extremes of life the cerebral cortex is less active functionally; that is to say, less irritable.

The headaches of childhood are not only much less frequent than those of adult life, but they are also more limited in variety, and as a rule less severe.

The influence of sex is notable. Women suffer more than men in the ratio of three to one.

It would appear that headache is more common in city life than among country folk, among the well-to-do than the poor, in the spring and autumn and in temperate climates. Highly educated persons and those whose time is devoted to letters and learning suffer more than others to an extent which cannot be fully explained by their sedentary habits.

Headache occurs as a symptom more or less prominent under the following conditions, which, taken together, may be regarded as forming the basis of an etiological classification:

1. Traumatism involving the head.

It is to be noted that headache is a constant symptom after recovery of consciousness in cerebral concussion and pressure, but that it occurs infrequently as a direct symptom after surgical operations affecting the cerebral cortex or after laceration of brain substance if pressure have been removed.

2. Circulatory disturbances, as:

(*a*) Passive congestion, such as is produced by posture, tight clothing about the neck, or the pressure of tumors upon the veins of the neck.

(*b*) Active hyperæmia, such as results from excessive or prolonged physical or intellectual strain, or attends the initial stage of acute meningitis, or the action of certain drugs, as alcohol, amyl-nitrite, nitroglycerin. The vessels of the pia are dilated, there is increased tension, and pressure upon related sensitive nerve filaments.

(*c*) Anæmia, either that following loss of blood or the ordinary forms of anæmia, and especially chlorosis. To temporary brain-anæmia is due the headache which occurs in chlorotic subjects upon effort, as ascending a flight of stairs. Local anæmia may accompany brain exhaustion from prolonged mental effort, and may be invoked to explain the headache which frequently arises under these circumstances.

3. Inflammation. Headache is a very prominent symptom in meningitis, whether due to local cause, secondary infection, or the primary infection of cerebro-spinal fever.

4. Toxæmia. The offending substance or substances in the blood may be the result of

(*a*) Infection, as in the acute specific fevers and malaria.

(*b*) Incomplete or perverted physiologico-chemical processes, or defective elimination of waste. This variety of headache occurs in uræmia, diabetes, gastro-hepatic derangements, gout, rheumatism, and lithæmia.

(*c*) The action of drugs and poisons.

α. Acute: quinine, opium, narcotics generally, the nitrites, alcohol, ether, carbon dioxide.

β. Chronic: lead, tobacco, alcohol, opium, chloral.

5. Changes in the arteries. Endarteritis, arterio-sclerosis; hence in syphilis, degenerative nephritis, chronic alcoholism, lead poisoning.

6. Organic disease of the brain, such as abscess, tumor, aneurism (not miliary), especially when pressure is directly exerted upon the brain-membranes.

7. Caries of the cranial bones.

8. Neuropathic conditions, neurasthenia, hysteria, epilepsy.

9. Reflex irritation; ocular, nasal, pharyngeal, auditory, gastric, and irritation from the reproductive tract.

The etiology of a large proportion of headaches is not simple, but, as will be shown later, complex, two or more of the foregoing factors often being concerned in its causation.

Pathological Considerations. It is customary to speak of headaches due to lesions of the skull or to intra-cranial disease as organic; of those due to other causes as functional. The latter group includes by far the greater number of headaches encountered in general practice.

We know practically nothing of the actual mechanism by which the pain of headache is produced. Our knowledge is summed up in the statement that it is the result of the irritation of sensory nerve fibres, caused by derangements of pressure or tension, inflammatory processes, toxic states of the blood, and reflex disturbance. We know, however, that the membranes, and not the brain substance, are chiefly concerned in the production of headache. The substance of the brain in man and the lower animals does not respond to direct irritation by pain, and lesions of the brain tissue, not directly or indirectly involving the membranes, may exist without causing headache, such disturbances of sensibility as they produce being referred to distant parts of the body. The membranes of the brain, and especially the dura, on the other hand, are supplied with nerves of sensation, and are directly or indirectly implicated in the morbid conditions which give rise to headache. The sensory nerve supply of the dura in the anterior three-fourths of its extent, that of the falx, and probably that of the tentorium are derived from the trigeminus, while the dura mater of the posterior fossa of the skull is supplied with sensory fibres from the vagus.

In by far the greater number of headaches the diffuse pain appears to be deep-seated and intra-cranial. In a small proportion of cases it is referred to the scalp. It is to be noted in this connection that the trigeminus is the nerve of sensation to the scalp as far back as the vertex, while the posterior branches of the upper four cervical nerves supply the muscles and the skin of the back of the neck and the occiput, and that this group of nerves is in close central relation with the trigeminus on the one hand, while, on the other, the peripheral distribution of these branches in the scalp overlaps that of the trigeminus.

In certain rare cases superficial headaches have been due to myalgia of the occipito-frontal, temporal or sterno-mastoid muscles, the affected muscle, at its origin, insertion or elsewhere, being the seat of points of tenderness (Hirt).

It may be assumed that functional headaches can occur either in consequence of the action of adequate irritants directly upon the normal end-organs of sensory nerves, or from the abnormal reaction of diseased or under-nourished nerve fibres to slight pathological changes in the blood. Due consideration of this postulate sheds light upon some varieties of troublesome headache, and is not without bearing upon treatment. It may be added that no anatomical changes in the nerve filaments of the dura have been described and that the processes affecting the nerves in the functional forms of headache are merely nutritional and mostly transitory, albeit they frequently show a tendency to recur.

Clinical Considerations. Headaches are usually bilateral; they may be grouped according to the region to which the pain is referred, as frontal, occipital, parietal and temporal, vertical and diffuse. Frontal headache is most common. Next in order of frequency are the diffuse forms; then come the vertical, occipital and temporal. But headache sometimes changes from one part of the head to another, and by no means is constantly confined to regions limited by anatomical boundaries. Again, headache may be grouped according to the character of the pain, as

(*a*) Pulsating or throbbing: This form of pain is characteristic of the headaches due to circulatory disturbances. It is often diffuse.

(*b*) Dull, heavy: This is the character of the pain in headaches due to toxæmia, which are frequently frontal, though sometimes occipital.

(*c*) Binding or constrictive: Patients sometimes describe the sensation as that of a tight iron band around the head. This is the headache of neuropathic conditions, such as hysteria and neurasthenia. Its focus of intensity is often referred to the parietal and temporal regions.

(*d*) Burning or sore: These are adjectives frequently used to qualify the headaches of anæmia, rheumatism and gouty states.

(*e*) Boring or sharp: These headaches are often localized as "clavus—the sensation as if a nail were being driven into the head," and are symptomatic of hysteria.

The course and duration of headache depend upon the underlying conditions. It may be transient, lasting a few hours; or it may persist with exacerbations and remissions and rare intermissions for days or months.

Again there may be slight continuous pain with exacerbations of varying intensity. This occurs in certain forms of reflex headache, notably those which arise from defects of accommodation. In point of fact, persistent headaches are often reflex. Persistence is also a characteristic of organic headaches, such as arise in cerebral tumor or abscess, or pachymeningitis. The headaches caused by over-indulgence in tobacco, those of chronic alcoholism, of uræmia, of syphilis, of the toxæmia and malnutrition of chronic dyspepsia, and those which occur after sunstroke, are likewise persistent, though usually showing brief and irregular periods of remission.

As pointed out in speaking of the etiology, headache is a very constant symptom of injuries to the head, after the recovery of consciousness. The headache which follows concussion or laceration of the brain tissue is usually severe and often continues for a long period. It is not infrequently circumscribed and may be limited to a region corresponding to the site of the injury, or upon the opposite side of the head. When thus restricted it is often associated with local tenderness on light percussion. On the other hand, the headache following injury is often diffuse. In either case it is apt to be associated with vertigo, lassitude and indisposition to mental effort. As the case progresses toward ultimate recovery, head pain is in many cases readily excited by any of the numerous causes of symptomatic headache.

Congestive headache may be produced by mechanical interference with the return of the blood from the head. The headache caused by improper clothing is usually slight and quickly disappears upon the removal of the cause. That due to venous obstruction from the pressure of tumors is not usually severe, as the obstruction comes about gradually. The cerebral congestion caused by violent paroxysmal or frequently repeated cough not rarely gives rise to headache of great intensity.

Active cerebral hyperæmia may follow excessive and prolonged mental effort; or it attends the initial stage of acute meningitis, and results from the action of certain vaso-dilator drugs, among the more common of which are alcohol and the nitrites. The headache attending cerebral congestion, whether it be passive or active, is commonly frontal or diffuse, and, especially in active hyperæmia, is pulsating or throbbing.

Headache occurs in the anæmia due to blood-loss, especially if the amount has been considerable. It occurs also in the various forms of anæmia due to other causes and especially in chlorosis. Anæmic headache is often severe. It is usually frontal or diffuse, is frequently accompanied by a distressing sensation of pressure, and is often associated with vertigo, and in rare instances with tinnitus aurium. In some cases the patient perceives and is greatly annoyed by the systolic murmurs which develop in well-marked anæmia and chlorosis. The headaches of anæmia are usually intensified by temporary effort.

Headache is a prominent and distressing symptom in all forms of cerebral meningitis, whether they be acute or chronic. It may for a time be localized in cases where the infection reaches the meninges from the middle ear or by way of the nasal passages and frontal sinuses, or when it occurs in consequence of disease or injury of the cranial bone. It soon, however, becomes diffuse, and is associated with rigidity of the neck, vomiting, often explosive in character and unaccompanied by nausea; with hyperæsthesia, delirium, irregular fever, unequal pupils, and intolerance to light. The headache is usually continuous, with exacerbations of great severity. As the disease progresses to the stage of depression and paralysis, signs of compression of the brain develop, headache passes into stupor, which deepens to coma. Optic neuritis is a later manifestation. Exceptionally headache is not a prominent symptom of cerebral meningitis, and it may be absent in the early stages of the lepto-meningitis of slow development.

Sudden intense headache is among the earlier symptoms of epidemic cerebro-spinal fever, in which disease painful rigidity of the back of the neck and cerebral vomiting occur as early associated symptoms. The headache in this disease is due in part to toxæmia and in part to meningeal inflammation.

Among the early symptoms of tubercular meningitis is intense headache, paroxysmal in character, with darting pains in the head, vertigo, vomiting without nausea, constipation, and great mental irritability. As the disease develops the headache becomes persistent, and is accompanied by irregular fever, ocular symptoms and other signs of meningitis.

The headache of pachymeningitis is at first usually local. It is accompanied by fever, delirium, and sometimes by convulsions.

Severe frontal headache, usually unilateral, is a distressing symptom of acute congestion or catarrhal inflammation of the frontal sinuses.

Poisons circulating in the blood are among the most common cause of symptomatic headache. The morbid condition of the blood may be transient or persistent.

A large proportion of toxæmic headaches are due to infection. Chemical poisons are elaborated during the growth and multiplication of pathogenic germs which have found access to the body under favorable conditions, or, as in the case of the exanthemata, by the development of other infecting principles of the nature of which we are as yet ignorant.

The headache caused by these poisons is very commonly frontal; it may, however, be occipital or general, but is very rarely one-sided. It is often at first neuralgic or superficial, but soon becomes dull, deep-seated and severe.

Headache is an almost constant symptom of the period of invasion of the acute febrile diseases. In typhus it is early and severe, and is associated with pain in the back and limbs. In the course of several days it gives place to stupor.

Headache is also a constant symptom in enteric fever during the period of prodromes and the stage of invasion. It is associated with languor and becomes aggravated toward night. The headache of enteric fever subsides spontaneously during the second week of the disease, and is followed by delirium, usually of the wandering form.

The onset of relapsing fever is marked by sudden severe headache, with chill, pain in the back and legs, and wakefulness. The headache persists until the relapse, when it suddenly diminishes, or ceases altogether.

The headache of influenza is diffuse, with foci of intensity in the region of the frontal sinuses and behind the eyeballs. It is attended with great restlessness and malaise, mental and physical depression, irregular fever, neuralgic and myalgic pains, and in many instances with profuse sweating. Headache constitutes one of the most troublesome and persistent sequels of influenza.

Allusion has already been made to the headache of cerebro-spinal fever. The intense headache that marks the period of invasion of smallpox is often accompanied by excruciating back and joint pains; these symptoms usually undergoing together a remarkable remission upon the appearance of the rash.

Headache occurs in some cases of early syphilis as one of the manifestations of the general infection, independent of coarse nerve lesions. The headaches of late syphilis are usually symptomatic of gummata, meningitis or arterial changes. Headache is also a symptom in many cases of hereditary syphilis.

Certain protozoa, as the blood parasites of malaria, are capable of producing toxic conditions of the blood of which one of the manifestations is headache.

The headache of ordinary intermittent fever is a conspicuous symptom of the hot stage of the paroxysm. Headache is also a very prominent symptom in remittent fever; while periodical headache attends chronic intermittents of blurred type, of which it is often the only sharply defined characteristic.

A second group of toxic substances of great importance in the causation of symptomatic headache consists of principles developed within the body in the course of irregular or imperfect tissue metabolism or accumulated by reason of delayed or arrested excretion. The headaches due to this group of causes usually refuse to yield to symptomatic treatment. Here especially must the treatment be directed to the cause.

Many intractable headaches are due to chronic uræmia. Headache arising in this condition is often intense; it is apt to be frontal or temporal, and is continuous, with irregular exacerbations of great intensity.

Headache of the same general character occurs in diabetes, in gout and in lithæmic states. To this class also must be referred the headaches of chronic lead poisoning; and those of gastro-hepatic derangements and constipation. These headaches are due to a condition of toxæmia that is complex. They have this in common—that they are all intensified by alcoholic beverages and mostly relieved, for a time, by free purgation.

Certain drugs cause headache as a manifestation of their medicinal or toxic action. This effect may arise from overdose or in consequence of idiosyncrasy. The full, tense headache, with tinnitus aurium as a frequent accompaniment, which follows large doses of quinine or the salicylates, is well known. So also is the headache of opium, with floating sensations and nausea, and vomiting increased in the upright posture. The tense vertiginous headache which rapidly follows the administration of full doses of amyl nitrite or nitroglycerin is due in part to the direct action of the drugs upon the nervous system, and in part to the over-distention of the bloodvessels.

Headache is a significant symptom in the chronic intoxication of lead, tobacco, alcohol, opium, and chloral. In the case of lead and of alcohol vascular changes doubtless play a secondary part in the production of head-pain. The headaches of those addicted to opium and chloral are largely to be attrbuted to the depression arising from temporary withdrawal of the accustomed stimulant. They are, as a rule, increased during periods of abstinence and relieved by the administration of the habitual dose, though in the case of chloral, binding head pains sometimes occur, not in the intervals between the doses, but rather shortly after the usual dose.

Excesses in alcohol—the acute alcoholism of debauch—are apt to be followed by transitory headache of intense character, not unlike that of migraine.

Headache is attributed to changes in the walls of the minute arteries. The headache of late syphilis, of the various forms of degenerative nephritis, of chronic alcoholism and of lead poisoning, have been regarded as symptomatic of the endarteritis and artero-sclerosis which occur in these conditions. It is probable these head pains are in part, at least, due to persistent changes in the blood.

Headache plays an important rôle in the symptomatology of the coarser intra-cranial lesions, abscess, tumor, pachymeningitis interna hemorrhagica, and aneurism.

In acute abscess headache is usually very severe and persistent. It is associated with vertigo, and more or less pronounced mental dulness and irritability. Vomiting is a common, but not by any means a constant symptom. Chronic abscess, which is often latent, may present no other symptoms than headache, vertigo, mental dulness, irritability, and physical depression. The headache is, as a rule, most intense in the region of the lesion. Hence, in abscess due to ear disease, the pain is especially referred to the parietal or occipital region of the affected side; in abscess arising from disease of the nasal cavities or ethmoid bone the pain is referred to the brow; while in abscess from traumatism involving the bones of the skull, the focus of pain is located in the region of the injury, which is in the majority of the cases frontal or temporal.

Headache is present in by far the greater number of cases of cerebral or cerebellar tumor. This symptom occurs with about equal frequency in adults and children. Its frequency and intensity vary according to the site of the new growth, the rapidity of its development, and, to some extent, according to its character. Thus, headache is more persistent and severe in cerebellar than in cerebral tumors; in those of the cerebral hemispheres than in those of the base, and in those that directly involve the meninges, the pain being caused by intra-cranial pressure and irritation of the terminal filaments of the trigeminus; headache is a more prominent symptom in tumors of rapid than in those of slow growth, irrespective of the nature of the pathological process.

In the earliest periods of life the sutures readily yield to intra-cranial pressure, and the symptoms of new growths within the skull are correspondingly modified.

Aside from the matter of rapidity of growth and the yielding of the cranial bones in the very young, the character of the new growth exerts little influence in causing headache. To this general observation the exception must be made that gliomata are more frequently painless than any other form of coarse intra-cranial disease.

The headache of brain tumor is almost always a distressing symptom. It is sometimes dull and boring; sometimes lancinating; usually intense, often agonizing. While commonly continuous, with exacerbations, it not infrequently recurs with a regular quotidian or tertian periodicity suggestive of malaria. It is often worse at night—a peculiarity which is of diagnostic value. In some instances the focus of the head pain is in the region of the tumor, in others the pain centres in the brow or in the occiput, and in a large proportion of cases it is diffuse. Very often a headache which, while of moderate intensity, is localized, becomes, during exacerbation, diffuse. Local pain cannot alone be depended upon as an indication of the position of the tumor. It acquires, however, in connection with other clinical data, some degree of value in this respect. Upon light percussion with the finger-tips there is very often localized tenderness of the scalp and underlying bones, occasionally most marked in the region corresponding to the tumor. Vomiting, vertigo, hebetude, spells of somnolence, and general convulsions are associated symptoms. Optic neuritis, almost always double, occurs in 80 per cent. of all cases. Focal symptoms are usually present, though tumors of the prefrontal and temporal or temporo-sphenoidal areas and of the lateral lobes of the cerebellum, the so-called latent regions, may, if of small size, give rise to no localizing phenomena.

Headache has been a prominent symptom in a large proportion of the recorded cases of pachymeningitis interna hemorrhagica (hæmatoma of the dura). The pain is, in the earlier stages, usually referred to the vertex; later it becomes generalized. This affection, more frequent in males and after middle life, presents no well-defined symptomatology, and is usually first recognized upon the post-mortem table. Cases of this kind are very rarely encountered in private practice, but are of moderately common occurrence in that of asylums.

Intra-cranial aneurisms are of two kinds: Miliary, which involve small arterial twigs within the brain substance, and are always minute, multiple, and until rupture takes place, unattended by symptoms; and arterial dilatations of considerable size, which affect the larger arteries at the base of the brain. Even the smaller aneurisms of this second group are frequently also latent. Those of larger size occasion symptoms which are sometimes indefinite, sometimes those of a tumor of the base. The most common symptom is headache, which may be either continuous or paroxysmal. The location of the headache, when circumscribed, has little relation to the position of the aneurism, though aneurisms of the basilar artery usually give rise to occipital headache. Giddiness, mental dulness, irritability, and palsies of cranial nerves may occur. Optic neuritis is not common. Murmurs have been heard upon auscultation of the head, and in some instances perceived by the patient, who has occasionally also felt the pulsations in his head. Murmurs are, however, in the greater number of the cases absent.

Caries of the bones of the skull, of which the common causes are injury and syphilis, the rarer tuberculous ulcerations and enteric fever, is often attended with local headache. The dura, under these circumstances, resists the spread of the local inflammatory process in a remarkable manner, and general meningitis rarely supervenes.

Headache attends with great frequency such neuropathic conditions as neurasthenia, hysteria, and epilepsy.

In neurasthenia it is frontal, occipital, or diffuse. It is apt to be continuous, and is aggravated by mental application and by physical effort. As a rule, it is of only moderate intensity. It is accompanied by sensations of pressure in the head, aching in the back of the neck and spinal pains. Slight attacks of vertigo are of frequent occurrence.

The persistent or recurrent headaches so frequently encountered in recent years as a sequel of influenza are neurasthenic. The infecting principle of influenza causes profound nutritive changes in the tissues of the nervous system, of which headache is one of the manifestations. Recovery from this condition takes place in many cases, but slowly.

Hysterical patients suffer during the inter-paroxysmal state much from headache. The head pain is usually referred to the vertex, and is often severe and persistent. It is associated with the characteristic emotional state and the multitudinous symptom-complex of hysteria.

The emotional and precocious children of neurotic parents sometimes exaggerate symptoms, probably in themselves trifling, and describe, for the sake of sympathy, headaches of great severity with associated symptoms, such as inability to stand the light, brow pains, or pains in the back of the neck. It will often be found upon close inquiry that such children have a parent, especially a mother, who suffers from similar aches and pains (Mills). Mills has suggested that these so-called hysteric headaches of children might in most instances be better termed imitative headaches. They are closely allied in causation to the headaches of hysteria.

Headache in many instances develops in connection with the paroxysm of epilepsy. It may precede or follow the convulsive attack. In the latter case it is associated with post-epileptic drowsiness and hebetude. It is also a common symptom in *petit mal*, and a symptom of importance in the inter-paroxysmal state of a considerable proportion of the cases both of *grand mal* and *petit mal*.

Heat, pressure, and other abnormal sensations in the head—the so-called cerebral paræsthesiæ—are common in neurotic individuals and in brain-workers. These sensations do not amount to actual pain, though they frequently alternate with it. They are, nevertheless, often intense and distressing. They are common in adolescence and early adult life and in women at the time of or after the menopause. They occur more frequently in men than in women; in those of sedentary habits and intellectual pursuits than in the laboring classes, and in those who are lithæmic or suffer from gout. They are referred to all parts of the head, and described in the most varied terms, most of which are clearly exaggerated. The sensation in a given case may persist or recur for years, and be always the same, or it may vary from time to time. It is usually increased by mental application and by disagreeable emotions, and invariably intensified when the attention of the patient is directed to it either spontaneously or through the inquiries of his physician or friends. On the other hand, such sensations are usually not felt when the patient's interest is aroused in matters outside of himself.

Many persistent and troublesome headaches are to be attributed to reflex irritation. Such headaches then become symptomatic of morbid conditions or functional derangements in distant parts of the body. While headache of this kind is in many cases an urgent and distressing symptom, other symptoms, and in particular those of the local disease, may be insignificant or absent altogether. The causal diagnosis may then acquire a difficulty equal to its importance, and demands of the practitioner always the most painstaking application of the methods of clinical investigation, and very often the co-operation of confrères who have devoted themselves to the special departments of medicine.

Errors of refraction constitute a common cause of reflex headache. The pain is commonly frontal, sometimes in the temples, frequently occipital. The patient is very often unaware of any difficulty in accommodation, though upon inquiry it will be found that the headache is invariably brought on or aggravated by close or prolonged use of the eyes.

The head pains of glaucoma cannot be regarded as reflex. Nevertheless it seems proper to mention them in this connection. They involve the distribution of the trigeminus, having their focus of intensity in the eyeball or at the supraorbital notch. In the acute cases pain is sometimes agonizing, and is associated with depression, pallor, nausea, and vomiting. It may, however, be subacute, and in chronic glaucoma pain may recur in paroxysms of no great severity, described by the patient as attacks of neuralgic headache. As it frequently begins on one side, there is a misleading resemblance to migraine. The importance of an immediate recognition of glaucoma arises from the necessity of prompt treatment in order to check the progress of the disease. The diagnosis of glaucoma is based upon increase in the intra-ocular tension, irregular, or dilated pupil, with inactive iris, haziness, and anæsthesia of the cornea, and various visual derangements. In suspected cases the services of an ophthalmic surgeon are to be at once invoked.

The headache which arises in chronic nasal disease is usually limited to the temporal region or the vertex. It is associated with the usual signs of nasal trouble. Nausea, sensitiveness of the nasal wall of the orbit, and a hyperæsthetic area on the mucous membrane of the middle turbinated bone, which, when touched with the probe, immediately excites the localized head pain, are common associated conditions.

Among the manifold derangements of health which in children result from adenoid vegetations in the naso-pharynx, headache is conspicuous. This form of headache is especially associated with mouth-breathing, chest-deformity, retarded intelligence, and irritability, and constitutes a member of the group often vaguely spoken of as school headaches, headaches of the period of growth, and the like. Carious teeth and exposure of the pulp may be the occasion for reflex headache. Disease of the auditory apparatus may also be the unsuspected cause of persistent headache. The headache which arises in acute indigestion and in gastro-intestinal catarrh is sometimes spoken of as reflex. It is probable that this form of headache is often in part, and in most cases altogether, due to toxæmia.

The part played by disease of the sexual organs in the causation of headache has been doubtless frequently over-estimated; yet this symptom is often prominent in the general ill-health of those of both sexes who suffer from actual disease of the sexual organs or are the victims of disordered psychical processes in regard to them. In many cases these headaches are due not so much to reflex irritation as to the attendant neuropathic condition.

Certain symptoms are associated with headache with a frequency that indicates a common causation. The more prominent of these are vertigo, nausea, vomiting, drowsiness, and abnormal mental states, such as irritability and hebetude. In symptomatic headache these symptoms are, as a rule, less constant and less severe than in organic headaches. Vertigo is common in headache due to gastro-intestinal disorder—the so-called bilious headache; nausea and vomiting in the acute toxæmia; somnolence in chronic conditions, as malaria, anæmia, and syphilis. But this association of symptoms is by no means constant, and cannot be relied upon for the purpose of diagnosis. In organic headaches, however, the association of these symptoms, their persistence and the change in mental condition shown in hebetude and irritability, are very important and suggestive.

Diagnosis. The direct diagnosis of headache is, as a rule, unattended with difficulty. Diffuse pain in the head, not limited to the branches, but referred to the peripheral distribution of sensory nerves, constitutes the symptom. The sensation of pain is usually within the skull, exceptionally outside of it. Headache is more or less persistent. It is essentially a symptom, and careful examination and inquiry should reveal some general or local cause.

The differential diagnosis between headache on the one hand and migraine and neuralgia on the other, present little difficulty.

Migraine is a general disease, of which headache is a principal symptom. It is a periodical neurosis, the paroxysms of which affect not only the trigeminus, but also other cranial nerves, and, to some extent, the sympathetic. The pain of migraine is often unilateral, especially at the beginning of the attack. It is accompanied by nausea, vertigo, and subjective visual phenomena. It lasts a comparatively short time and wholly disappears with the other symptoms at the end of the paroxsym, leaving the patient in his usual health. The paroxysms recur at regular or irregular intervals.

Neuralgia is due to irritants acting chiefly upon nerves in their course. The pain is referred to the trunk or branches of the nerve rather than to the terminal distribution. It is unilateral, localized, sharp, or shooting in character, continuous with paroxysmal exacerbations, and frequently shifts from one branch to another of the same nerve or to a neighboring nerve. Characteristic tender points are present. Neuralgia of the first branch of the fifth is often attended with suffusion of the eye and œdema of the lids.

It is not always possible to distinguish between a neuralgia and a neuritis of mild intensity affecting the nerve trunk; nor, indeed, is the pathological distinction between these conditions sharply defined.

The causal diagnosis of headache, while often obvious, is sometimes extremely difficult. Its true significance in many cases becomes apparent only after an exhaustive study of the associated morbid phenomena. It is necessary to bear in mind that headache is merely a symptom, often one of many that make up the morbid complexus.

The differential diagnosis between functional and organic headaches is of fundamental importance.

The headache due to organic intra-cranial processes is usually continuous. It varies from time to time in intensity, and in certain cases undergoes violent exacerbations, but it is rarely absent altogether. The headaches of organic disease are apt to continue through the night, and very often interfere with sleep. Organic headaches are not only more persistent, but they are also, as a general rule, more severe than functional headaches. Headache due to organic disease is usually aggravated by mental or physical effort, by excitement, by alcoholic stimulants, by stooping or coughing or other influences that increase the intra-cranial vascular tension. It yields less readily than other forms of headache to systematic treatment. As the processes which give rise to it are, as a rule, progressive, it tends likewise to progressively increase in severity until at length it is replaced by the stupor, drowsiness, or coma of the terminal stage of the disease. The prominence and intensity of the associated symptoms, vomiting, vertigo, hebetude, and irritability are of diagnostic value; while the development of double optic neuritis, convulsions, and localizing symptoms, as monospasm, paralysis of cranial nerves, cerebellar titubation, forced movements or hemianopsia widely separate organic headaches from the ordinary forms of functional headache. It must, however, be borne in mind that double optic neuritis also occurs in cases of chronic lead poisoning, of grave anæmia, and various forms of diseases of the kidneys; conditions in which headache is likewise a prominent symptom.

The prognosis of headache is that of the underlying condition of which it is a symptom.

Treatment. The treatment of headache is: 1. Symptomatic; 2. Causal.

1. Whenever headache is severe, pain becomes the primary therapeutic indication, and its immediate relief must be the first object of treatment. For this purpose the new analgesic drugs are especially effective. In point of fact the prompt relief of headache constitutes their greatest field of usefulness. It is needless to enumerate in this connection the already long and constantly increasing list of the drugs and their combinations. Among the most useful are the following:

Antipyrin, gr. v (0.3) every twenty minutes until two or three, or at most four doses are taken; phenacetin, gr. x–xv (0.6–1.0) in a single dose, or preferably, gr. iij–v (0.2–0.3) repeated at short intervals; antifebrin, gr. ii–iij (0.15–0.2) repeated cautiously. Exalgin, in ordinary doses, gr. ii–iv (0.15–0.25), is less satisfactory. Mixtures of antipyrin and phenacetin, or of antipyrin, caffein, and sodium bicarbonate are also useful. Caffein and its salts, the ammonio-citrate, the citrate, the hydrobromate and valerianate are especially useful in the headaches of nervous exhaustion and of neurasthenia. Combinations of caffein citrate with sodium salicylate or sodium benzoate or of these with phenacetin or antipyrin often prove more efficacious than the single drugs. Such combinations constitute many of the preparations extensively advertised. Their efficacy is unquestionably enhanced when they are dispensed in the form of the popular effervescing granules. Chloralamide, gr. v–x (0.3–0.6), repeated at intervals of an hour or two, or in single doses of gr. xv–xxx (1.0–2.0) frequently produce relief, with drowsiness and prolonged sleep, from which the patient awakes free from pain. Sulphonal may be used in the same way. Chloral and the bromides, separately or in combination, are especially useful in headache associated with great restlessness and insomnia. In cases of habitual headache the nervous system becomes accustomed to analgesic and hypnotic drugs, and they presently lose their power to relieve suffering. This difficulty may be to some extent overcome by frequent change in the medicament, or by varying the admixture and proportions of drugs, the danger of toxic effects and of the formation of a drug habit being constantly had in mind.

Local applications sometimes mitigate the pain, seldom wholly relieve it. They are, nevertheless, a source of satisfaction to many patients. Among those to which the patients willingly have recourse are the ice-bag, fomentations of hot water, the menthol pencil moistened with alcohol or cologne water, or a 20 per cent. solution of menthol in alcohol, chloroform, stimulating liniments of various kinds, especially those containing chloroform, tincture of aconite and tincture of belladonna. The impression produced by volatile applications of this kind is increased by preventing their evaporation, but care must be observed lest blistering result.

Among local measures mustard-plasters of varying strengths are much relied upon, especially in occipital headache. After a time many sufferers from habitual headache narrow the symptomatic treatment to quietude, a darkened room, abstinence from food, with immediate recourse to purgatives, the salicylates, or such other treatment of the underlying constitutional disturbance as the experience of each suggests.

The immediate treatment of headaches due to intra-cranial organic disease demands for the relief of suffering the remedies above mentioned, given for effect. Large doses are required. In many cases it is, however, necessary to use opium or its derivatives in full doses. So soon as it becomes evident that ultimate recovery is not to be hoped for the danger of the formation of the opium habit may be disregarded.

2. The causal or constitutional treatment must be based upon the underlying local or general condition.

Thus in headache from traumatism, rest, free use of the bromides, and surgical treatment become necessary.

Where the headache is due to circulatory disturbances the treatment must be directed not only to the mechanical condition of the vascular system, but especially to the etiological factors in each case. The cause of cerebral congestion and hyperæmia must be sought out and relieved. Purgatives are especially useful. They act both derivatively and depuratively.

The headaches of anæmia are increased by movement and effort. Rest must, therefore, enter largely into the treatment for their temporary relief. Rest must, however, be alternated with carefully regulated and systematized exercise. Here also a highly nutritious dietary is indicated. Purgatives are important and the exhibition of suitable salts of iron in large doses, with arsenic in proper cases.

The headaches of intra-cranial inflammatory processes, involving the meninges, call for the administration of purgatives and in suitable cases for the local abstraction of blood.

In cerebro-spinal fever opium in large doses, with or without the bromides, still retains the confidence of practitioners.

The intelligent treatment of headaches due to toxæmia depends upon the recognition of the condition of which the headache is a symptom. In the acute febrile infections it must be symptomatic; in malaria the treatment consists in the prompt administration of quinine or other cinchona alkaloids; the headaches of syphilis usually yield to mercury and potassium iodide properly administered.

The headaches which arise in consequence of incomplete or defective physiological processes or of defective elimination constitute a large and important group. They are best treated by regulation of the diet, purgatives, the systematic employment of measures determining to the skin, including various measures of hydrotherapy.

The toxæmic headaches which arise in consequence of the action of drugs and poisons are to be treated by the exhibition of appropriate physiological antidotes and by measures of elimination. Acute toxæmic headaches of this kind in individuals previously in good health tend, if the dose be not lethal, to spontaneous recovery. In the chronic intoxications it is important that the patient be removed from conditions which expose him to the further action of the poisonous agent and that continuous systematic treatment, directed to the elimination of the offending substance, be carried out. Thus, in chronic lead-poisoning the administration of potassium iodide in connection with systematic purgation by means of salines must be instituted.

Headaches due to changes in the arterial walls are not, as is obvious, in all instances amenable to treatment. Much, however, can be done to mitigate the sufferings of the patient by regulation of the diet, a quiet life, occasional purgation, and the use of those remedies as the nitrites, the tendency to which is to reduce arterial tension.

The headaches due to coarse organic disease of the brain, such as tumor, abscess, and aneurisms of the larger arterial branches, are capable of partial relief by systematic treatment, especially by the skilful employment of opium and its derivatives. In a small proportion of the cases the conditions justify surgical intervention, the results of which have been in some few instances brilliant.

The surgical treatment of diseases of the cranial bones is frequently followed by speedy and permanent relief of headache, together with the other symptoms.

The headache of neurasthenia, hysteria, and epilepsy yields only to the proper and judicious employment of appropriate general measures. Finally, the headaches of reflex irritation usually disappear under the appropriate treatment of the local condition. It is in this group of headaches, especially those arising from disease of the eyes and nose, that some of the most satisfactory achievements of modern medicine have been obtained.

MIGRAINE.

Definition. An affection characterized by periodical nervous disturbance, consisting of severe paroxysmal headache, usually unilateral, commonly associated with nausea and vomiting and frequently with disorders of vision.

SYNONYMS. Hemicrania; Sick-headache; Neuralgic Sick-headache. English writers sometimes employ the word Megrim. The most complete systematic account of the affection is to be found in Edward Liveing's work, *Megrim, Sick-headache, and Some Allied Disorders.* Migraine is a substantive affection, a constitutional neurosis, and is considered in the present work under the head of Symptomatic Disorders, not in accordance with correct nosological principles, but simply as a matter of convenience.

Etiology. 1. Predisposing influences. Heredity plays an important part in the predisposition to migraine. There is very often a history of direct inheritance, and not infrequently it will be found upon inquiry that the disease has shown itself in several successive generations. Those who suffer from it are frequently members of neurotic families, in the history of which neuralgia, gout, or epilepsy figure. Many men of distinguished intellectual attainments, among whom are Fothergill, Sir John Herschell, Du Bois Reymond, the astronomer Sir George Airy, his son, Dr. Hubert Airy, and Anstie, have been sufferers from migraine.

This affection is decidedly more common in women than in men, the proportion being not less than three or four to one. It may show itself as early as the second year of life. Eulenberg early called attention to the frequency of the affection in childhood. It almost always shows itself in the first half of life, generally at or a little before puberty. Late childhood, the period of puberty, and early adult life constitute the epochs of onset in the greater number of cases. Much less frequently the periodical attacks first show themselves after the thirtieth year of age, and Gowers has seen a well-marked case which began at sixty.

The attacks are more frequent in winter than in summer.

Occupation exerts no great influence in predisposing to migraine. It is certainly a disease more common among refined persons and those devoted to intellectual pursuits than among the laboring classes. The difference in the liability of the two sexes is less in the higher than in the lower walks of life.

When the attacks first show themselves in early life it is difficult to assign any immediate cause for their development; when they come on during adolescence the cause may frequently be found in overwork at school, and in cases developing later in life there is very often a history of over-application, injury, shock, or wasting disease. A certain proportion of the cases are correctly ascribed to reflex causes, as disease of the nose, naso-pharynx, eyes, teeth, and the pelvic organs in females. Refractive errors and insufficiency of the eye muscles (accommodative and muscular asthenopia) are etiological factors in the development of migraine, both frequent and important.

2. The exciting causes. The conditions which are capable of inducing the paroxysm are manifold. They vary in different individuals, but usually consist of influences which produce a sudden and more or less profound impression upon the nervous system. If the usual interval between the attacks has nearly elapsed, trifling impressions will precipitate the paroxysm, whereas, shortly after the attack has passed over, the same influence, acting with far greater intensity, is without effect. The attack may be brought on by fatigue, excitement, depressing emotions, digestive disturbances, sometimes even by the eating of particular articles of diet. In some patients it follows exposure to cold; in others, certain visual impressions, as the moving figures in a procession or crowd, or sudden bright light, or sudden changes from light to darkness, or the reverse; or, again, it may follow loud or disagreeable noises, or certain peculiar odors. In many instances, however, the attack occurs at intervals, which may be constant or variable, in the absence of any appreciable exciting cause. Rachford attributes the attack to leucomain poisoning, and has found paraxanthin and xanthin in the urine.

Pathology. The essential pathological process is unknown. There are no anatomical lesions. Three principal theories have been advanced: 1. That the disease is a vasomotor neurosis, the early symptoms being due to a vasomotor constrictor (angio-spastic), the later to a vasomotor dilator (angioparetic) process. 2. Migraine has been regarded as a form of neuralgia of the first division of the trigeminus. 3. The view at present generally accepted is that of Liveing, that migraine is a fulgurating neurosis in which there are periodical discharges or nerve-storms from sensory centres, corresponding to the periodical discharges from motor centres in epilepsy. That is to say, that migraine is the sensory equivalent of epilepsy, in which the paroxysm corresponds to the epileptic seizure.

Symptomatology. Prodromes are very common. They usually last for a few hours or perhaps a day, and consist in feelings of malaise, dulness, mental depression, or somnolence. The patient is frequently able to foretell the approach of the attack. The onset very often occurs in the morning, the patient waking up with pain, usually unilateral and referred to the temple, forehead, the region back of an eye, or to the occiput. This rapidly increases in intensity and spreads until it involves the whole of one side of the head or both sides. It is intense, throbbing, blinding in character, increased by movement, light, and noise. Headache is the most constant and characteristic symptom. It is always paroxysmal and severe. In many attacks it constitutes the chief or even the only symptom; the others, with perhaps the exception of nausea, being so subordinated to it as to escape notice, or being absent altogether. Visual disturbances are present in about half the cases, usually at the beginning of the attack. They take the form of dimness of vision, flashes of light, variously colored bright or dark spots, fortification spectra, and hemianopsia, which is always homonymous. Much less common are visual hallucinations, phantasmagoria, figures of animals and the like in crowds, not unlike those of delirium tremens. Pupillary changes occur in a small proportion of the cases. They consist of contraction followed by dilatation of the pupil in the affected sides, or in rare instances of alternate rhythmical contraction and dilatation. In severe paroxysms the eyeball sometimes appears to be retracted.

Disturbances of the other special senses are rare, though temporary deafness, followed by noises in the ear and loss of taste, followed by subjective sapid phenomena, have been noted. Tingling, numbness, and partial anæsthesia have been observed in a small proportion of the cases. These sensory disturbances are usually limited to the hand and arm, face, lips, or tongue on one side. They are of transient duration, and are sometimes followed by a slight degree of motor weakness. Transient aphasia occasionally occurs, usually at the beginning of the attack. Vertigo, often not amounting to more than mere giddiness, may occur. It may be associated with tinnitus aurium.

Psychical disturbances are not constant. When present they consist of confusion of thought, depression, and slight stupor. Exceptionally there is great mental excitement. Nausea occurs in a large proportion of the cases. It may come on at any time, but as a rule does not appear until the headache has reached its height. It is frequently followed after a time by retching and vomiting, the vomited matters consisting at first of the contents of the stomach or mucus, later of bile in consequence of retro-stalsis. Hence the term "bilious headache," frequently applied to the paroxysm. This term is entirely inappropriate, for, notwithstanding the frequency of nausea and vomiting, migraine is not due to an affection of the liver. Profound nausea and vomiting are followed by great depression, and at this period of the attack the condition in many respects is not unlike severe sea-sickness. It is common for the headache to commence in the morning, nausea to come on in the course of the day, and vomiting in the evening (Gowers).

Vasomotor phenomena are present. The face is usually pale, a difference in the degree of pallor on the two sides being sometimes manifest. Toward the end of the attack there may be flushing, more marked on the side upon which the pain first shows itself, or to which it is restricted. The expression is that of acute suffering.

The pulse is small, tense and very often slow. In children the temperature is often slightly elevated.

The duration of the attack varies from several hours to a day or two. In the milder paroxysms the whole process is complete in a period of from six to twelve hours; in the more severe two or even three days elapse before the patient is well again. The decline of the symptoms is gradual rather than abrupt. This period is very often covered by sleep. As the intensity of the pain subsides, frequently after retching or vomiting, the patient falls asleep and awakes after some hours, or the following morning, refreshed, free from pain and often feeling better than before the attack.

The intervals between the attacks vary within wide ranges. The periodicity is usually irregular. Sometimes, however, it shows remarkable regularity. Thus the paroxysm may at times occur every week, even upon the same day, or every fortnight, or once a month. In women it may coincide with the menstrual periods. The attacks are in many cases absent during gestation. After the menopause or at a nearly corresponding age in men, they very often cease or become less severe, and are in some instances replaced by attacks of neuralgia or other paroxysmal nervous disturbance. I have recently seen two cases in which paroxysms of migraine have been succeeded by attacks of severe vertigo without head-pain recurring at the former intervals.

Other variations occur. Thus, headache may be absent, and the attack may be replaced by paroxysmal visual disorders or other sensory symptoms, by mental depression, or confusion, or by an attack of gout.

Diagnosis. The direct diagnosis of migraine is based upon hereditary predisposition, the periodicity, the evolution of the attack, unilateral pain, nausea, and the associated visual and other sensory symptoms. The brief course and definite conclusions of the attack are important in diagnosis.

The differential diagnosis from other forms of headache is sufficiently indicated by the above points.

In the absence of the sensory accompaniment, the paroxysmal character of the attack, its periodicity, its severity, and its definite course are to be taken into consideration.

The differential diagnosis between migraine and the headache of organic intra-cranial disease rests upon the following facts: that in the latter when intermissions occurs they are neither so long nor so complete as are the inter-paroxysmal periods in migraine; that the periodicity when present is far less definite; that the evolution of the attack is not characteristic; that the accessory symptoms are usually well marked and of a wholly different character from those of migraine. The visual aura sometimes present in *petit mal* may suggest migraine, but a careful study of the paroxysm shows differences so essential that errors in diagnosis can hardly occur.

The differential diagnosis between migraine and neuralgia of the ophthalmic division of the trigeminus rests upon clinical and pathological data so evident that they demand no discussion in this connection. It is, however, to be borne in mind that the same individual may suffer from migraine and other forms of headache or from neuralgia, or may be the victim of disease of the kidneys or organic brain disease.

Prognosis. The prognosis, as regards the expectancy of life, is good. The recurrent attacks of migraine seem to have in the majority of cases little or no unfavorable influence upon the general health. As regards cure, the prognosis is not favorable. Nevertheless, in a considerable proportion of the cases the paroxysms cease to occur at or about middle life, and in a larger proportion their intensity and frequency are diminished. Many cases are improved by treatment, the prospect of benefit being greater when treatment is instituted shortly after the development of the disease, and especially in cases due to reflex irritation, in those in which there are unfavorable conditions in the general health or mode of life that can be corrected, and in those in whom there is no hereditary predisposition to the disease.

Treatment. The children of neurotic families, or those in whom a distinct hereditary predisposition to migraine exists, should be carefully watched. The occurrence of periodical headache, or of a single attack of hemicrania, should, even in the absence of associated symptoms, suggest a careful examination of the eyes, nose, naso-pharynx, or other region of possible reflex irritation. Careful attention should be paid to the hygiene in such cases. Excessive application to study and long hours at school should be interdicted.

Treatment should be early instituted. Those causes which are known to provoke the attack should be carefully avoided. The treatment of the attack demands absolute rest in the recumbent posture in a darkened room, with complete abstinence from food. Under these circumstances alone many of the milder attacks terminate spontaneously in the course of a few hours. When the attacks are severe the early administration of antipyrin, phenacetin, sodium salicylate, sodium benzoate, and caffeine, alone or in various combinations, very often result in prompt and decided mitigation of the sufferings, and sometimes appear to shorten the attack. Moderate doses repeated at intervals of from half an hour to an hour are more satisfactory than single large doses. The chemical mixture of antipyrin, citric acid, and caffein, sold under the name "migrarin," exerts, in doses of a gramme, a very decided effect in relieving the symptoms. When the attacks are frequent these drugs gradually lose their power to alleviate suffering, and new combinations and increased doses become necessary. The internal administration of guarana, or menthol, or the exhibition of large doses of muriate of ammonia and the bromides are also followed by good results. Rachford found potassium permanganate of use, especially in a case of migrainous epilepsy, in which other treatment, medical and dietetic, failed entirely.

If the attack develop after a meal, relief very often follows free emesis, which may be produced by a draught of mustard water, a dose of ipecacuanha or the hypodermatic administration of apomorphine.

Galvanism yields decided relief in a limited number of cases. Sufferers from migraine very often acquire a knowledge of the use of analgesics and narcotics which is not without danger, and in severe attacks learn to resort to the use of chloroform by inhalation or to such powerful narcotics as morphine, codeine, and chloral, remedies that are frequently efficient, but always attended with the danger of the formation of a vicious habit and sometimes with that of immediately fatal narcosis.

The constitutional treatment during the interparoxysmal periods includes the employment of hygienic measures directed to the maintenance of the general health, carefully regulated diet, and systematic exercise. Any lesion capable of giving rise to reflex irritation, and especially those involving the eyes or nasal passages, should be promptly corrected. Long-continued courses of Indian hemp, gr. ⅓–½ (0.02–0.03), t. d.; nitroglycerin, gr. $\frac{1}{100}$–$\frac{1}{50}$ (0.006–0.003) or more t. d.; arsenious acid, gr. $\frac{1}{40}$ (0.0015), t. d.; or the bromides, gr. xv.–xx. (1.0–1.3), t. d.; or protracted alternating courses of these drugs have been followed by a diminution in the frequency of the paroxysms and in their intensity.

The daily application of galvanism to the head must be included in the list of measures of general treatment, though the proportion of cases in which demonstrable benefit has resulted appears to be small.

VERTIGO.

Definition. Vertigo, literally a turning, is a derangement of the nervous mechanism governing the relationship of the body to external objects, characterized by sensations of movement on the part of surrounding objects which are really at rest—objective vertigo; or on the part of the person himself—subjective vertigo.

SYNONYMS. Dizziness; Giddiness.

Vertigo is a symptom. Although it is in many cases a symptom of prominence, it cannot be regarded as in itself constituting a substantive disease. The term "essential" has been applied to those cases of vertigo in which no underlying pathological condition has been discoverable. Vertigo may be a symptom of organic intra-cranial disease, but it occurs much more frequently as a symptom of peripheral or functional disturbance.

Etiology. Vertigo is a common nervous symptom; it may arise in the course of diverse pathological conditions in many of which headache also occurs. Thus vertigo is a common immediate symptom of (1) mild cerebral concussion. It arises also (2) from circulatory disturbances, as cerebral anæmia and hyperæmia. (3) It occurs as a result of local nerve irritation, such, for example, as irritation or inflammation of the middle ear, or that which follows the application of electrical currents to the head. A special form of vertigo, true auditory vertigo, occurs as a symptom of disease of the labyrinth, implicating the end organs of the eighth nerve. (4) Various toxæmic conditions give rise to vertigo; thus, vertigo and headache are occasionally associated symptoms at the onset of the acute infections. Some degree of vertigo is a common symptom in acute and subacute gastro-hepatic derangements and in lithæmia. Alcohol and tobacco in persons unaccustomed to their use, or in undue amounts, produce, among other symptoms, vertigo, and in many persons the administration of narcotic drugs, particularly opium, is followed by this symptom. (5) It occurs also as a symptom of endarteritis and arterio-sclerosis; and (6) in valvular disease of the heart, especially aortic insufficiency, and in degenerative diseases of the cardiac muscle (fatty heart, chronic myocarditis). (7) In neuropathic conditions, especially neurasthenia and epilepsy. (8) In reflex disturbances, such especially as arise from disease of the visual apparatus or the stomach. (9) In organic disease of the brain. Finally (10), vertigo arises from mechanical causes, such as swinging, certain unusual postures, or rapid rotary movements, and in the irregular disturbance of the equilibrium of the body which occurs at sea.

Pathological Considerations. The equilibrium of the body is maintained under ordinary circumstances by the active contraction of muscles. The nicely adjusted and constantly changing motor impulses by which the balance of the body in its ever-changing relation to surrounding objects is secured are determined in cerebral centres in response to sensory impressions, which are as constantly in action as the motor impulses which respond to them.

The most important of these sensory impulses are visual; that is, those from the eye and its muscles, and aural, which proceed from the semicircular canals and the ampullæ. Scarcely inferior, however, in importance are the combined sensory impulses from the muscles directly concerned in the maintenance of equilibrium from the joints, from the skin and from the viscera. Any cause suddenly disturbing the continuous and systematized, though unconscious, sensory impulses from these sources, may cause a derangement of the nervous mechanism by which the body is maintained in its relation to external objects. This derangement manifests itself as vertigo. These sensory impressions, except those from the skin, are not felt in normal consciousness. It is only when they are interrupted, or when the nutrition of the cortex is suddenly lowered, that consciousness in regard to them is perverted and vertigo ensues. Hence, vertigo implies a disturbance, not a loss of consciousness. In true vertigo consciousness is always retained. Gowers has pointed out the fact that we are chiefly conscious of the effect on the action of some centre, not of the imperfection (of the centripetal impressions) itself.

Clinical Considerations. Vertigo varies in intensity from a slight feeling of imperfect equilibrium—mere swimming of the head—to the most active and distressing sensations of rapid or irregular movement or whirling of the body itself or of surrounding objects. The attack comes on suddenly, and is usually of short duration. The floor or the bed upon which the patient is lying appears to rise and sink, or objects whirl around, usually in a definite direction—objective vertigo; or the patient himself appears to be whirling around or rising and sinking in space—subjective vertigo. These sensations are often accompanied by compensatory movements on the part of the patient, which may result in a fall. There is mental confusion, together with a sense of alarm and often faintness. Consciousness is impaired but not lost. There may be nausea or vomiting; these symptoms are almost always present when the vertigo is severe. The attacks may continue to recur whilst the condition to which they are due persists. Hence vertigo may become chronic, and to the severer form of persistent vertigo the term *status vertiginosus* has been applied. The patient is often compelled to keep the eyes closed. Vertigo is increased by change of posture and particularly by sudden movements of the head. It is very often diminished, or ceases altogether, when the patient assumes the recumbent posture.

The above-named conditions in which vertigo often occurs as a symptom may be taken together as the basis of an etiological classification. Though almost always regarded by the patient and his friends as serious, vertigo is in many cases an insignificant symptom.

The following forms of vertigo require brief separate consideration: Aural vertigo, including true auditory vertigo—Ménière's disease; toxic vertigo; arterio-sclerotic vertigo; cardiac vertigo; neurotic vertigo, including that of neurasthenia, epilepsy, the so-called stumbling or paralyzing vertigo and paroxysmal vertigo; reflex vertigo; the mechanical vertigoes, and, finally, the vertigoes which arise in coarse intra-cranial disease.

AURAL VERTIGO. A distinction must be made between the vertigo which is sometimes symptomatic of irritation or disease of the middle ear or of the external auditory canal, and that which arises in lesions of the eighth nerve involving its end-organs in the labyrinth or its nuclei. The latter constitutes true auditory or labyrinthine vertigo, and is the chief symptom of Ménière's disease.

Vertigo often arises from the pressure of accumulated cerumen in the external auditory canal, especially if the mass has become slightly displaced. Vertigo may follow the sudden forcing of air against the membrana tympani from a blow upon the ear, or the entrance of water in diving or surf-bathing, or the forcible injection of water in syringing. Vertigo may also arise from irritation of the Eustachian tube in the surgical treatment of the middle ear. This symptom also occurs, though it is not common in cases of otitis media. The vertigo arising under the above circumstances is usually slight and transitory. Whether it is directly due to local irritation, or is reflex, has not yet been determined.

MÉNIÈRE'S DISEASE. An affection characterized by noises in the ear, sudden attacks of vertigo with nausea and vomiting, and nervous deafness, which in many cases is progressive. The attacks are often apoplectiform with momentary loss of consciousness.

This affection is an important, very often a serious, disease. It must be regarded as a substantive affection of which vertigo is the chief symptom. It was first described as such by Ménière in 1861. The term "Ménière's disease" is frequently applied to attacks of vertigo occurring in any disease of the auditory apparatus. It should be restricted to the affection characterized by the complexus of symptoms about to be described.

Age plays an important part in the predisposition. The affection is very rare in early life. In a large proportion of the cases the attacks first show themselves between forty-five and fifty-five, but they may come on much later. Men suffer more frequently than women. Nothing is known of the exciting causes. The disease is paroxysmal, the attacks occurring at irregular intervals, and very often in series, several of which may take place in one day or on successive days. Such series or single attacks may be separated by intervals of weeks or even months. The attack is very frequently apoplectiform, beginning suddenly with tinnitus aurium and subjective or objective vertigo of such intensity that the patient, in order to prevent himself from falling, is obliged to immediately catch some support or to sit or lie down. If loss of consciousness occur it is as a rule momentary. Occasionally ocular symptoms accompany the attack. These consist in diplopia or nystagmus. Forced movements may occur, and in the intervals of frequent attacks there is an impairment of equilibrium, so that the patient walks with difficulty. The attack is usually of short duration. As the vertigo passes off the patient is pale, breaks into a profuse sweat, suffers from nausea, or there may be actual vomiting. As a rule, there is no disease of the middle ear. When present, the association is merely accidental. The deafness, which is nervous, usually affects one ear only. It is progressive, but never complete. When deafness becomes complete, the vertigo ceases, the end-organs of the nerve being destroyed.

Three principal theories have been suggested to account for the phenomena of Ménière's disease:

1. That the symptoms are due to lesions of the labyrinth. There is progressive degeneration of the nerve or its end-organs.

2. That the disease is a vasomotor neurosis of the vessels of the labyrinth. This view finds some support in the paroxysmal character of the affection and in the fact that acute lesions with hemorrhage have been found upon post-mortem examination.

3. That the primary trouble consists in an affection of the centres for hearing and equilibration.

Of these three, the first, namely, that the symptoms are due to lesions of the labyrinth involving the end-organs of the eighth nerve, is at present generally accepted.

The direct diagnosis of Ménière's disease rests upon the paroxysmal vertigo, the apoplectiform seizure, the occurrence of tinnitus, nausea, and vomiting and the nervous deafness, usually progressive. The differential diagnosis between the vertigo which is so prominent a symptom, and other forms of vertigo must in the main depend upon the association of the foregoing symptoms, the paroxysmal nature of the attack and the absence of other pathological states usually attended with vertigo.

The prognosis is uncertain. A small proportion of the cases terminate, after a variable duration, in complete recovery, with total loss of hearing in the affected side; much more commonly the disease proves persistent and intractable, and, with periods of exacerbation and improvement for which no explanation is to be found, continues throughout life. In some instances the symptoms are so severe that the patients become bed-ridden.

The treatment of Ménière's disease does not rest upon a very satisfactory basis; nor are the results in general brilliant. Nevertheless, many cases are for a time at least, greatly benefited by systematic medication. As nothing is known of the cause, nothing is to be said of prophylaxis. The general health must be looked after as in every chronic disease. The bromides are useful in doses of gr. xx-xxx 1.5 t. d., or, when other plans of treatment are employed, in a larger single dose at bedtime. Potassium iodide may be administered in all cases in which there is a history of syphilis; its use has occasionally been followed by improvement where no such history has been elicited. The method of Charcot consists in the use of quinine pushed to cinchonism; the drug is then discontinued, and as the physiological effects pass away improvement is noted. Upon the recurrence of the attacks, the quinine is again given in the same manner. Quinine may be given in connection with the bromides or with hydrobromic acid. The salicylates pushed to the production of their physiological effects have been followed by improvement. Thus, the sodium salicylate may be given in gr. x-xv (0.6-1) doses t. d. until persistent tinnitus or other evidence of its effect upon the nervous system arise, and then stopped. Pilocarpine and ergot have been employed, and I have seen decided relief follow the long-continued administration of ergotin and cannabis indica.

TOXIC VERTIGO. The vertigo which occurs at the onset of the acute infections is usually associated with headache. It is always of secondary importance as compared with the general symptoms, and quickly passes away. It requires no special treatment.

Vertigo is a symptom of gastro-hepatic catarrh, or *biliousness*. This form of vertigo is usually more troublesome on rising in the morning, and is one of an annoying group of symptoms to which persons of bilious temperament and sedentary lives are peculiarly susceptible, especially if they are given over to the pleasures of the table. Constipation, nausea, a furred tongue and foul breath, loss of appetite, headache, and depression of spirits make up the clinical picture. The occasional vertigo of lithæmic patients is an allied condition. Vertigo, sometimes of high grade, also occurs as a symptom of acute indigestion, and especially that following a surfeit. Many vertigoes now known to be due to other causes were at one time ascribed to gastric disorder, and the *vertigo a stomacho laeso* of Trousseau occupies a much less important place in nosology than formerly. Gastric vertigo is probably in part reflex, in part toxic.

The vertigo caused by narcotics, alcohol, tobacco, opium, cocaine, nitroglycerin, and many other drugs is transitory and demands merely passing consideration. Idiosyncrasy and habit play a large part in determining the occurrence and degree of vertigo as a drug-symptom. A peculiarity of the vertigo which in many persons follows the administration of opium and its derivatives is the extent to which it is influenced by posture, being wholly absent while the patient remains recumbent and at rest, and coming on with distressing intensity upon movement or in the erect position.

CARDIO-VASCULAR VERTIGO. Vertigo is a symptom of cerebral anæmia. When the cause does not act instantaneously, swimming of the head precedes syncope. This is the case in sudden blood-loss. Mechanical defects in the circulatory apparatus produce this form of vertigo. Cardiac asthenia, whether due to defective innervation or changes in the heart-muscle (myocarditis; fatty heart), is frequently attended by some degree of vertigo. Upon effort, or during excitement, or when the tidal blood is accumulated in the abdominal organs during the digestion of a too hearty meal, the enfeebled heart may fail to adequately supply the brain with blood, and vertigo, followed by faintness, result. Under such circumstances valvular defects, and in particular aortic insufficiency, may produce like effects. Sclerotic changes in the branches of the cerebral arteries, producing local anæmia and impairment of nutrition, cause vertigo. Such changes may be brought about by disease or by old age. The association of vertigo and tinnitus aurium as persistent symptoms is sometimes a very distressing senile condition.

In this connection is to be noted the vertigo of anæmia and chlorosis, which occurs especially upon exertion, and is due to cerebral anæmia. Vertigo is occasionally a marked symptom in leukæmia.

NEUROTIC VERTIGO. (*a*) Epileptic vertigo occurs as an aura; also in petit mal. (*b*) Neurasthenic vertigo is a common and distressing symptom. It occurs in attacks that are frequent, but not usually severe nor prolonged. Slight nausea may be present, but vomiting does not often take place. The vertigo of neurasthenia is frequently reflex—ocular or gastric. It is usually subjective. (*c*) Stumbling or paralyzing vertigo. There is a sudden loss of power in the legs without the ordinary sensations of vertigo, though it is probable that there is momentary impairment of consciousness. This symptom has been noted in exophthalmic goitre, and has occurred endemically in summer in certain cantons of Switzerland. (*d*) Paroxysmal vertigo occasionally occurs in individuals of nervous temperament after excitement or excessive fatigue. The attacks come on suddenly, and sometimes last for several hours. The sensation of vertigo is very distressing; it is accompanied by intense nausea and vomiting. All the symptoms are increased by movement and in the upright posture. The attacks present all the phenomena of sea-sickness, with which they have been compared.

REFLEX VERTIGO is much less common than formerly supposed. Ocular vertigo may in rare instances occur alone, more commonly in connection with brow-pains or other forms of headache as a symptom of defective refraction or want of harmonious action in the ocular muscles—eye-strain. The close central anatomical relationship of the vagus and auditory nerves has been invoked to explain the occurrence of reflex vertigo in gastric derangements.

MECHANICAL VERTIGO. This symptom quickly arises upon certain movements, as sudden lowering of the head, whirling around, or swinging, in persons not used to them, and as part of the morbid complexus in sea-sickness, car-sickness, and in very susceptible individuals from the motion of elevators. Exceptionally chronic vertigo of mild kind troubles those who, as elevator boys, are continuously exposed to such movements.

THE VERTIGO OF INTRA-CRANIAL DISEASE. Vertigo is a very common symptom in disease of the brain and its meninges. It is, however, of inferior importance as a rule, both as regards the subjective sensations of the patient and in diagnosis, to many of the symptoms, such as headache, vomiting, mental hebetude, etc., with which it is usually associated. It occurs in cerebral anæmia and hyperæmia, in meningitis, in acute and in chronic or latent abscess of the brain, in cerebral arterio-sclerosis, and as a premonitory symptom in the acute softening due to thrombosis; as a general symptom of cerebral irritation in tumor of the brain, and as a symptom of special significance in tumor and other coarse lesions of the cerebellum. Vertigo, so common in arterio-sclerosis, acquires especial importance in the diagnosis of cerebral syphilis.

Diagnosis. Vertigo is a morbid phenomenon *sui generis*, and may be recognized without difficulty from the description of the patient. It is to be regarded in all cases, even in those in which the underlying pathological condition is not at once discoverable, as a symptom which acquires significance and importance chiefly as indicating the disease of which it is a manifestation. The character of the vertigo is to be carefully investigated; whether it be subjective or objective, intermittent, paroxysmal, or chronic. Especial attention must be given to the associated symptoms. The causal diagnosis can alone afford a basis for intelligent prognosis and rational treatment.

Prognosis. The prognosis varies according to the cause. Many forms of vertigo are amenable to treatment. Those cases in which the symptom is due to organic disease of the brain or to epilepsy are not hopeful. Vertigo caused by labyrinthine disease ceases, as a rule, when the hearing upon the affected side is wholly lost.

Treatment. The treatment of vertigo must be determined by the cause. The attack is best managed by rest in the recumbent posture and the administration of the spirit of ammonia. Among merely symptomatic remedies the bromides are the most useful, their proper employment being usually followed by temporary good effects.

The vertigo of neurasthenia yields to management by rest, a proper and sufficient dietary, laxatives, the mineral acids, and strychnine; that of gastric disorder to laxatives, simple bitters, and a regulated diet. The actual condition of the stomach must be ascertained by the modern methods of gastric diagnosis, and treated accordingly; that of biliousness, lithæmia and gout by appropriate regimen and remedies; that of old age and arterio-sclerosis is partially relieved by nitroglycerin, potassium iodide, systematic purgation, and a restricted diet, from which meats and sugars are largely excluded. In the last group of cases cardiac tonics may be indicated. In persistent vertigo, the cause of which may not be obvious, counter-irritation over the nape of the neck or the mastoid process is occasionally useful.

INSOMNIA.

Definition. Insomnia is a term used to designate a condition of disturbance of the nervous system, characterized by habitual incomplete sleep or by periods of entire absence of normal sleep.

SYNONYMS. Sleeplessness; Abnormal Wakefulness; Ahypnosis.

Sleep is a physiological condition in which consciousness is suspended, and during which the body is in repose while its constituent elements undergo nutritional repair. The activity of the cells of the cortex of the brain is in abeyance; the higher cerebral functions, and among them consciousness, cease. The muscular system and, to some extent, other organs share in the repose. On the other hand, the intimate physiological activities which constitute processes of repair go on, with the result that the awaking is attended with sensations of refreshment and renewed vigor. The recurrence of sleep under normal conditions is rhythmical; it is a manifestation of the normal alteration of irritability and loss of irritability characteristic of cell life in general. In the highest sense the changes which bring about sleep, and equally those which bring it to an end, are chemical and intra-cellular. The blood conveys nutritive principles, and may supply drug principles, as caffein, which excite irritability and postpone sleep; or which, as morphine, allay it and hasten sleep. Some degree of cerebral anæmia is present; but there is no proof that it is the cause of sleep, the probability being that the deficiency of blood is merely the result of the suspension of functional activity. In so far as the circulation has to do with causing or preventing sleep, it is not mechanically, that is to say, not in the increase or decrease in the amount of blood supplied to the brain, but in the composition of the blood, that it acts. Thus drowsiness may be present with all the signs of cerebral congestion or under circumstances in which there is manifest anæmia of a high degree; while the presence in the blood of waste products, various drugs or poisons, is apt to cause drowsiness, pathological sleep, stupor, or coma, according to their amount in relation to the body-weight of the individual. Herein is to be sought the essential difference between normal sleep and other states in which consciousness is lost and which are purely pathological. These states of sleep, stupor, and coma are not, as some have taught, simply the expression of different degrees of suspension of the functions of the cerebral cortex, but sleep and the others, notwithstanding their points of resemblance in the sick-room, are essentially different conditions, due to different causes, producing diverse effects in the economy and terminating in results that are not to be compared.

Normal sleep, both in character and amount, varies with age, sex, individual peculiarity, occupation, and to some extent with race and climate. In the newborn sleep is a nearly continuing state, interrupted in health only by the taking of food and the offices of the toilet; a little later there are periods of wakefulness, and the healthy child of two years passes half its time in slumber. The adult requires from seven to eight hours of sleep daily; aged persons not more than five or six hours. Women need, as a rule, more sleep than men. Individual peculiarities are observed in regard to the amount of sleep required, just as as in the amount of food necessary to health and comfort. Thus there are persons to whom nine or even ten or twelve hours of sleep are essential, while others seldom sleep more than six hours out of the twenty-four. Nor is this difference to be explained by the completeness of the sleep, since those whose sleep is longest often sleep deeply, and those who habitually require short hours of sleep are easily aroused. Those whose occupations keep them much in the open air and involve prolonged muscular effort take far longer hours of sleep than brain workers and those of sedentary habits, and the former class sleep more soundly. The Northern races are accustomed to longer hours of sleep than those who dwell in more favored climates.

Under normal circumstances the sleeper is readily aroused. Sleep when interrupted tends to recur, but may be postponed by vigorous external impressions or by the power of the will. After a prolonged period, however, the need of sleep asserts itself, and no sense-impression, no moral stimulant, no effort of the will is sufficiently powerful to keep the exhausted sufferer awake. Continuous insomnia terminates in the course of two or three weeks in fatal exhaustion.

The term insomnia cannot be properly employed to designate the temporary wakefulness due to powerful external impressions. Persons who cannot sleep because they are disturbed, sleep well enough when permitted to do so. Those who suffer from insomnia sleep badly or not at all under the most favorable circumstances.

Etiology. Insomnia may be simple or functional and symptomatic.

(*a*) Simple or functional insomnia occurs in neurotic individuals and especially in overtaxed brain-workers. This form includes hereditary and habit insomnia. The disorder of sleep is not dependent upon any discoverable underlying disease or morbid condition, and is frequently present in persons whose general health is in other respects fairly good. After a time habitual wakefulness produces impairment of nutrition, brain exhaustion, and very often severe mental symptoms.

(*b*) Symptomatic insomnia. Habitual inability to sleep is an important symptom in a great variety of morbid conditions. The causes may be arranged in groups as follows: Nervous, psychic, cardio-vascular, diathetic, toxic, and infectious. Not infrequently two or more of these conditions act in combination.

Insomnia is a prominent secondary symptom in painful diseases, especially cancer, aneurism, and the intractable neuralgias; also in acromegaly.

Insomnia is frequent in many diseases of the nervous system. It occurs in neurasthenic subjects usually in the form of irregular, fitful, and unrefreshing sleep, troubled with dreams, and as complete wakefulness after fatigue or excitement. It is common in the various forms of insanity both in the prodromic period and in the declared disease. It is sometimes prolonged and distressing in general paralysis of the insane, but is not common in focal lesions of the brain. It is a prominent symptom in acute delirium; while in melancholia sleep is broken, unresting, disturbed by dreams of the most distressing character; often for prolonged periods absent altogether. Diseases of the heart and arteries are not rarely attended by sleeplessness, partly due to cerebral anæmia, partly to the state of the blood, and partly to the inability of the patient to assume the recumbent posture. As these factors increase, wakefulness is progressively superseded by somnolence, stupor, and coma. In this connection we note also the insomnia of chronic nephritis, especially those forms in which artero-sclerosis is pronounced. The want of sleep is here due to mechanical defects in the blood-supply and to toxæmia (uræmia), while the insomnia so marked in certain cases of acute nephritis must be ascribed chiefly to toxic principles circulating in the blood. In anæmia and chlorosis there is frequently sleeplessness by night and drowsiness by day. Diathetic states, such as undeveloped gout and lithæmia are common, often unsuspected causes of protracted and tormenting sleeplessness. Disturbed sleep, rather than absolute insomnia, is among the symptoms of gastric disorder.

Tea and coffee have in many persons, and especially in those unaccustomed to drink them at night, the power of inhibiting sleep; caffeine has the same effect. Alcohol, in moderate doses, is usually sleep-compelling, while excesses stupefy, and complete insomnia is a conspicuous phenomenon in delirium tremens and alcoholic mania. The onset of the acute infectious diseases is often marked by wakefulness. The early stages of enteric fever, influenza, croupous pneumonia, afford striking instances of the insomnia caused by the toxic agents of the acute febrile infections. Sleeplessness is one of the rare nervous symptoms of secondary syphilis, and is sometimes the precursor of syphilis of the brain. Insomnia occurs in certain cases of acute malaria and in the malarial cachexia, and is sometimes a stubborn and troublesome symptom of trichinosis.

The sleeplessness of the period of convalescence from acute disease must be attributed not to toxæmia, but rather to the general asthenia of the stage of recovery.

Women are less prone to insomnia than men, children less than adults, and open-air workers than those engaged in sedentary occupations.

Symptomatology. Insomnia shows wide variations in kind and completeness. It may take the form of troubled and unrefreshing sleep of only a few hours' duration, or of fitful and broken slumber with intervals of painful wakefulness, or sleep may be absent for days together. A very common form of simple insomnia is that in which the patient on going to bed falls asleep, but awakes in the course of two or three hours, and tosses until morning. In the insomnia of neurasthenia there is often great but irregular mental activity, in which the cares, anxieties, and worries of daily life are rehearsed with torturing iteration. With this there is also great restlessness, a peculiarity also present in the sleeplessness of insanity. Querulousness and irritability are associated moods. Insomnia is comparatively uncommon in children; when present it is attended with great restlessness and mental excitement, and is of greater significance than that of adults. The insomnia of old persons is usually tranquil and unaccompanied by excitement or irritability.

The differential diagnosis between simple insomnia and symptomatic insomnia must be made by exclusion.

Treatment. The treatment is symptomatic and causal. Both forms of insomnia, the simple and the symptomatic, may require symptomatic treatment for their successful management; but many cases of the latter recover under treatment properly directed to the relief of the morbid condition of which the sleeplessness is a symptom. The subject naturally arranges itself under three general headings: The general management of cases of insomnia, medication by means of hypnotic drugs, and the treatment of the underlying disease.

The General Management of Insomnia. Simple insomnia is in many cases the outcome of an overtaxed nervous system, too much work or too much worry; or it is the result of an irregular and self-indulgent life, a mere vicious habit. Under such conditions the indications for treatment are obvious, but not often readily carried out. It follows, however, that diminished work, especially in the field of intellectual endeavor, lowered ambitions, the renunciation of efforts after the practically unattainable, a systematized and regulated life, not only enable many individuals to recover the sleep habit which they have lost, but also secure to them a measure of happiness unknown in the too earnest struggle for objects beyond their powers or circumstances. Simple hygienic measures that invite calm and refreshing sleep are early rising, light meals of wholesome food, moderate exercise in the open air, systematic bathing and great regularity in everything. Mental work should be restricted to the early day and limited to four or six hours. The afternoon should be spent in out-door exercise; the evening occupied with conversation or amusements that do not greatly tax the attention. Much time should be devoted to the preparation for bed. Let the patient potter about till he is sleepy with prolonged attention to the mindless details of the toilet; then, in a cool room, with warm feet, a hard bed, and light covering, his chances of sleep are at their best. Above all, as the head touches the pillow let thought and the problems of the day be banished and the attention given to the monotonous, inarticulate sounds of the outer world. Hot foot-baths, warm baths with cold affusion to the spine, muscle beating and massage are measures as likely to arouse and excite the nervous system as to tranquillize it, and, while sometimes useful, are more frequently of doubtful advantage. A glass of warmed milk, a cup of hot bouillon, a toddy or a glass of beer often aids in the invitation to sleep. Sometimes the monotonous reading of a familiar book may be useful, but it is better for the patient to be wholly left to himself. Many persons who suffer from insomnia greatly exaggerate, without meaning to do so, the loss of sleep.

Tea or coffee taken late in the day are hostile to sleep.

Hypnotic Drugs. Drugs that induce sleep are to be used with caution. The moral effect of their habitual employment is bad and tends to invalidism. Their unguarded administration exposes the patient to the danger of the formation of the drug-habit, a danger increased on the one hand by the neurotic temperament and on the other by familiarity with narcotics and their dosage. They are, however, in many cases indispensable. One or another may be employed in adequate amounts for a few nights, and then abruptly or gradually abandoned. Occasionally good results are obtained by a full dose every second or third night; or, again, a very moderate dose may be given for a long time with satisfactory effects. The details of the proper employment of narcotics to induce sleep require great tact on the part of the physician. Insofar as possible the precise drug and the dose used should remain unknown to the patient, and from time to time different hypnotics employed. Physicians, nurses, and apothecaries who suffer from insomnia are especially liable to the formation of habits of vicious dependence upon narcotics. Opium and its derivatives and chloral are in this respect most seductive and dangerous. Chloral is, in the opinion of the writer, the surest of our modern hypnotics and by far the most satisfactory in its effects.

The immediate danger of cardiac depression is not to be overlooked. It can be guarded against, and its effect reinforced by the simultaneous administration of some alcoholic beverage. In exceptional cases chloral is followed by great excitement and wakefulness or by symptoms of acute gastric catarrh. Chloralamid is less active, less sure in its effects, but altogether safe in proper doses. I have used it of recent years more than any other drug as a hypnotic. Sulphonal, which should be dissolved in hot water, milk, or bouillon, which is swallowed as soon as cool enough, is uncertain in its effects and often followed by headache and prolonged dulness after waking. Paraldehyde is safe and sometimes a very effective drug, but its disagreeable taste and still more disagreeable, persistent odor stand in the way of its general employment. Hyoscine hydrobromate is to be chosen in those cases, and especially in the insomnia of the insanities, in which great motor excitement is present. The virtues of urethane, amylene hydrate, somnal, trional, and tetronal as sleep-compelling drugs are now established.

Of inferior value, but still most useful in proper cases, are antipyrin in large doses, the bromides and valerian.

Opium, morphine, and codeine alone or in combination with chloral, hyoscine, or the bromides are to be reserved for cases otherwise unmanageable, the insomnia of painful diseases, the incurable and the aged.

But the inefficacy of poppies, mandragora, and all the drowsy syrups of the East in certain cases has long been known. In some of these, life in the wilderness, and what Mitchell has called the mindless labor of the camp, the rod, the gun, the axe, have wooed back sleep.

The Treatment of the Underlying Disease. The obvious indication for treatment in symptomatic insomnia is to be found in the existing pathological condition. To discuss the details of such treatment does not fall within the scope of the present article. Suffice it to say that the physician should be on the alert to discover latent or obscure maladies, the phenomena of which are often overshadowed by sleeplessness and its attendant exhaustion and nervous irritability. Among these are gastric, cardiac, and uterine neurasthenia, inflammatory and degenerative diseases of the kidneys, diathetic states, especially lithæmia, the alcoholism of secret tippling, the opium and cocaine habits and syphilis.

DISORDERS OF SLEEP.

Normal sleep is usually comparatively light at first, reaching its deepest stage in the course of an hour or more after it begins, and becoming lighter again before spontaneous waking. To this general rule there are, however, many exceptions, some persons habitually, and especially after fatigue, falling at once into deep and prolonged sleep, or waking abruptly from sleep, which appears to have been continuous and profound. The period of light initial slumber has been termed the *prædormitium.* At this time many persons of nervous constitution, and in particular brain-workers, experience momentary muscular startings or shocks, like the discharge of a Leyden jar, which affect the whole body, and are accompanied by disagreeable sensations in the head. These attacks are sometimes violent, and two or three of them may occur before sleep is fully established.

DREAMS. It is probable that dreaming does not occur in complete sleep. It is certain that dreams are common and vivid in proportion as sleep is light, irregular, and fitful and in the lighter sleep which ordinarily precedes waking. The assumption from these facts is warrantable that dreams occur when the suspension of consciousness is incomplete. The starting point of dreams is usually some sensory impression, often disagreeable, arising from local causes, as an uncomfortable posture, light or sound which is perceived, but does not arouse, an overloaded stomach, a distended bladder or rectum, a lesion which is painful, or a condition which interferes to some extent with the functional play of some organ, as the heart or lungs. Such impressions, not fully recognized and correlated in the sub-consciousness of sleep, set in motion trains of ideas which are irregular and bizarre, and which not infrequently centre about some controlling thought or memory of waking hours. These facts serve to render intelligible the occasional startling appropriateness and apparent significance of dreams and justify the importance ascribed to them by ignorant persons and uncivilized tribes. They account to some extent for the definiteness of dreams in certain morbid conditions, as in acute indigestion, the dreams of which are usually of a frightful character, and of valvular disease of the heart, in which, when there is rupture of compensation, the patient often dreams of dying. They account also for the recurrent dreams of fixed character which sometimes occur before the definite signs of organic disease show themselves, and which have been called prodromic dreams.

NIGHTMARE. A frightful dream, attended with sensations of great physical and mental distress, which take the form of an oppressive weight upon the chest, intense fear, horror or anxiety, and inability to move or cry out. The attack ends in a groan and the recovery of consciousness. The synonym Incubus was originally the male demon supposed to attack women in their sleep, lying on them, and causing nightmare. The corresponding female demon that attacked men was called Succuba. Ephialtes, literally one who leaps upon, also expresses the idea of oppression. The attack occurs in incomplete sleep. Nervous persons, especially after unusual fatigue or excitement, those suffering from chronic wasting affections, malaria, cardiac diseases, and anæmia, or chlorosis are particularly liable to nightmare. It occurs more frequently in females than in males and in the young, though exceptionally persons are encountered who suffer from attacks of nightmare throughout life. The exciting cause may be gastric repletion, any indigestible article of food, excesses in alcohol or tobacco, or the menstrual molimen. Sleeping upon the back or in any constrained or uncomfortable posture increases the liability to the attack.

NIGHT TERRORS. Pavor nocturnus occurs in young children as a paroxysmal disturbance of sleep. It presents points of resemblance to nightmare and to somnambulism; but differs from the former in the gradual rather than abrupt subsidence of the attack and the persistence of terror and distress after waking. It differs from the latter in the fact of the gradual waking, the less complete automatism and the element of terror. The attack usually comes on early in the night. The child starts up in bed, screaming with fear. It runs to its parents and seeks protection, trembling and sobbing. Usually the dream-images are indefinite; sometimes they are animals, hobgoblins or monsters, or, again, persons seeking to carry it off, the form being largely determined by the stories and conversation of the nursery. Although the child rises from its bed, it is for a time evident that consciousness is in abeyance. The awakening is gradual; the terrors of the night pass, and the little one soon sobs himself to sleep again. Night terrors occur usually in delicate or badly-nourished children. They may be brought about by eye-strain, intestinal parasites, dentition, an attack of indigestion, or may follow fatigue, vivid impressions or intense emotion during the day. They are not, as a rule, of much importance; nor do they necessarily indicate any serious or permanent morbid condition of the nervous system. As the child grows older the liability to the disorder diminishes, though in exceptional cases occasional paroxysms occur later in life. In rare instances the attacks present characters suggestive of nocturnal epilepsy.

SLEEP-DRUNKENNESS (SCHLAFTRUNKENHEIT.) This term has been applied to a rare condition resembling maniacal delirium, which occurs on waking from profound sleep. The sufferer does not recognize his surroundings nor his friends; he is excited, boisterous and incoherent, and labors under delusions of immediate danger to his life or liberty, in consequence of which acts of violence may be done.

SOMNAMBULISM (SLEEP-WALKING.) A disorder of sleep in which consciousness and volition are suspended while the activity of certain nerve-centres is exalted and complex co-ordinated movements are automatically performed. Somnambulism is, in fact, an acted dream.

It occurs chiefly in adolescents and young adults, usually the offspring of neurotic parents. The condition itself, in some instances, affects successive generations in the same family. It is more common in females than in males. The victims are often sensitive and impressionable persons. The exciting causes are those which ordinarily give rise to dreams and other disorders of sleep; they include indigestion from excesses at table or unwholesome articles of food, uncomfortable posture, especially sleeping with the head too low, and intense excitement or violent distressing emotions during the period preceding sleep. The attacks are apt to be repeated, and when habitual, often occur without discoverable cause, frequently observing a regular periodicity of some days or weeks.

The difference between sleep-talking and sleep-walking is simply a question of the automatic activity of different centres during the unconsciousness of sleep. The former is of common occurrence and attracts little attention; the latter is fortunately somewhat rare and acquires importance from the completeness of the automatism. The attack is usually brief, but may continue for an hour or more, during which period difficult and complicated actions are performed with much of the appearance of conscious intention. Yet the eyes are closed, or if open, they are staring and fixed, the expression is blank and impassive and the ears apparently deaf to every sound. The somnambulist may traverse difficult passages or walk upon roofs. From these excursions, if undisturbed, he returns to his bed and falls into quiet sleep. He is aroused with difficulty, and may when disturbed, become violent. On waking he has no recollection of his wanderings. Much confusion may arise in consequence of actions performed during somnambulism. The subject has been utilized by novelists and playwrights. It may acquire medicolegal importance, and not infrequently serious and even fatal injuries are sustained by persons falling from windows during the somnambulistic state.

MORBID SLEEP. Drowsiness during the ordinary waking hours may occur as the result of habitual insufficient sleep, or it may be a mere indolent habit. Usually, however, it is symptomatic of cerebral malnutrition or some form of toxæmia. Hence, it is common in old persons with feeble hearts or diseased bloodvessels and in malarial, anæmic, cholæmic and diabetic toxication. The drowsiness after a full meal, that produced by exposure to intense cold, that which follows excesses in alcohol, are familiar conditions. The impure air of crowded assemblies causes drowsiness, and its effects are augmented by quiet and lack of interest in the proceedings. Obese persons are apt to be drowsy. Morbid sleep, due to reflex irritation, is very rare. Cases have, however, been reported in which prolonged deep sleep has ceased upon the discharge from the bowel of masses of lumbricoid worms.

NARCOLEPSY. This term has been applied to abnormal sleep of unknown cause. The attacks are often of short duration with intervals of complete wakefulness or they may be prolonged and continuous. In some instances they occur many times a day, or daily; or, again, at longer intervals. Sometimes the spells of sleep are profound, lasting for days continuously. Such cases may present equally prolonged and remarkable periods of continuous wakefulness. Instances of sleep, apparently idiopathic, progressively deepening, and finally terminating in death, have been noted.

Morbid sleep occurs in organic brain disease, as syphilis, tumor, or arteriosclerosis. It is a symptom in insanity, occurring both in the prodromic period and in the developed state. More commonly the condition in organic brain disease and insanity is one of hebetude or of partial coma, which presents merely a superficial resemblance to sleep.

THE SLEEPING SICKNESS OF AFRICA ("NELAVAN;" MALADIE DU SOMMEIL; SLEEPING DROPSY). A peculiar disorder, probably infectious, endemic among the negroes of the west coast of Africa, and occurring occasionally in the West Indies. The symptoms consist of malaise, headache, drowsiness, at first after meals; later, continuous and progressively deepening congestion and prominence of the eyes, disturbances of gait and cervical adenopathy. The disease is very fatal, death taking place during coma or in epileptiform convulsions. No characteristic lesions are found upon examination post-mortem.

NIGHT PALSY (SLEEP PALSY.) A paræsthesia affecting one or more extremities upon waking, The sensation is usually that of numbness, which may be accompanied with tingling. Its distribution usually involves one arm or leg, or it may affect the whole body. In a little while it passes away. It resembles the acroparæsthesia which occurs at or after the climacteric in nervous and hysterical women, and is probably an allied condition. It is not very important.

Paroxysmal disturbances of the nervous system, both physiological and pathological, frequently occur during sleep. Thus seminal emissions, the venereal orgasm, urinary incontinence, are accidents of sleep; while epileptic seizures are not uncommon, and sufferers from asthma and migraine very often awake in an attack.

The treatment of the disorders of sleep must be directed to the removal of their cause. Attention to matters of hygiene is of first importance. Open-air exercise, a readily digestible and nutritious diet, light suppers, systematic bathing, a regulated life, free from exhausting mental work and undue excitement are in many cases all that are required to end the annoyances of evil dreaming, nightmare, pavor nocturns, and somnambulism. Normal and undisturbed sleep is favored by a cool, well-ventilated chamber, a hard bed, light covering, and moderately high pillows. In older persons change in surroundings and the fatigue of hunting or fishing exert a very favorable influence. Drugs are disappointing, but good effects often result from the combined use of cardiac tonics and laxatives, or, in proper cases, from the administration of alkalies and the salicylates. Some of the accidents of sleep may be prevented by a device such as a spool fixed to a belt at the spine in such a way that sleeping upon the back is rendered impossible.

CHAPTER XXXIII.

SURGERY OF THE BRAIN, SPINAL CORD, AND NERVES.

By W. W. KEEN, M.D.

THE surgery of the nervous system is peculiar in several points:

First, in respect to the localization of different functions in various parts of the brain and cord—a modern discovery of the greatest importance.

Second, in respect to the distant influences of operative procedures, in that not only the part operated upon undergoes healing, inflammation, and other surgical processes, but that the operative procedures may be followed by results in far distant organs. Thus, if a certain portion of the brain is excised, we will have paralysis in face, arm, or leg; if a nerve or the cord is operated upon, sensation in the skin is altered or destroyed, and motion in the muscles supplied by that portion of the cord or by the nerve operated upon may also be altered or destroyed. This is in sharp contrast to the surgical results in other organs, in which, as a rule, the consequences are limited to the organ operated upon.

Third. It is peculiar by reason of the fact that, while the nerves are easily and safely accessible in their places amidst the soft parts, the brain and cord are contained within a bony case, the skull, or a bony canal, the spinal column, making access to the contained organs difficult and not seldom dangerous.

Fourth. It is peculiar in reference to many points of special surgical technique.

I propose, therefore, to treat in this chapter (1) of the general surgical technique of operations on the brain, and then of such special surgical affections as require particular treatment; (2) the general technique of operations on the spine, to be followed by a brief description of the special technique of particular diseases or injuries.

I shall include, also, in connection with the brain, not only the neurological affections already described in the preceding portions of this work, but also briefly consider the strictly surgical affections, such as fractures of the skull, which are important, not so much by reason of the bones which are broken as by reason of the possible injury to the contents of the skull, and, in connection with the spine, the more strictly surgical injuries, such as fractures and dislocations of the spine.

(3) Finally I shall take up the surgery of the peripheral nerves.

SURGERY OF THE BRAIN.

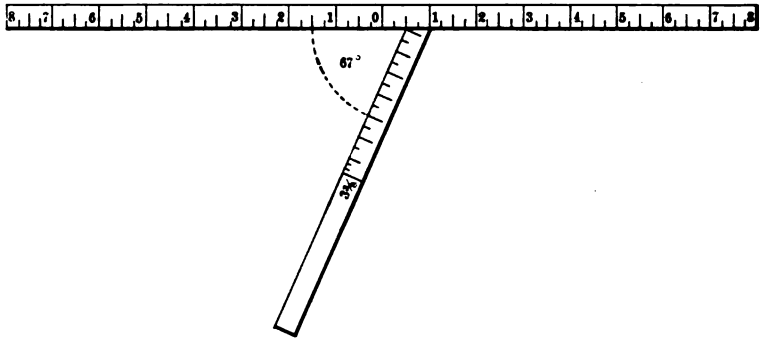
GENERAL TECHNIQUE OF SURGICAL OPERATIONS.

In spite of the rapid progress and brilliant achievements of modern antiseptic surgery, cerebral surgery made little advance until 1886. At that time Horsley, of London, and Macewen, of Glasgow, introduced radical changes, which have been but little altered, even by a large experience. These are in brief as follows:

Disinfection. With very few exceptions, the entire head should be shaved. The loss of a portion of the hair is so marked and so grotesque that it is better, even from an æsthetic point of view, that the whole head should be shaved, and, surgically, it is essential, in order to cleanse the entire scalp, so that we may avoid infection. Even persons who are most careful of the scalp cannot thoroughly cleanse it, on account of the presence of the hair. After the head has been shaved the patient should, of course, wear a cap or silk handkerchief, in order to avoid taking cold.

The fissures of the brain, especially the median fissure, fissure of Rolando, and in some cases the fissure of Sylvius, of Bichat, and others, should be marked on the shaven scalp with an aniline pencil. The median fissure lies about one-eighth of an inch to the right of the median line, since the left cerebral lobe is slightly larger than the right. The fissure of Rolando can best be marked by Horsley's first cyrtometer, as modified by Morris J. Lewis. This consists of an antero-posterior arm 14 inches long, and a lateral arm about 6 inches long (Fig. 308). The lateral arm is fixed at an angle of 67°, and starts from the antero-posterior arm at a point half an inch back of its middle. The zero point of the scale of the antero-posterior arm is at the middle, and the scale is graduated in inches forward and backward, The lateral arm marks the fissure of Rolando, which is about 3¼ inches long. In using the instrument the antero-posterior arm is placed one-eighth of an inch to the right of the median line, and in such a position that the inion and glabella will each correspond to the same figures on the scale. The zero point then marks the mid-point between the inion and the glabella, and the lateral arm starts half an inch back of this mid-point. This cyrtometer can be made out of an ordinary piece of stout paper or cardboard, if a metal one is not at hand. Buchanan, of Pittsburg, has constructed another form of cyrtometer, consisting of an aluminum triangle curved to fit the head, the angle being 67° (Fig. 309).

Fig. 308.

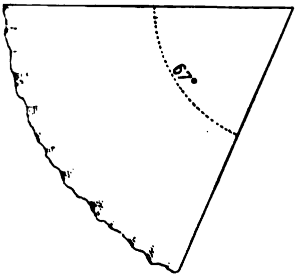


Horsley's cyrtometer.

Another simpler method which can be used in an emergency has been devised by Chiene, of Edinburgh. Fold a square piece of paper diagonally twice, starting from the same corner. This divides the apex into four angles of 22½° each (Fig. 310). Three of these angles give us 67½°, which is within half a degree of the direction of the fissure of Rolando. In order to fix the other fissures of the brain, the reader is referred to *Gray's Anatomy*, edition of 1887, p. 681, edited by the writer.

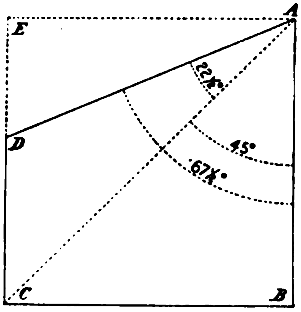
The day before the operation the head should be shaved anew if necessary, well scrubbed with soap and water, then with ether, then with a sublimate solution, not stronger than 1-2000, lest the scalp should be vesicated. A sublimate dressing, the three or four inner layers of which are wet with the solution, should then be applied and left in place until the operation, when the disinfection should be repeated. Of course, the ordinary precautions as to the thorough cleansing of the finger-nails, hands, and arms of the operator and his assistants, and of the instruments, dressings, etc., should be scrupulously carried out.

FIG. 309.



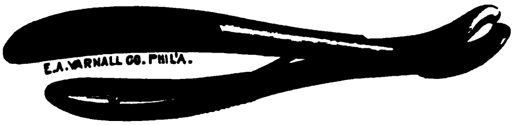
Buchanan's cyrtometer.

FIG. 310.



Chiene's method of fixing the fissure of Rolando.

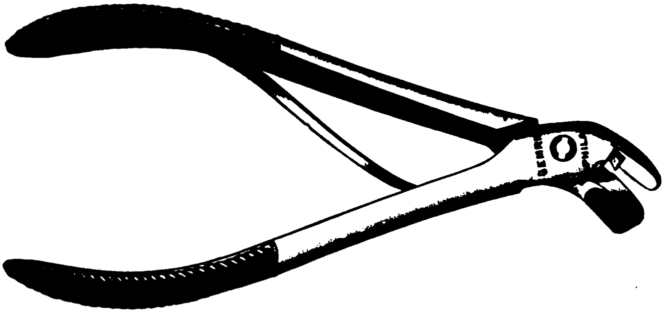
FIG. 311.



Lüer's double rongeur forceps.

POSITION. While I have often operated with the patient in the recumbent position, the best position is the semi-recumbent, in order to diminish hemorrhage. Practically this can be best affected by placing the patient on a lounge, with a sheet passed between the thighs and fastened around the head of the lounge to prevent his slipping down.

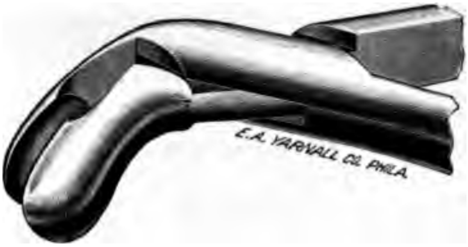
FIG. 312.



Keen's rongeur forceps.

MARKING THE BONE. Three points should be marked on the bone: the two ends of the fissure of Rolando and the point at which the centre-pin is to be placed, in case the trephine is used. The reason for marking the bone is that when the scalp has been raised, the position of the fissure of Rolando and the exact point for the application of the trephine are not easily redetermined. This marking of the bone can be done by a small gouge or by the centre-pin of another trephine, the rongeur forceps serving as a hammer.

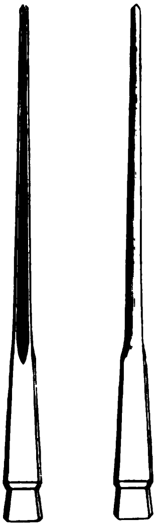
FIG. 313.



Hopkins's rongeur forceps, modified by Weir.

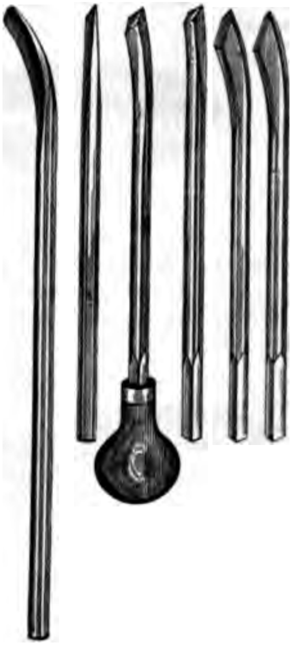
ACCESS TO THE BRAIN. Usually the ordinary operation of trephining suffices. In many cases in which we simply desire to get access to the skull cavity, as in linear craniotomy, fractures, etc., a half-inch or an inch trephine is large enough. When, however, we desire to expose any considerable portion of the brain, we should use either a 1½-inch trephine, or multiple small trephine openings may be made, the intervening bridges of bone being chiselled or sawn away or removed by various rongeur forceps (Figs. 311, 312, 313).

FIG. 314.



Hartley's chisels.

FIG. 315.

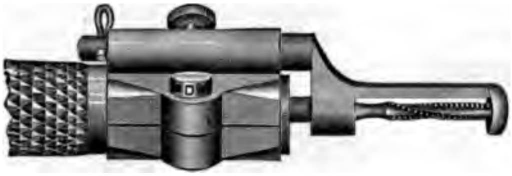


Pyle's chisels.

A different method, however, has recently gained considerable favor, from the large area of brain which it exposes, both for determining the fissures and convolutions and for operative procedures, and also for the re-establishment of the integrity of the skull. Wagner (*Centralbl. f. Chir.*, 1889, No. 47) introduced the method known as temporary osteoplastic resection. In this method a horseshoe shaped incision is made through the soft parts, directly down to the bone, the scalp not being separated from the skull. The external table of the skull is then chiselled through with the chisels employed by wood and ivory carvers, which can be had at any good hardware store, or, better, by Hartley's[1] or Pyle's[2] chisels (Figs. 314 and 315). The diploë having been reached, care should be taken, in going through the vitreous table, not to wound the dura. For this reason it should be divided with an osteotome, and not with the chisel. Instead of the chisel, we can use a rapidly rotating cylindrical drill, driven by the improved dental engine or an electric motor, or a suitably guarded circular saw. One or more elevators then being inserted under the edges of the bony flap, the portion of bone between the two ends of the incision is fractured, and the united flap of bone and scalp is turned downward, the scalp serving as a hinge (Fig. 318). This method of operation, however, will be very difficult in very thick skulls. When the operation is completed the bone is replaced by simply turning it back on its hinge, a portion of it being gnawed away by the rongeur forceps, if drainage is to be employed, and the flap

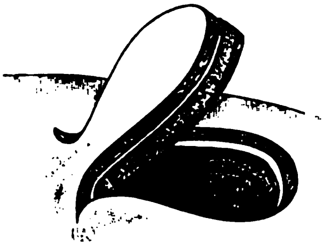
FIG. 316.

FIG. 317.



Cryer's drill.

FIG. 318.



Osteoplastic resection of skull. Wagner-Wolff's method (ESMARCH and KOWALSKY.)

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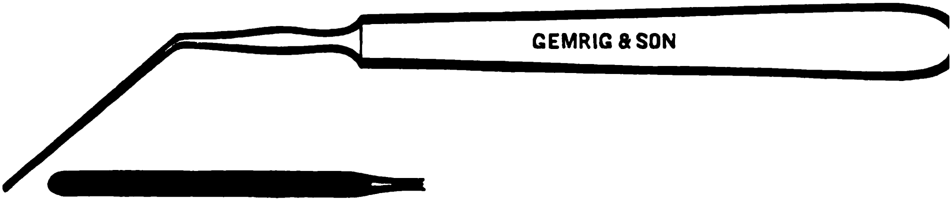
[1] Annals of Surgery, May, 1893.

[2] N. Y. Med. Record, Feb. 10, 1894.

held in place by ordinary sutures through the scalp. Care should be taken not to cut away the margins of the vitreous table projecting from the under surface at the margin of the opening in the skull, so that when the flap of bone is replaced these will serve as supports. Before turning the flap back into position the edges of the fracture should be freed from any splinters.

When the cranial cavity has been opened the dura can be separated from the under surface of the bone by Horsley's dural separator (Fig. 319), which will enable us to explore the inner surface of the skull for any irregularities. The little finger can also be readily introduced between the dura and the bone, and later, if need be, between the dura and the brain. As a rule, the dura should be opened, as the additional danger is but little and the additional information may be very great. The dura should be lifted by rat-tooth forceps and carefully incised by a knife, the rest of the incision being made with blunt-pointed scissors curved in the flat. The dura should be opened parallel to the opening in the skull, the margin being about a quarter of an inch from the margin of the opening on the bone, so as to allow room for subsequent suturing of the dura. Great care should be taken not to wound the underlying large veins of the brain. If we are dealing with a tumor or other lesion involving increased intra-cranial pressure it is often wiser to make one or two small incisions which can be enlarged or united if need be.

FIG. 319.



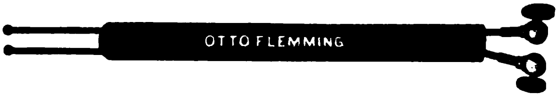
Horsley's dural separator.

Hemorrhage from branches of the middle meningeal can be arrested either by ligature at the point of rupture, or not uncommonly better, by passing a ligature through the dura and under the vessel by means of the finest semicircular Hagedorn needle. Hemorrhage from the large vessels, especially the veins of the brain itself, is one of the most troublesome difficulties in cerebral surgery. Some of the larger vessels can easily be ligated, but the veins are extremely fragile. Often they are best ligated by passing a small semicircular Hagedorn needle under them directly through the brain substance. The ligature should then be tied with great gentleness with equal traction on both ends of the ligature. The knot should not be tied too tightly lest it cut through. Hemorrhage can often be easily arrested by pressure, either by gauze or sponges, or by pressure combined with hot water (105° to 115° F.). In hemorrhage from the sinuses the lips of the wound can sometimes be seized by hemostatic forceps, which may be left in place for thirty-six to forty-eight hours, being of course entirely enclosed in the dressing in order to prevent infection. Plugging the sinus with iodoform gauze is, however, usually successful in controlling the bleeding. As the large cerebral veins approach the superior longitudinal sinus they suddenly broaden into large bays, called the para-sinoidal spaces. These should always be carefully avoided if possible. If they are wounded the hemorrhage can usually best be controlled by packing with iodoform gauze or by ligature.

CONDITION OF THE BRAIN. The first point to be observed is whether the brain bulges, and the amount of bulging, which is an indication of the degree of intra-cranial pressure, due to tumor, abscess, internal hydrocephalus, etc. If we have reason to suspect greatly increased intra-cranial pressure, the opening in the dura should *always* be small at first, lest the brain should protrude to such a degree as to make its replacement difficult, or not seldom even impossible. If desirable the opening can be enlarged later at will. The color of the brain may either be normal, or livid, yellow, or yellowish brown, indicating tumor, abscess, or old laceration. Œdema of the membranes is not uncommon, and is sometimes of such an extent as to obscure or even prevent any recognition of the sulci or convolutions. It may even be bloody. The membranes should then be nicked at several points, when the serum will escape, and the surface of the brain may then be recognized. Absence of pulsation of the brain is almost always observed in cases of large tumors, cysts, or abscesses. The consistency of the brain should be determined by touch.

RECOGNITION OF THE MOTOR CENTRES BY FARADIZATION. If it is desired to locate the motor centres, this can be done by an ordinary faradic battery. In this case no antiseptics should be used after the dura has been exposed, as they diminish the reaction of the cortex to electrical stimulation. Recognition of the motor centres is also possible through the unopened dura. The electrical current used in either case is generally one of sufficient strength to make the thumb muscles contract. The ends of the conducting cords may be used, or a double brain electrode of my own (see Fig. 320). The metallic points before being applied to the brain should be sterilized in carbolic acid, and the handle wrapped in antiseptic gauze. Before faradization the face and the four extremities should be uncovered, and one observer be requested to note the phenomena in each of these regions. The points stimulated by the battery should be exactly measured from the middle line, laterally, and antero-posteriorly from the fissure of Rolando. A stenographer who can rapidly record the observed phenomena as they are dictated is of great service.

FIG. 320.



Keen's double brain electrode.

REMOVAL OF PORTIONS OF THE BRAIN. If it is desirable to remove any motor centre or any abnormal portion of the brain, this should be done thoroughly; and it must be remembered that in doing this we can remove the brain substance much more freely antero-posteriorly than vertically, since in the latter direction we more quickly encroach upon adjacent centres. The area to be removed is to be determined by the battery.

DRAINAGE. Ordinarily in closing the wound, drainage may be dispensed with. In this case a little more space should be left between two of the sutures anteriorly and posteriorly, for the escape of wound fluids. If at the end of twenty-four hours there is any accumulation of such fluid under the scalp, a pair of hemostatic forceps or a probe may be inserted between these stitches and the redundant fluid pressed out. In operations for cysts drainage is often required. With abscesses, gunshot wounds, operations for hemorrhage, etc., it is a necessity. Rubber tubing is as a rule the best. If the bone is replaced a portion should be bitten away at the edge by the rongeur forceps to allow of the egress of the tube.

CLOSURE OF THE DURA. When the operation is completed the dura should be sutured, either by interrupted or continuous catgut sutures. It is important to direct the *point* of the needle toward the bone in order to allow plenty of room for manipulating the needle holder and the eye end of the needle.

CLOSURE OF THE OPENING IN THE SKULL. If the osteoplastic method of resecting the skull has been used, the method of closing the opening is that already described. (See page 961.) If a trephine button has been removed, however,and we intend to replace it, great care must be bestowed upon it. I usually place it in a teacup containing a 1-2000 bichloride solution. The teacup is placed in an ordinary basin containing hot water, the temperature of the water being determined by a thermometer. It should be from 100° to 105° Fahr. It is not necessary to cut the bone into small pieces, as Macewen first advised. I have often replaced a button an inch and a half in diameter, and have never seen it produce the slightest trouble. If the bone is diseased it should not be replaced, and the same rule should be followed in those cases in which we wish to alter or allow for increased intra-cranial pressure, as, for instance, in cases of headache or of irremovable tumors. If the bone is not replaced the opening becomes closed by a firm, fibrous membrane, giving efficient protection to the brain excepting from sharp, penetrating objects. The opening can be further protected by a piece of tin sewed inside of a skull-cap.

In some cases in which there has been a large loss of bony substance, it is desirable to close such bony openings by secondary operations. For this purpose the operation of Kœnig (*Centralbl. f. Chir.*, 1890, No. 27) is the best. The scar and fibrous tissue having been removed, a flap of adjacent scalp, of similar shape, but slightly larger, is cut down to the bone on three sides, but not separated from it. The fourth side serves as a pedicle. By a narrow chisel the outer table of the skull lying under the flap so outlined is then chiselled loose in small fragments which remain adherent to the under surface of the flap. This flap of scalp with its adhering bony fragments is now shifted so as to fill the bony defect, and is sutured in place. The raw surface of diploë from which the flap has been chiselled is then covered by Thiersch's method of skin-grafting. The rough under surface of transplanted bone becomes smooth and approximates the inner table in its character. When the dura mater has been removed such a flap has even been placed directly in contact with the brain without any ill results.

Fragments of bone have also been transplanted from the lower animals. Decalcified ox-bone has also been used, and Fraenkel (*Centralbl. f. Chir.*, 1890, 821) has proposed to insert a plate of polished celluloid, a procedure which has been followed with success in several instances. In all these cases drainage should be employed in order to provide escape for the underlying wound fluids.

COMPLETION OF THE OPERATION. The closure of the scalp wound should be made by interrupted sutures with silkworm-gut. The wound should then be covered with an ample antiseptic or sterilized dressing, covered by rubber dam and retained in place by a wet-gauze bandage, covered in turn by the ordinary muslin bandage, and, in the case of children or restless adults, by a night-cap. As soon as the dressing becomes moistened to its edges by serum or bloody discharge, the wound should be redressed, and attention be given to the evacuation of any retained wound fluids. If a drainage-tube has been used, it should be removed at the end of from twenty-four to forty-eight hours, except in cases of abscess or other similar conditions, when it must remain for some time. Usually by the fifth or sixth day one-half of the stitches may be removed, and the remainder on the seventh or eighth day.

Absolute quiet, both of body and mind, should be insisted on, especially for the first week, and not uncommonly longer. In cases of severe injury this may be wisely prolonged even for months.

SECONDARY OPERATIONS are occasionally required, in which the bone and the membranes must be dissected free from the brain with care, lest extensive injury be done to the cerebral centres. After primary removal of a motor centre, paralysis of the part supplied by it, and very frequently of neighboring parts, even amounting to a hemiplegia, may follow, but, as a rule, disappears to a great extent after some weeks. After secondary operations this paralysis or paresis is apt to be less pronounced, unless the brain has been extensively injured.

When the dura has been removed, and especially if this has been followed by any interference with the brain, there is always a marked tendency to fungus cerebri. In order to prevent this I suggested some years ago that the gap in the dura might be supplied by a portion of the pericranium from the under surface of the flap of scalp. The osteogenetic surface should be turned upward and the piece of pericranium sewed to the dura. I have done this successfully in three cases.

The limits of operative procedure are constantly being widened. By the dural separator, the probe, and the finger, a large part of the inner surface of the skull and of the cortex of the brain can be examined. Anteriorly, the brain can be lifted as far back as the anterior clinoid process. Laterally, the entire petrous bone can be uncovered, and posteriorly, both by the eye and the finger, we can reach the foramen magnum, both within and without the skull. The sinuses can be exposed and the brain may be punctured almost with impunity with a blunt instrument, such as the grooved director; and the lateral ventricles can be tapped.

EPILEPSY.

Epilepsy, in its various forms, has been discussed in Chapter XII.

SURGICAL TREATMENT OF EPILEPSY.

1. GENERAL EPILEPSY. For general, or so-called idiopathic epilepsy, no surgical treatment can be recommended.

2. FOCAL EPILEPSY. The surgical treatment of this form of epilepsy has consisted of the exposure of the centre in which the attack begins, its recognition by the faradic battery, and its excision. The results have not thus far been such as to give very great encouragement to the continuation of this treatment; but, on the other hand, sufficient time has not yet elapsed to determine definitely the results. The frequent improvement of epileptics after any operation, often far away from the head, makes us suspect that the temporary improvement often reported may be due to the operation, irrespective of its nature or site. Moreover, if the epilepsy has been caused by scar tissue the operation will be followed by the re-formation of a scar with its consequent irritation. It is possible, however, that the scar following an aseptic operation which results in immediate primary union is much less likely to leave an irritating cicatrix than the coarse, irregular cicatrix following an infected wound. In a certain number of cases there is, no doubt, that the excision of a traumatic scar in the brain has been followed by amelioration and even by cure, in spite of the secondary scar following the operation. The tendency, however, of surgeons I think is rather toward less than more

interference in focal epilepsy. The mortality is not very large, and, if further experience gives us fairly good results, it is a reasonable risk to take. At all events, it is unlikely that the patient would be made any worse.

In doing such an operation a large area of the brain should be exposed by an opening not less than an inch and a half in diameter, or better still, by a larger opening made by the Wagner-Wolff method. We can thus recognize the convolutions, and then by the battery, used in the manner already described, we can ascertain the exact location and limits of the cerebral centre which is sought. The entire centre, and even a little more, especially antero-posteriorly, should be removed by the methods already described. In doing this we can sometimes lift the pia and any large veins which run in the sulci, and, working under them, can remove all of the desired portion of the cortex without lesion of the vessels. All of the gray matter should be removed down to the white substance.

The surgical after-treatment is the same as that already described under the heading of Technique. In all cases medicinal after-treatment, especially by the bromides, and the dietetic treatment should be carefully carried out.

3. JACKSONIAN EPILEPSY. The treatment of this form is the same as for the last, but in both it should be remembered that it is important to excise the cerebral centres early, before the epileptic habit has been formed. The prognosis in Jacksonsian epilepsy is better than that of focal epilepsy, especially in traumatic cases. The published results have been more favorable both as to betterment and cure.

4. TRAUMATIC EPILEPSY. The site of the operation, if operation is decided upon, should be determined rather by the localizing symptoms than by the external scar. Moreover, if the scar is tender, and especially if pressure upon it produce an attack, the scar itself should be excised before any operation is done upon the brain. If this minor operation does not cure, then the brain may be attacked by the usual methods. The results in some excessively severe cases have been excellent. Thus one of Mr. Horsley's cases (*Brit. Med. Journ.*, 1887, I. 864), following an old depressed fracture of the skull, had 2870 convulsions in thirteen days, and another, related by Miles (*Lancet*, 1891, II. 1159), following a blow on the top of the head, had 3597 fits in forty-two days, and as many as 219 in a single day, and both cases were cured by operation, the cure being persistent after a long interval of time. Sometimes, even without any apparent lesion of the brain, as in the case of Mr. Miles, the trephining may result in cure.

In all these cases of epilepsy it is desirable to open the dura so as to inspect the condition of the brain. If a scar exists in the dura it should be excised and the gap filled by the pericanium. If one exists in the brain it should be removed down to the white matter, or as deeply as the scar goes. If the bone is diseased it should not be replaced, and, in fact, in all forms of epilepsy it is better not to replace the bone, at least if removed by the trephine. Even in the Wagner-Wolff method, if the bone is found to be diseased it should be removed. Sometimes a cyst will be found, in which case it should be excised. If adhesions have formed, Beach (*Boston Med. and Surg. Journ.*, April 3, 1890) proposes to insert a piece of aseptic gold foil between the brain and the dura, a procedure successfully accomplished by Park. If there has been a primary trephining at the time of the fracture of the skull, followed by thickening of the edges of the opening, and also by the formation of scar tissue, I have found in some cases that the simple removal of this thickened bone has been followed by benefit, amounting sometimes almost to cure. Possibly the gold foil might be of use here.

ARRESTED DEVELOPMENT AND MALFORMATIONS, INCLUDING PORENCEPHALUS AND MICROCEPHALUS.

The only successful operation that I have seen reported for a case of arrested development is that of Felkin and Hare (*Manchester Med. Chron.*, October, 1891, p. 17). The patient was a girl of seventeen years, who at the age of ten months had had her skull fractured. This was followed by paralysis and imperfect development of the right arm and leg. An extra-dural cyst two inches in depth and an osteophyte half an inch long were found. Marked improvement followed the operation.

In the congenital cerebral palsies the general conclusion is against operation rather than in favor of it; but occasionally, especially in those few cases in which hemorrhage is the cause of the palsy, an immediate operation might be of value.

In porencephalus and athetosis it is doubtful whether any operation should be done, if the porencephalic condition can be diagnosticated in advance. Oppenheim (*Deutsch. Med. Woch.*, July 3, 1890, 595), however, has reported a case of athetosis accompanied with cerebral palsy of childhood and epilepsy, in which a cyst was found, and both the athetosis, the contracture, and the epilepsy were improved. Kocher (*Deutsch. Zeit. f. Chur.*, 1893, vol. xxxvi, 72) has reported two cases of recovery after operation for porencephalus. The lateral ventricle was opened and the choroid plexus and the corpus striatum were seen.

Surgical Treatment of Microcephalus. For the purpose of determining whether the size of the head is abnormally small or large, the following table from Finlayson (*Keating's Encycl. Dis. of Children*, vol. i. p. 94, footnote) is of value:

No of cases.	Age.	Head, inches.	Chest, inches.	Difference between head and chest.
100	1 day	13.75	12.94	Head more than chest, 0.81
66	6 to 12 weeks	15.25	14.25	" " " " 1.00
75	6 " 8 months	16.68	15.58	" " " " 1.10
71	11 " 13 "	17.80	17.20	" " " " 0.60
67	21 " 24 "	18.38	17.85	" " " " 0.53
50	34 " 36 "	18.70	18.61	" " " " 0.09
60	4 " 4½ years	19.20	19.72	Chest more than head, 0.50
46	6 " 6½ "	19.51	20.76	" " " " 1.25
40	9 " 10 "	19.56	21.31	" " " " 1.75
31	11 " 12 "	20.00	23.46	" " " " 3.46

In microcephalus, Lannelongue (*l' Union Méd.*, July 8, 1890), first proposed to exsect a strip of bone in the skull about a quarter of an inch wide. This excision may be made on one or both sides of the sagittal suture and parallel to it, extending from the forehead to the occiput. To this antero-posterior groove may be added a lateral branch on each side. If the faulty development preponderates in as particular region as in the frontal, a transverse groove may be made. Soon after Lannelongue's article appeared reporting three cases, I reported a fourth (*Med. News*, Nov. 29, 1890), and since then a large number of cases have been recorded. I have myself done a dozen operations. The result in general may be stated as follows: The mortality is large, averaging from 20 to 25 per cent., as would be expected in children of such faulty development. The loss of blood is, as a rule, not such as to imperil life, but the shock attending not only the moderate loss of blood, but the wide separation of the scalp, which is inevitable, and the injury to the bone, is very great. For this reason it is therefore never desirable to operate on both sides at once, but at an interval of a few weeks. Moreover it is best not to subject children under, say nine or ten months of age, to the shock of such an operation, nor, perhaps, is it desirable to operate after nine years of age.

The result as to the mental development varies greatly. The majority of the children who recover are not improved. A moderate number show some improvement; in a few it has been very great. Hence, in such a distressing and otherwise irremediable disease I think that operation is at least permissible, but I should never be disposed to urge it against even the slighest hesitation of parents. Certain it is that the head, which may not have grown at all prior to operation, may after operation become noticeably larger.

After the usual preparation of the scalp, the incision should be made in the direction chosen, directly down to the bone. If the incision is anteroposterior, by making a lateral incision just behind the anterior border of the hair, the scalp from the forehead may be drawn well down, so that the bone operated on may be reached half-way down from the hair to the eyebrows. This position of the incisions enables them to be hidden by the hair. Very frequently the hemorrhage will be so slight that no forceps will be required, but should there be any marked bleeding the vessels should be immediately seized by the hemostatic forceps. Make a half-inch trephine opening far enough from the median line to avoid the superior longitudinal sinus, and with a pair of rongeur forceps which I devised (Fig. 307) the operation can be completed in from twenty to thirty minutes. Only occasionally will any bloodvessels have to be ligated. The sutures in the wound are usually sufficient to control the bleeding. Before replacing the flap it is well to remove the pericranium corresponding to the groove in order to prevent the reproduction of the bone. The edges of the wound are then united by sutures, and the ordinary care of such a wound is carried out.

HYDROCEPHALUS.

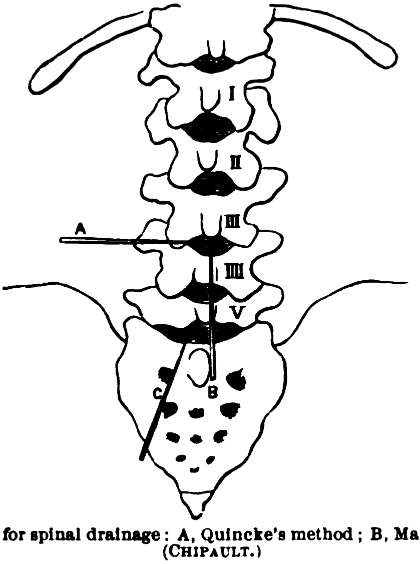
The great majority of cases operated on for hydrocephalus have died. However, Broca (*Rev. de Chir.*, 1891, 37), Phocas (*Rev. Mens. des Malad. de l'Enfance*, February, 1892), Mayo Robson (*Brit. Med. Journ.*, December 6, 1890), Hahn (*Med. News*, May 6, 1893, 500), Soderbaum (*Annals of Surgery*, 1892, xv., 467) and McCosh (*New York Med. Record*, September 16, 1893, 376, and *Amer. Journ. of Med. Sci.*, March, 1894, 239), have all reported cases of acute or chronic hydrocephalus in which puncture of the lateral venticles has done great good. The methods by which the increased intra-cranial pressure has been relieved have been first, by puncture of the lateral ventricles; second, by puncture of the membranes of the cord in the lumbar region; and third, the puncture of the fourth ventricle.

1. Puncture of the Lateral Ventricles. In the *Medical News* for December 1, 1888, I published a paper, read November 7, 1888, before the College of Physicians of Philadelphia, in which was first distinctly formulated the technique of puncture of the lateral ventricles. I showed that the lateral route is decidedly the best, as it avoids the important brain centres, and the ventricles can with ease and accuracy be reached. A half-inch trephine opening is made an inch and a quarter behind, and the same distance above the meatus. The brain is then punctured by a tube, about No. 5, of the French catheter scale, or by a grooved director or other suitable instrument, directed toward a point two and one-half inches vertically above the opposite external auditory meatus. At a depth of one three-quarter or one one-half

inches or less, according to the distention of the ventricle, its cavity will be reached. The moment the ventricle is reached the diminution of pressure is readily perceived by delicate fingers, and the immediate escape of the cerebrospinal fluid assures us that the instrument is in the ventricle. The ventricle can of course be reached, especially if distended, from almost any part of the cerebral surface. I have, also, in a case which I trephined and punctured upon both sides, washed out the lateral ventricles from side to side with a boric acid solution with subseqnent comfort to the patient. Should it be desired to keep up continuous drainage, this can be effected either by a rubber tube, or by horse-hairs doubled and introduced with the curved end first. In the *Medical News* of September 20, 1890, will be found the *résumé* of a full paper relating to this method of puncture.

2. PUNCTURE OF THE MEMBRANES OF THE CORD IN THE LUMBAR REGION. Wynter (*Lancet*, 1891, 1, 931), proposed to substitute for puncture of the lateral ventricles, puncture of the membranes of the cord in the lumbar region, and Quincke (*Verhandl. des 12ten Kongress f. Innere Med.*, 1893, 197), has reported 41 operations. Quincke has proposed to puncture in the third or fourth intervertebral space, which measures 18 to 20 millimetres transversely and 10 vertically. This is below the termination of the cord, and there is an interspace of 5 mm. between the two bundles of the cauda equina. The depth of the puncture to reach the cord in infants is about 2 centimetres, and in adults 4 to 6 centimetres. Chipault has proposed to puncture between the last lumbar and the first sacral vertebræ by preference, the procedure in other respects being the same.

FIG. 321.



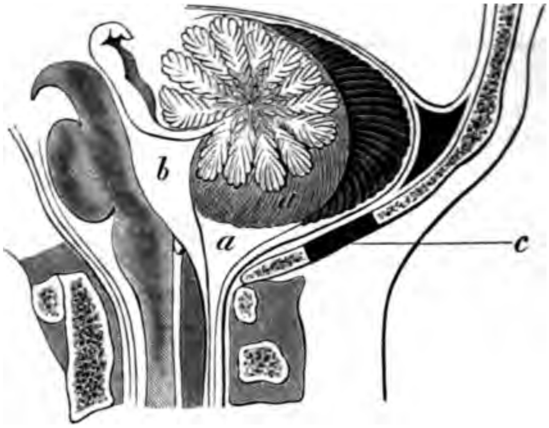
Method of puncture for spinal drainage: A, Quincke's method; B, Marfan's; C, Chipaut's. (CHIPAULT.)

Fürbringer (*Lancet*, 1895, 1, 1022), has reported more than 100 punctures, in 86 patients, without any ill effect. The bacillus tuberculosis was found in the fluid in thirty of thirty-seven cases of tubercular meningitis, in which the diagnosis was doubtful. Four cases of cerebral tumor were relieved by the operation.

3. PUNCTURE OF THE SUBARACHNOID SPACE. Parkin (*Lancet*, 1893, ii. 21), has proposed and operated by the following method: He trephined three-

quarters of an inch below the superior curved line of the occipital bone, just to the right of the middle line, and enlarged the opening downward by the rongeur forceps. After opening the dura he inserted a probe into the fourth ventricle, lifting the cerebellum to a slight extent, when 2 to 3 ounces of fluid gushed out. Great care should be taken not to wound the floor of the fourth ventricle. He reports four cases with two recoveries, and Ord and Waterhouse (*Lancet*, 1894, ii. 873), have reported a successful operation in a case of tubercular meningitis.

FIG. 322.



Vertical section of base of skull immediately to the right of the median line; *a*, basal subarachnoid cavity and its relation to the cerebellum; *b*, fourth ventricle; *c*, site of trephine aperture. (PARKIN.)

MENINGITIS.

Mr. Barker (*Brit. Med. Journ.*, 1888, i. 777) has reported a noteworthy case in which he evacuated an ounce of odorless pus from the fissure of Sylvius, with a successful result. Sawtelle (*Occid. Med. Times*, Feb., 1892, 76) also evacuated five drachms of fluid, followed by recovery, and Mr. Parkin (*Lancet*, July 1, 1893, 21) has reported a similar case, both of these cases being traumatic. It is rare, perhaps, that we shall be as successful in non-traumatic meningitis as Mr. Barker was, but occasionally it may be done. The technique is simply that of trephining and opening the dura, followed by puncture toward the source of the pus or serum, as in Mr. Barker's case, should it be necessary. Fortunately, often simple trephining will answer.[1]

Similarly in chronic ossifying pachymeningitis, sometimes the remote result of injury which causes thickening and induration of the bones of the skull and severe and persistent headaches, simple trephining will not seldom give relief. The bone should not be replaced in these cases.

In persistent meningitis the only possible relief is found in speedy trephining and drainage, without replacement of the bone. The chances of death are, of course, much greater than those of life, but the patient should at least have the benefit of this small chance.

For the methods of operating in both these cases see the section on Technique.

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[1] See other cases by McArdle, Dublin Journ. of the Med. Sci., 1892, xciv. 17; and Tobin Brit. Med. Journ., 1892, ii. 21.

PACHYMENINGITIS HEMORRHAGICA.

In pachymeningitis hemorrhagica, Dennis, (*N. Y. Med. Journ.*, Dec. 24, 1892, 701) has urged that operation should be undertaken, *e. g.*, in cases of alcoholism followed by such hemorrhage. Ceci (*Chir. Behandl. v. Hirnkr. Bergmann*, p. 112) has performed such an operation in a case following accident. Buchanan (*Pittsburg Med. Rev.*, Sept., 1894), and Harris (*Brit. Med. Journ.*, 1892, i. 503) have reported two successful cases, and Stewart (*Brit. Med. Journ.*, 1887, i. 877) a fatal case. When the headache has been followed by paralysis, and the location of the lesion is clearly indicated, when the pupils, which were contracted and immobile before unconsciousness supervened, have become dilated, when optic neuritis, and finally coma, have set in, it would seem proper to operate without further delay. This might be either by the ordinary trephining or by an osteoplastic resection, the dura being opened, the clot removed, and if need be, drainage established.

ABSCESS OF THE BRAIN.

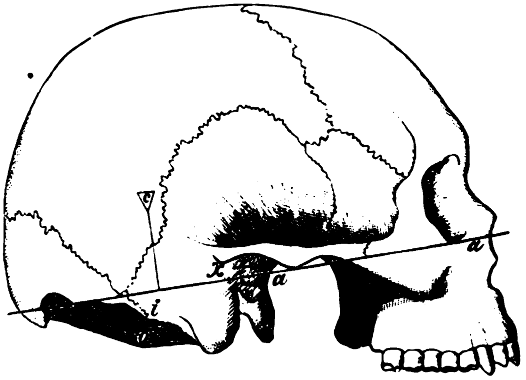
TRAUMATIC CEREBRAL ABSCESS. In operating for abscess of the cerebrum, the spot selected for the operation should be determined by the localizing cerebral symptoms, and not by the scar, should such be present, or by the history. If, however, the abscess, as is not infrequently the case, is situated under the site of the injury, the operation should, of course, be at this place. The skull may be opened by a half-inch trephine, and the dura opened by a small incision. If an abscess exists, the brain will not pulsate and will bulge into the opening from the increased pressure. The brain is then to be punctured. The best instrument for this purpose is the grooved director, since it allows the exit of the pus in its groove, and, being blunt-pointed, will not injure any vessels. Should the first puncture not reveal the abscess, the instrument should be withdrawn exactly in the line in which it has been introduced, so as not to wound the brain any more than is possible, and be reintroduced in the next most likely direction in the same manner. A third, and if necessary a fourth and a fifth, puncture may be made. The injury from the punctures is but slight, if they are made with the proper precautions. The fatality of the disease warrants any reasonable interference.

When the abscess has been found a pair of hemostatic forceps should be introduced with the blades closed, the blades then opened half an inch or an inch, and so withdrawn in order to afford free exit for the pus. The wound may then be washed out very gently with a boric-acid solution, four or five grains to the ounce, and if deemed best, the cavity of the abscess may be gently curetted. A drainage-tube should then be introduced, a suitable opening bitten away for it, if the bone is replaced, and a corresponding opening made in the scalp, the drainage-tube being secured to the latter by a stitch. The abscess cavity may be washed out daily, but with great gentleness, and the drainage-tube gradually shortened as the amount of discharge diminishes, and finally be removed. Should reaccumulation take place, the cavity should be reopened through the original opening in the skull. Care should be taken not to make the opening in the skull any larger than is necessary, as a fungus cerebri is almost certain to follow if a large opening exists. For the same reason, if there is necrosed bone, but little of it should be removed. When later the bone has loosened it should be removed by a secondary operation. Should the abscess not be found at the site of the first operation, a second, and, if need be, a third trephine opening may be made at the next most probable points. On no account should the abscess, if it exists, be left undiscovered.

OTITIC ABSCESS. The most common cause of abscess in the cerebrum is, however, not traumatism, but chronic suppurative otitis media. (See page 374). In these cases the abscess may be (1) extra-dural, that is, between the dura and the petrous bone; (2), sub-dural, that is, between the dura and the brain, or (3) in the substance of the cerebrum, separated from the surface, it may be, by an inch or more of apparently healthy brain tissue. Almost always the mastoid will already have been opened, but should this not have been done, it should be the first step in the operation.

MASTOID ABSCESS. *Operation.* The external meatus should first be thoroughly cleansed antiseptically. A vertical curved incision is then made in the axis of the mastoid from its base to its tip, the ear being drawn well forward. The vessels are seized with hemostatic forceps, the bone laid bare, and the mastoid antrum first opened at a point one-third to one-half an inch behind, and the same distance above, the centre of the meatus. (Fig. 323).

FIG. 323.



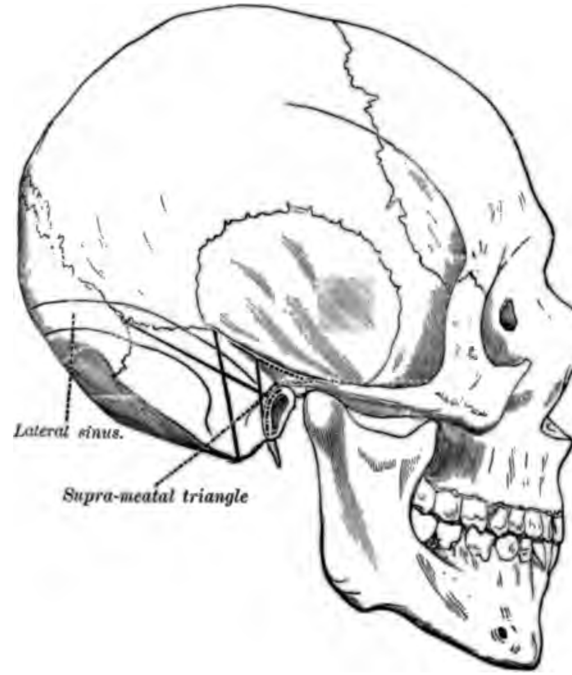
a, a. Reid's base line from the lower border of the orbit through the meatus: *c*, the point for trephining to reach an abscess in the temporo-sphenoidal lobe or to puncture the lateral ventricle 1¼ inches behind the meatus, and 1¼ inches above Reid's base line; *i*, the mastoid vein; *o*, the point for trephining to reach an abscess in the cerebellum: *x*, the point for trephining the mastoid antrum. (BARKER.)

This opening is made towards the base of Macewen's supra-meatal triangle. (Fig. 324). This triangle is bounded above by the root of the zygoma, anteriorly by the posterior margin of the bony meatus, and posteriorly by a line joining these two. An opening here is free from danger of wounding the sigmoid portion of the lateral sinus, which always lies behind the triangle.

The mastoid antrum and cells should then be thoroughly laid open by the gouge and chisel, and the posterior wall of the meatus chiselled away to the cavity of the middle ear. Twitching of the face on the same side should be watched for, as it would indicate proximity to the facial nerve, and no more chiselling should be done in that direction. All the inspissated pus found in this bony cavity should be thoroughly removed by a small sharp spoon or gouge, care being taken not to wound the lateral sinus. The wound should then be lightly packed with iodoform gauze daily.

TEMPORO-SPHENOIDAL ABSCESS. The mastoid operation may be sufficient to relieve the patient, but if not the abscess should then be sought in the temporo-sphenoidal lobe, unless there is reason to believe that it is situated in the cerebellum (*vide infra*, also page 377). The axis of the temporo-sphenoidal lobe I have shown to be a line drawn from a point 1¼ inches above and behind the external meatus to the opposite wing of the nose. A half-inch trephine should be applied as indicated by Barker, 1¼ inches above the external auditory meatus, and 1¼ inches above Reid's base line (Fig. 323 *c*). The dura is then opened by a small cruciform incision, and a grooved director inserted in the axis of the temporo-sphenoidal lobe, as above indicated, to the depth of 2 or 2½ inches, unless the abscess has already been reached. Once that abscess has been discovered, it should be treated as above indicated. If the first puncture does not reveal the abscess, it may be needful to make repeated punctures.

FIG. 324.



Surface guides for the sigmoid sinus and the supra-meatal triangle. (MACEWEN.)

Three artificial lines drawn upon the skull indicate the following: 1. The short vertical line from the posterior border of the external auditory meatus to the posterior root of the zygoma marks the base of the supra-meatal triangle. The broken line indicates the anterior border of the supra-meatal triangle, its base being the dotted line marking the part of the root of the zygoma. This broken line also indicates the course of the facial nerve. 2. The second vertical line, extending from the parieto-squamo-mastoid junction to tip of mastoid; the upper two-thirds of its length indicate the position of the sigmoid sinus. 3. The oblique line, passing from the asterion to upper limit of the external auditory meatus, indicates in its posterior two-thirds the sigmoid sinus from its commencement to its knee.

EXTRA-DURAL ABSCESS. This is caused not uncommonly by caries of the petrous bone from disease of the ear. The temperature in this form of abscess is usually 102° to 104°, rather than normal or subnormal, as in ordinary cerebral or cerebellar abscess. The mastoid should be opened as already described, unless this has already been done. The skull should then be opened an inch directly above the meatus, unless a fistula through the bone indicates any other point of preference. The dura should then be separated from the bone, but not opened, and the abscess cavity irrigated with an antiseptic solution and curetted. Carious or necrosed bone should be removed and free drainage provided. Bircher (*Centralbl. f. Chir.*, 1893, 482) chiselled away all the necrosed bone, except the carotid canal.

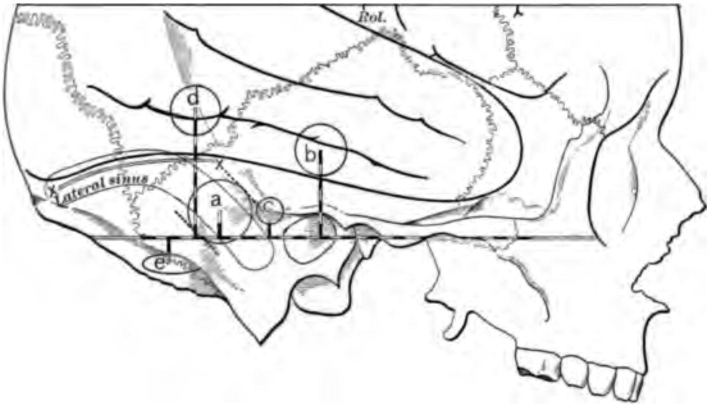
CEREBELLAR ABSCESS. An abscess of the cerebellum is best reached at a point midway between the mastoid and the inion, and sufficiently far below the line from the mastoid to the inion, which corresponds to the lateral sinus, to avoid wounding the latter (Fig. 323 *o*). The occipital bone is bared by a semilunar incision, with the convexity upward, and the bone penetrated either by a trephine or by the chisel, or in children simply by the gouge. The opening may then be enlarged at will by means of the rongeur forceps. The dura is then opened to a small extent. As in the cerebrum, so in the cerebellum, the brain will bulge, and will not pulsate if an abscess is present. A grooved director should then be inserted in the axis of the lobe of the cerebellum. The opposite lobe can be reached by an oblique puncture, care being taken not to injure the superior vermiform process.

THROMBOSIS OF THE LATERAL SINUS AND THE INTERNAL JUGULAR VEIN.

Zaufal, in 1880, first carried out the proper treatment for this disease (*Prager Med. Woch.*, 1880, p. 576). In 1886 Horsley again called attention to the method, and Mr. Arbuthnot Lane (*British Med. Journ.*, 1889, i. 997) reported the second case soon afterward, with a favorable result. From that date it has assumed its proper place as the rational treatment of this otherwise almost uniformly fatal disorder. I have records of 84 cases which have been operated upon, with 56 recoveries, a mortality of only 33.3 per cent.

The operation consists not only in the exposure, but also the opening, of the sinus and the proper cleansing of its cavity, and also, if the internal jugular vein is involved, the similar treatment of this vein. If the mastoid antrum and cells have not already been opened and cleansed, this should be done at once. Next the sinus should be exposed by the chisel or trephine at a point half an inch behind and one-quarter of an inch above the middle of the external auditory meatus (Fig. 325 *c*). The opening can be enlarged either by the rongeur forceps or the chisel and gouge. Pus will very likely be found in the groove for the sinus. Should the sinus be thrombosed, it will be harder than normal to the touch. If no thrombus has formed, it will be soft. In either case, should the symptoms warrant it, it should be opened. If the blood flows freely it can be arrested, first by the finger and then with strips of iodoform gauze, with which the sinus should be plugged. These strips should be made ready before the operation is begun. If a clot exists it will probably be very foul, and should be evacuated by the small spoon or curette and the sinus then disinfected. Cleansing and disinfection may have to go as far as the torcular Herophili. The sinus should be completely cleaned out, until free hemorrhage shows that all the clot has been removed. It should then be packed, in order to arrest the hemorrhage. The internal jugular vein should next be exposed in the neck and ligated at a point *below* the thrombus, the extent of which can be determined by touch. This step is of very great importance, as, if done sufficiently early and low enough down, it will prevent the extension of the infection to the lungs. The vein

FIG. 325.



Lateral aspect of a small adult skull. (BALLANCE.)

The illustration shows the relations of the lateral sinus to the outer wall of the cranial cavity and the position of the trephine opening (a), which should be made when it is deemed necessary to expose it. The base line (Reid's) passes through the middle of the external auditory meatus and touches the lower margin of the orbit: it is marked out in eighths of an inch, as are also the perpendicular lines drawn from it. The measurements are made along the base line from the middle of the bony meatus. The drawing also shows the convolutions of the temporo-sphenoidal lobe, the Sylvian fissure, and the position of the lower end of the furrow of Rolando (*Rol.*) x x indicates the site of the tentorium as far as it is in relation to the external boundary of the skull. The anterior x shows the point where the tentorium leaves the side of the skull and is attached to the superior border of the petrous bone. a, trephine opening to expose sinus, five-eighths of an inch in diameter, its centre being one inch behind and a quarter of an inch above the middle of the bony meatus. This opening can easily be enlarged upward and backward and downward and forward (see the dotted lines) by suitable angular cutting bone forceps. It is always well to extend it forward, so as to open up the mastoid antrum (c) and the gutter of the carious bone (if there be one), which leads from the antrum, tympanum, or meatus, down to the bony groove. The position of the trephine openings, which must be made for the relief of inflammatory intra-cranial affections, secondary to disease of the ear, other than for sinus pyæmia, have been added to the drawing for the sake of contrast and completeness. They are as follows: b, trephine opening to explore the anterior surface of the petrous bone, the roof of the tympanum, and the petro-squamous fissure, half an inch in diameter, its centre being situated a short inch (seven-eighths of an inch) vertically above the middle of the meatus. At the lower margin of this trephine hole a probe can be insinuated between the dura and bone and made to search the whole of the anterior surface of the petrous. c, trephine opening for exposing the mastoid antrum, a quarter of an inch in diameter, and a half an inch behind and a quarter of an inch above the centre of the meatus; or a quarter of an inch above the centre of the meatus and a quarter of an inch behind its posterior border. The trephine should be directed inward and slightly downward and forward. When a superficial disk of bone has been removed it is well to repeat the operation with the gouge. A larger trephine may with advantage be emplpyed, especially in adults. d, trephine opening for temporo-sphenoidal abscess, half an inch in diameter. Situation recommended by Barker, one inch and a quarter behind and one inch and a quarter above centre of meatus. The needle of the aspirator is to be directed at first inward and a little downward and forward. Birmingham prefers one and three-fourths of an inch above, in order to avoid the lateral sinus. e, trephine opening for cerebellar abscess, half an inch in diameter and one inch and a half behind, and a quarter of an inch below the centre of the meatus. Birmingham prefers two inches behind and one inch below to avoid the occipital artery. The anterior border of the trephine should just be under cover of the posterior border of the mastoid process. The drawing shows that a trephine hole made in this situation is far away from the lateral sinus, and that the trocar and canula of the aspirator if directed forward, inward, and upward, would hit an abscess occupying the anterior part of the lateral lobe of the cerebellum, which is the usual site of collections of pus in this part of the brain.—From *Brain Surgery*, by Starr, p. 193.

should then be cut above the ligature, the upper end attached to the skin, the vein and the sinus being washed out antiseptically and as thoroughly as possible.

TUMORS OF THE BRAIN, INCLUDING CYSTS.

The tumor having been located and its size and character approximately determined, the first step in the operation, after the preliminary cleansing, as described in the section on Technique, is to mark the median line and the fissure of Rolando on the shaven surface. The upper and lower ends of the fissure of Rolando should be marked by puncturing the bone by means of a gimlet, small gouge, or the centre-pin of another trephine. Should it be determined to open the skull by the trephine, the centre of the probable location of the tumor should be similarly marked. The bone is then exposed by a large semilunar or horseshoe flap, with the position of the tumor as its centre. A large opening should then be made in the skull, from an inch and a half to three inches in diameter, either by a single or by multiple trephine openings, or by a chisel. Instead of this, however, the skull may be opened by an osteoplastic resection (see p. 961). If a tumor is present, the dura will not pulsate and will bulge to a greater or less extent, according to the size of the tumor. The dura should then be opened about a quarter of an inch from the margin of the opening in the bone. This opening should be small at first, until the size of the tumor can be gauged to some extent by the amount of the bulging. Should exploration through this small opening indicate that the tumor can probably be removed, the opening in the dura may then be enlarged to such an extent as is necessary. The tumor having been found, if it extend in any one direction beyond the limits of the opening in the bone, this may be enlarged at will by the rongeur.

The tumor may be occasionally enucleated by the finger. If this is not feasible, the knife and scissors, sharp spoon, or the handle of an ordinary teaspoon may be used to remove it, either whole or piecemeal. Should it be an infiltrating tumor, and therefore probably malignant, unless inoperable, not only the tumor but a considerable amount of apparently healthy brain-tissue should be removed beyond its margins, so as to be certain that all the diseased tissue has been removed.

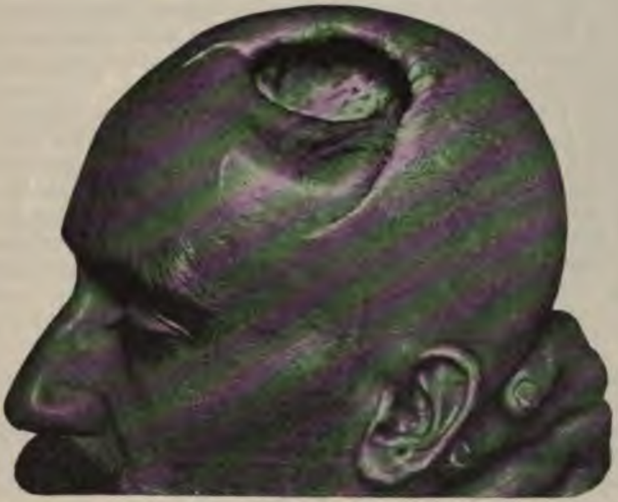
If subcortical tumor be suspected, an incision is made in the brain, and the tumor sought for by the little finger, inserted with great gentleness, or by a probe or grooved director, in order to recognize its size, depth, density, etc. If it be so large that it is not wise to attempt its removal the operation should immediately be terminated, the dura sutured and the flap of scalp replaced, the bone being left out permanently. In case of a large tumor, sometimes only a portion has been removed, with great benefit. It may be attacked a second or a third time in case of need. I must especially draw attention, however, to the importance, in case the tumor is very large, of not doing too much, since I am sure that death has followed in some cases by reason of too extensive interference.

Hemorrhage is to be treated as has already been described under the head of Technique. The closure of the wound and after-treatment are described in the same section.

Should the presumed tumor prove to be a case of actinomycosis the diseased tissue should be removed if possible. Buzzi and Galli-Valerio (*Brit. Med. Journ.*, 1893, ii. epitome 23), refer to a case by Van Itersen, and record another of their own in which, after the failure of operation, gr. xxx. of iodide of potassium daily effected a complete cure. Should the case be suspected to be one of actinomycosis, the iodide treatment should of course be tried before operation is resorted to, especially as the improvement in reported cases has begun quickly and progressed rapidly.

In many cases of tumor it is best to do the operation in two stages several days apart. The first stage consists of opening the skull and dura, determining the site, character, and size of the tumor, and then temporarily closing the wound. If operable the removal may be undertaken from three to six days later by reopening the wound.

FIG. 326.



Result seven years after the removal of a large fibroma of the brain. (KEEN.)

Fig. 326 shows the result seven years after the removal of a cerebral fibroma weighing over three ounces, which I reported in the *American Journal of the Medical Sciences*, in October, 1888. The operation was followed by a large fungus cerebri. The opening of the skull is protected by a piece of tin covered with silk and sewed inside a skull cap.

PERSISTENT HEADACHE.

In a few cases of persistent and severe headache, unconquerable by medical means, the patient has been trephined with good results, by Horsley, Warren, Weir, myself, and others. The bone disk should not be replaced, inasmuch as change or relief of the intra-cranial pressure is sought.

The intense headache which accompanies tumor of the brain has been most happily relieved in a number of cases by trephining, even when the tumor itself has not been removed. Thus Horsley (*Brit. Med. Journ.*, December 6, 1890, has reported a case in which he removed nearly one-half the occipital bone for an inoperable intra-cranial tumor. The relief was so great that, when from the growth of the tumor the headache returned, the patient sought relief by the removal of the other half of the same bone. The same relief has followed in two of my own cases of irremovable tumor.

TREPHINING FOR PSYCHOSES.

These cases may be divided into (1) cases of traumatic insanity, (2) non-traumatic insanity, and (3) general paralysis of the insane.

In Traumatic Insanity there will usually be a scar to guide us to the proper site of the operation. The trephining should be done in accordance with the rules already laid down under the head of Technique. Any spiculæ of bone, cyst of the brain, lacerated dura, or injured brain-tissue or brain-scar should be entirely removed.

Non-traumatic Insanity. Burkhardt (*Allgem. Zeitsch f. Psychiat.*, etc., 1891, 463), has reported 6 cases in which he exposed the brain and removed strips of its tissue 2 centimetres in width. In some cases he did multiple operations, the operations lasting even over four hours. It is doubtful, however, whether such operations are justifiable.

General Paralysis of the Insane. Mr. Claye Shaw (*Brit. Med. Journ.*, 1889, ii. 1090), reported the first case of trephining for this condition. He removed a piece of bone one one-half by three-quarter inches in size, and evacuated considerable fluid. The operation has been repeatedly done, but, as a rule, without any improvement. To be of any value the operation should be done early. The exposure of the brain, opening of the dura and closure of the wound after replacement of the bone have already been described. (See Technique.) See also page 698.

CEREBRAL PALSIES.

Occasionally, but very rarely, indications for surgical interference are presented by these palsies. Operation should be limited to those apparently caused by hemorrhage, cyst, or tumors. The technique for exposing the brain has already been described. The clot, cyst, or tumor should then be removed and the wound closed as usual. If the brain is simply atrophied, as shown by a marked difference in the size of the two sides of the skull, no operation should be done.

REMOVAL OF THE GASSERIAN GANGLION FOR TRIGEMINAL NEURALGIA.

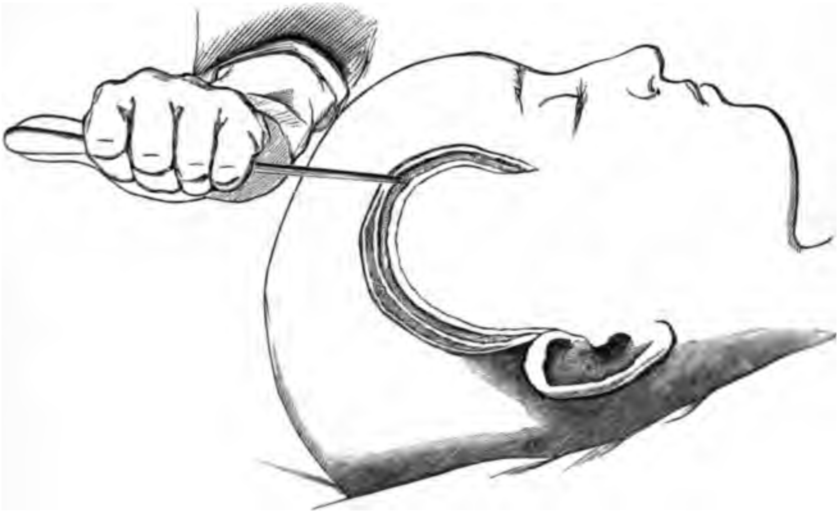
The Gasserian ganglion lies in a well-marked fossa on the anterior surface of the petrous bone, near its apex. It lies between two layers of the dura, the upper one being the dura proper, the other serving as the periosteum of the fossa. Removal of the ganglion was first suggested by Dr. J. Ewing Mears, of Philadelphia (*Trans. Amer. Surg. Assoc.*, 1884, p. 483). Mr. Rose, of London, was the first actually to perform the operation (*Brit. Med. Journ.*, 1890, i, 1012). Soon afterward, Andrews, of Chicago (*Journ. Amer. Med. Assoc.*, 1891, ii, 168), devised a similar operation. Hartley (*N. Y. Med. Journ.*, March 19, 1892, and *Annals of Surg.*, May, 1893, 512) has modified the operation, and Krause (*Arch. f. Klin. Chir.*, 1892, vol. xliv, 821) also independently described a similar procedure.

Rose's Operation. The eyelids were stitched together by Mr. Rose, but later experience seems to show that this is unnecessary. An almost semicircular incision is made from near the outer canthus to an inch below the external angular process, backward along the upper border of the zygoma, to its posterior extremity. It is then carried down just in front of the ear to the angle of the jaw, then forward along the lower border of the horizontal ramus, as far as the facial artery. This flap of skin is then dissected forward. Two holes are drilled in the zygoma to facilitate its later wiring, and the zygoma is then divided and displaced downward, along with the masseter muscle. The coronoid process is then similarly drilled, divided, and turned upward, with the tendon of the temporal muscle. (In Mr. Rose's later cases this fragment of bone was removed as useless, the drilling being therefore omitted.) The external pterygoid muscle is then scraped loose from the sphenoid bone and the foramen ovale is found. A long-handled, half-inch trephine is used, the centre-pin of which is placed a little external and anterior to the foramen ovale, so that the edge of the trephine opening just reaches the foramen. The thin lining of the fossa, in which the ganglion lies (the lower layer of the dura), is then divided, and the ganglion removed as far as possible by a hook and curette. If the internal maxillary artery or vein are in the way they are double-ligated and divided. Care should be taken not to divide the upper layer of the true dura above the ganglion, as by so doing the subdural cavity is opened. The carotid artery and the cavernous sinus lie immediately to the inner side of the ganglion and should be carefully avoided. The second and third divisions of the fifth nerve are readily found and removed by means of a small hook or sharp spoon. It is doubtful whether the motor root can be left intact.

A forehead electric light is a necessity in this operation. In that of Hartley it is a great aid, but the operation has been satisfactorily done without it.

Hartley's method is as follows. An osteoplastic resection of the skull is made, the base of which is two inches antero-posteriorly, and its vertical height three inches (Fig. 327). After the skull is opened (Fig. 328) the dura is lifted gently from the middle fossa by means of the finger until the second and third divisions of the fifth nerve are well exposed. Care must

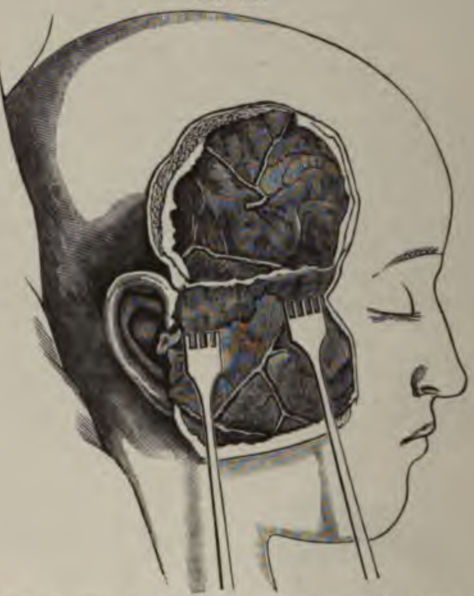
FIG. 327.



Hartley's operation for removal of the Gasserian ganglion. Chiseling the bony flap.

be taken not to rupture the middle meningeal artery at the foramen spinosum. If ruptured it can be ligated by passing a sharply curved needle under it through the dura. The hemorrhage can also be arrested by packing with iodoform gauze. The ganglion can be found by tracing the second and third divisions backward to their junction. As already stated, it lies in a pocket formed by the lower layer of the dura mater, which serves as a periosteum to the fossa in the petrous bone and the upper layer or dura mater proper. This lower layer must be perforated, but it is desirable to avoid opening the true dura, as that opens the brain cavity itself. (If there is not sufficient room, Tiffany has proposed to obtain more by deliberately incising the dura at the outer part, where it can be well sutured again, in order to evacuate some of the cerebro-spinal fluid.) The nerves should then be cut at their foramina, the ends forced downward, the foramina filled with dental paste, and the ganglion and nerve-roots removed. The latter can be easily removed, but the former must be broken up piecemeal. Great care is necessary to avoid wounding the cavernous sinus or the carotid artery which lie just beyond the ganglion and the origin of the second division of the fifth nerve. If there is too much hemorrhage to remove the ganglion at once, the wound may be packed with gauze, the skull closed temporarily, and reopened three or four days later, when the operation is completed.

FIG. 328.



Hartley's operation for removal of the Gasserian ganglion. The flap of bone and scalp turned down, exposing the dura and the middle meningeal artery.

I have recently collected all the reported cases of removal of the ganglion, 54 in number. Of these, 25 each were done by Hartley's and by Rose's method, with 3 deaths by Hartley's and 5 by Rose's method. Two were done by Horsley's method (opening the dura and avulsion or division of the nerve-roots at the pons), one by a method combining Hartley's and Rose's, and one in which the method was not stated. Two of these last 4 cases died.

INTRA-CRANIAL HEMORRHAGE.

Spontaneous Hemorrhage, especially in the form of the well-known lesions of apoplexy, arises from rupture of the lenticulo-striate artery distributed to the basal ganglia of the brain. For the relief of this surgery has very little to offer. Spontaneous rupture of the artery at one point is almost always an indication of widely distributed degeneration, and therefore no surgical measure can be of any use. Horsley has recommended, in the ingravescent forms of apoplexy, ligation of the common carotid. So far as I know, the two cases (one of which recovered) reported by Dercum and myself (*Journ. Nervous and Mental Dis.*, September, 1894) are the only two thus far recorded.

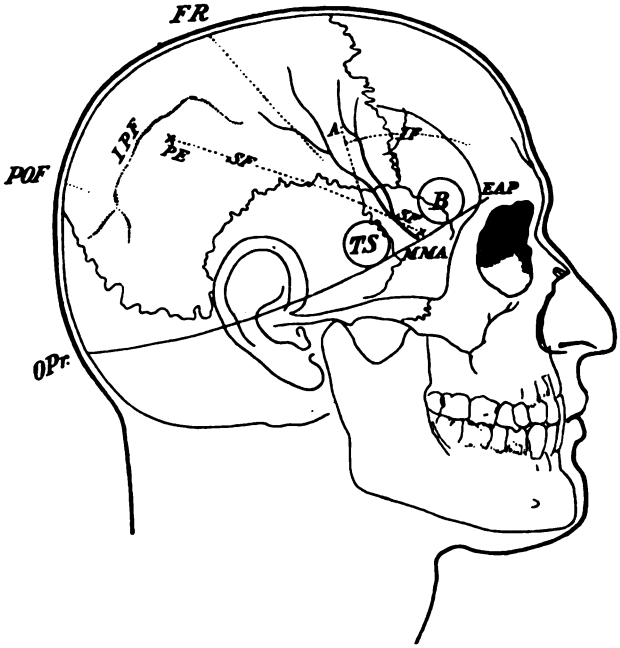
Traumatic Hemorrhage. This may be either (1) extradural, between the dura mater and the skull; (2) subdural, between the dura and the brain; or (3) cerebral, in the brain tissue itself.

1. *Extradural Hemorrhage.* The source of the hemorrhage in this variety is almost invariably the middle meningeal. Not uncommonly the injury may be so slight as not even to leave a bruise. Fracture, therefore, although frequently present, may be absent. More commonly by far, the rupture takes place in one of the branches rather than in the main trunk. Occasionally it is bilateral, and sometimes there are multiple ruptures.

Symptoms. As a rule, the patient is stunned by the blow, then recovers consciousness, and finally relapses again into a comatose condition. A large experience has shown that this period of consciousness between the unconsciousness produced by the blow and the unconsciousness produced by the clot is of the greatest importance. This temporary consciousness, which may not be absolutely complete, is due to the fact that while the rupture of the artery takes place at the moment of the injury, a certain time is required for the pouring out of a sufficient quantity of blood to produce coma from pressure. The larger the vessel which is injured, the more rapid will be the escape of the blood and the more quickly will unconsciousness recur. In case one of the small branches is ruptured, the interval of consciousness may extend not only to hours, but even to days. If a large branch or the main trunk is ruptured, the period of consciousness may be exceedingly brief or even absent. This symptom, therefore, should always be investigated with minute care. If the rupture takes place, as usual, in the neighborhood of the motor area, there may be paralysis, amounting, it may be, to complete hemiplegia. On the other hand, if the clot begins, for instance, over the face centre, there will be paralysis of the face; as it extends upward over the arm centre, and finally, the leg centre, the arm and the leg will successively be paralyzed. If the clot, instead of extending upward, gravitates toward the base, the pupil on that side will be dilated (though dilatation of the pupil sometimes occurs when the hemorrhage is over the vertex) and immobile, and if the rupture be on the left side of the head, there will be aphasia. The pulse, at first slow, later becomes rapid, the respiration, on the contrary, slow and stertorous. The temperature usually rises to 101° to 103°. If there be a compound fracture involving a perforation of the skull and the meninges, the brain substance may protrude through the wound. (See also page 363.)

Treatment. The instant that the diagnosis is made, operation should be undertaken as quickly as possible. No other means will arrest the hemorrhage. The statistics of Weissmann show that of 147 cases treated expectantly, 89.1 per cent. died, whereas of 110 cases in which operation was undertaken, only 32.7 per cent. died, and in the majority of the fatal cases the clot was not reached. The point at which the operation should be done, it cannot be too strongly stated, is to be determined not by the site of the injury, but by cerebral localization. In the great majority of cases the clot will best be reached by trephining 1¼ inches behind the external angular process, at the level of the upper border of the orbit (Fig. 329 between *B* and *TS*, and Fig. 330). Should this opening not disclose the clot, another trephine opening should be made at the same level, immediately below the parietal boss (Fig. 330). These two openings reach respectively the anterior and posterior branches of the middle meningeal artery. So soon as the clot is discovered it should be removed, and if the opening is not sufficiently large, more bone should be removed by the rongeur forceps. If dilatation of the pupil shows that the clot is gravitating toward the base, the trephine opening should be made half an inch below the first point. After the removal of the clot the cavity should be well washed out with cooled boiled water. The artery should be secured if it is still bleeding, by means of the small semicircular Hagedorn needle. Drainage should then be provided for and the wound treated as already described. It is sometimes necessary to trephine on both sides of the head.

Fig. 329.



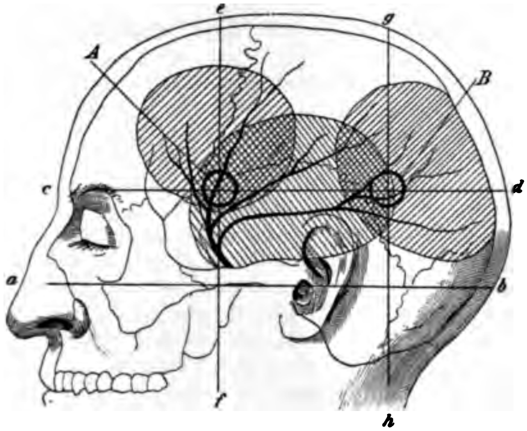
Head, skull, and cerebral fissures (adapted from Marshall by Hare).

B corresponds to Broca's convolution; *EAP*, external angular process; *FR*, fissure of Rolando; *IF*, inferior frontal sulcus; *IPF*, intra-parietal sulcus; *MMA*, middle meningeal artery; *OPr*, occipital protuberance; *PE*, parietal eminence; *POF*, parieto-occipital fissure; *SF*, Sylvan fissure; *A*, its ascending limb; *TS*, tip of temporo-sphenoidal lobe. The pterion (to the left of and above *B*) is the region where three sutures meet, viz., those bounding the great wing of the sphenoid where it joins the frontal, parietal and temporal bones.

2. *Subdural Hemorrhage.* This more commonly follows depressed fractures, gunshot fractures, rupture of the middle cerebral artery, etc. There are no means at present of distinguishing between subdural and extradural

hemorrhage. In a very recent case which I saw, the diagnosis of the location of the clot was very accurately made, from the fact that the patient had hemianopsia, the left half of each retina being blind. Trephining below the parietal boss would have disclosed the clot, but the patient was moribund when first seen.

FIG. 330.



Points at which to trephine in hemorrhage from the middle meningeal artery. (After KRONLEIN, ESMARCH and KOWALSKY.)

a b, horizontal line drawn through the meatus; *c d*, through the eyebrow; *e f*, vertical line, ¾ c.m. behind the external angular process; *g h*, immediately behind the mastoid process. The intersections *A* and *B* mark the points for trephining to find respectively the anterior and posterior branches of the artery.

Treatment. The only difference in the treatment of subdural and extradural hemorrhage is that the dura must be opened, and the opening in the bone may require to be much larger than in the former case. The vessels should be secured by catgut. Not a little disorganized brain-tissue may have to be removed, and I think it of some importance that all of this shall be removed, if possible, in order to lessen the probability of later epilepsy.

3. *Hemorrhage into the Brain Substance.* As a rule, traumatisms involving the brain so deeply are rapidly fatal. The cases resemble those of ordinary apoplexy, except in the cause, which is revealed by the history.

4. In addition to such hemorrhages from arteries, the sinuses of the brain may be injured and cause violent and alarming hemorrhage. Such wounds are due usually to compound fractures, the superior longitudinal or the lateral sinus suffering most frequently. Death may take place in a few minutes, from loss of blood, and shock, and hence the speediest method of checking the bleeding should be adopted.

Not infrequently it is necessary to trephine in the neighborhood of these sinuses. The trephine should be so placed that its edge is at a safe distance from the sinus. With the dural separator, grooved director or other blunt instrument, or the finger, the sinus may be safely separated from the skull. Then the trephine opening may be enlarged, as may be necessary by the rongeur forceps.

Should the sinus be ruptured either by operation or by fracture, instant packing with strips of iodoform gauze will almost always control the bleeding. Occasionally, if the rent is not too large, its margins can be seized by one or more pairs of hemostatic forceps, which should be left for two or three days, with, of course, an ample dressing outside of them. Care should be taken also that they be not displaced by the patient's movements. Lateral ligature or suture of the sinuses can occasionally be adopted.

5. The hemorrhage from pachymeningitis hemorrhagica has already been considered (page 971).

FRACTURE OF THE SKULL.

The danger from fracture of the skull is not so much from the injury to the bone as from the injury to the brain or its membranes. Even linear fracture itself is occasioned by such serious force that the brain is lacerated to a greater or less extent, and if fragments of bone are depressed or detached and driven into the brain, the injury becomes instantly severe, and very frequently fatal. Such injuries formerly were considered almost entirely hopeless; but a large number of cases of recovery after proper antiseptic treatment have now been reported, and the prognosis, even in cases of very widespread injury, is by no means so hopeless as it used to be. Next to very extensive fractures, the most dangerous are the punctured fractures, such as those caused by a knife-blade, nail, etc., because they are very frequently overlooked, or, if discovered, are improperly treated.

The division of fractures into closed and open, *i. e.*, simple and compound, obtains in the skull as elsewhere, but it is especially to be remembered that fractures extending into the nose, ear, or mouth are as truly compound as a compound fracture of the vault, and, like the punctured fractures, their existence is often overlooked, and hence the wound becomes infected, and death results. I shall especially dwell upon the treatment, as the symptoms and diagnosis are considered so fully in works on general surgery.

FRACTURES OF THE VAULT. In fissured fractures, as a rule, the injury can only be suspected. In a few cases a " cracked-pot " sound may be elicited by percussion and be observed even without the stethoscope. It may be heard also by the patient as well as by the surgeon. If there is a depression it can usually be easily felt by the finger, but care must be taken not to mistake the thickened tissue at the periphery of the injury for the edges of a depressed fracture, which they not uncommonly simulate. Fracture of the inner table can likewise only be inferentially established. Although such a diagnosis is rare, a number of well-authenticated instances are reported, and if the symptoms are such as to determine its localization, as for instance a palsy of speech, or of the right arm, etc., and the site of the blow be over these centres, we should be warranted in an immediate exploratory operation.

Treatment. The tendency of modern cerebral surgery is distinctly toward more frequent interference than formerly. The objection which ruled twenty years ago—that an incision through the scalp in a case of simple fracture converted it into a compound, and, therefore, more dangerous fracture—now falls to the ground, since the danger is very slightly, if at all, increased by such an incision, if done antiseptically. Moreover, the need for the elevation of depressed fragments for the removal of pieces of bone which have been driven into the brain, and in compound fractures especially, the need for thorough disinfection of the wound in order to prevent septic inflammation outweigh all other considerations. Fractures which occupy a limited area, for instance, one from a blow from a hammer, must be carefully distinguished from those in which the "bursting" force of a diffused injury may produce very widespread injury to both bone and brain. As a rule, in the latter, operation would rarely be advisable.

The dictum which seems at present to meet with the approval of the majority of surgeons may be summarized as follows:

If a fracture of the vault be simple, without depression and without symptoms of injury to the brain, or if moderate cerebral symptoms have been present but are subsiding, or if fracture be only suspected, and no cerebral symptoms are present, the expectant treatment is the proper one; but the instant that any serious symptoms of intra-cranial mischief arise, operation should be resorted to.

If the fracture be simple, with marked depression, immediate trephining should be done, even should there be no signs of cerebral mischief. Any injury sufficient to depress the bone will undoubtedly have lacerated the brain substance, and may be followed either by speedy inflammation, or if recovery ensues, in not a few cases by epilepsy or other nervous sequels which are best avoided by operation.

In all compound fractures of the skull operation should be immediately done. I am persuaded that in many cases infection follows from the limited area over which the scalp is shaven and disinfected. Hence, as a partial shaving is equally disfiguring, the proper course is to shave the entire head and carefully disinfect it. Even in women this rule should hold good. A large incision should be made in the scalp. If the fracture be found to be linear it must be carefully disinfected, and if impregnated with dirt, or if any of the hair has got into it, the outer table at least must be carefully chiseled away as a V-shaped groove. The fracture is of little importance, infection of great. Should a depression exist, trephining should be done and the bone elevated, independently of the existence of any cerebral symptoms.

Punctured fractures, as an absolute rule, should always be treated by immediate trephining for many evident reasons.

Should any cerebral mischief arise, even at a late period, after fracture, such as localized paralysis, epilepsy, optic neuritis, etc., operation should be done immediately.

FRACTURES OF THE BASE. These fractures may arise either directly or from extension of fractures of the vault, or by indirect violence through the spinal column. Occasionally they are punctured fractures, the vulnerating instrument obtaining access to the base either through the orbit, the nose, or the mouth.

Fractures of the base are not uncommonly only inferential from the violence of the blow, but occasionally we may be able to follow a fracture well toward the base. It must not be forgotten that fractures through the cavities just mentioned (orbit, nose, or mouth) may be overlooked from the fact that in the nose and mouth the cavity is closed, and in the orbit the wound in the skin, by its resemblance to a simple cut, may mislead us into believing that there is no deeper injury. Yet a splinter of wood or a knife blade may be hidden deep under the skin. Of course the escape of brain substance or cerebro-spinal fluid through the ear, nose, mouth, or orbit, is positive evidence. If blood has been vomited after a severe injury to the head, care should be always taken to determine whether there has not been a fracture of the roof or the pharynx, the blood being first swallowed and then vomited.

The commonest sign of fracture of the base in the middle fossa is the escape of blood or cerebro-spinal fluid from the ear. If either of these continues for a long time, and especially if the escape of the fluid is affected by the position of the head and is increased by any violent expiratory effort, such as sneezing, coughing, etc., it is always positive evidence of such a fracture. Sometimes, however, there may be fracture of the base without rupture of the drum-head, and therefore no cerebro-spinal fluid can escape. Such fractures, it must be remembered, are compound, as they communicate with the external air through the Eustachian tube, and the danger of overlooking them is very great. Injury to any of the cranial nerves, especially the optic, facial, and auditory, is strong inferential evidence of fracture of the base.

In fracture of the orbit, blood will appear in a day or two under the conjunctiva, before it appears in the eyelid.

In fracture of the posterior fossa, optic neuritis sometimes occurs, usually before the end of the first week. It is extremely important in fractures of this fossa that the mastoid region should be examined for ecchymosis. This is often overlooked in consequence of its being hidden by the ear, the pillow, and the hair. The blood first appears at the mastoid, some time after the injury, and spreads chiefly upward and backward, with a crescentic border. As Battle has shown, the reason for these peculiarities is that the blood cannot reach the surface below the occiput on account of the dense cervical fascia, but follows the inter-muscular planes sidewise, and first reaches the surface at the mastoid after two or three days.

Treatment. These injuries were formerly excessively fatal, but in numerous recent cases recovery has followed judicious treatment. First the cavity or cavities involved must be carefully disinfected. The ear should be cleansed of wax, dirt, and blood-clot, and disinfected by prolonged syringing with a 1 to 2000 sublimate solution. It should then be packed with sublimated cotton or iodoform gauze and covered with an ample sublimate dressing. In fractures through the orbit the same rule should be carried out, and if the fracture be a punctured one, thus almost positively insuring infection of the brain, the wound should be enlarged, very thoroughly disinfected, and as a general rule, drained, sometimes best through the nasal cavity.

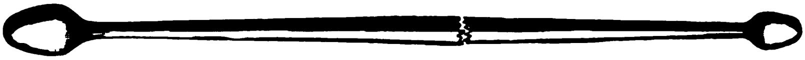
In the nose and mouth there would of course be danger in using the corrosive sublimate solution. They should, however, be thoroughly washed out with hot water and then with boric acid solution. This or other antiseptic solutions should also be sprayed through the mouth and nostrils. The nose should be lightly packed with sublimated iodoform or sterilized cotton. Boric acid or iodoform may also be insufflated into the naso-pharynx, thus reaching the orifice of the Eustachian tube. These measures only result in a partial asepsis, but the danger of infection is directly proportionate to the number of bacteria introduced into a wound, and the results in treatment have been so favorable that no surgeon would be excused if he neglected them. Of course the ordinary dietetic and hygienic treatment which has long been applied to such injuries should be most carefully carried out.

WOUNDS OF THE BRAIN, ESPECIALLY GUNSHOT WOUNDS.

In all wounds of the brain besides the usual careful disinfection, all dirt and foreign bodies should be removed. Fragments of bone, which may have been driven into the brain, should be removed, search being made by the finger and groove director, gently, but thoroughly, lest some of the fragments should be overlooked. The depressed bone should be removed. The brain tissue itself is also to be disinfected. The hemorrhage is arrested by the ligature, hot water, or pressure. If the sinuses have been wounded, packing with iodoform gauze, or the application of hæmostatic forceps, will usually suffice. If the dura has been only slightly lacerated, it may be sutured with catgut or fine silk. If there is any great loss of the dura, this may be supplied by a portion of the pericranium (see page 965). Drainage should be provided by rubber tubing, the scalp sutured, and an ample sublimate dressing be applied. Secondary abscesses are not uncommon after such wounds. The surgeon should be watchful, therefore, and if any symptoms of such abscesses arise, the wound should be reopened, and the pus evacuated as soon as possible.

In gunshot wounds the entire track of the wound, from that of entrance to that of exit, or to the probable site of the ball, if it have not escaped, should be disinfected. This may be done by a bichloride solution, 1-2000, terminating the disinfection by washing with warm boiled water. Should there be any serious hemorrhage either in this or in any other wound of the brain, the wound of entrance or that of exit, or both, should be enlarged by the rongeur forceps or by the trephine, and the vessels secured as usual. The bullet or fragments of bone, or other foreign bodies, should be removed. A counter opening may be necessary in order to remove the bullet, the axis of the wound having been determined by Fluhrer's aluminum gravity probe. (See Fig. 331.) The ball is sought for gently but thoroughly, and removed through the wound of entrance, or if it be much nearer the opposite side of the skull, by a counter trephine opening at the point determined by prolonging the axis of the probe. Drainage must be thoroughly carried out, if need be by a counter-opening, the drainage-tube being carried entirely through the brain. To locate the ball Girdner's telephone probe may be used. The mortality in the cases in which the ball is not removed is about 55 per cent., of those in which it is removed, about 33 per cent. If the ball cannot be found without too much laceration of the brain, the search for it should be abandoned. Thorough disinfection and drainage of the wound in a number of cases have been followed by recovery.

FIG. 331.



Fluhrer's aluminum probe (natural size except the length—12 inches).

CONCUSSION OR LACERATION OF THE BRAIN.

The old term concussion of the brain, while very convenient clinically, conveys pathologically a wrong idea, as it implies rather a functional than a physical lesion. As a matter of fact, almost all modern surgeons are in accord in the belief that any injury, certainly any which passes beyond a very slight stunning, practically involves a laceration of the brain tissue.

The surgical treatment consists, first, of absolute rest in bed, together with those means to induce entire quiet of mind and body, and reaction from shock, which are well known. To this end, hot water bags should be placed about the patient, great care being taken that he be not burned by them. The most careful diet with occasional purgatives and attention to general hygienic conditions are necessary for a more or less prolonged time. As a stimulant during the period of shock the aromatic spirit of ammonia is better than alcohol, which produces too much cerebral excitement. As to the later treatment, while there is considerable difference of opinion at the present time, my own opinion is, if serious symptoms arise indicative of intra-cranial mischief, and particularly if the symptoms are localized, that a semicircular flap of scalp should be raised and the bone inspected. If a fracture is discovered an exploratory trephining should be done, and the dura opened. Traumatic inflammation is always attended with proliferation and exudation, and if the exudate be not afforded the means of escape before inflammation or even suppuration have arisen great danger to life will exist. Trephining in these cases is simply equivalent to an incision down to the focus of inflammation in the soft parts, the only difference being (and this is a serious one) that in order to reach the focus of inflammation in the skull, an opening in the bone must be made. If this is not done there is no possible means of escape for the hemorrhage or the exudate, which threatens suppuration.

If the contusion be severe, and especially if the swelling of the scalp is so thick and dense that it is difficult to determine whether a fracture exists or not, such an exploratory incision should undoubtedly be made, and followed by trephining if necessary. The old objection that we thus convert a simple fracture into a compound one does not, at present, hold good in view of the safety of modern antiseptic surgery, and even if there were considerable danger, the dangers of not operating, and, therefore, of an uncertain diagnosis and unsuitable treatment, outweigh any possible dangers of such an incision if properly done. Even after a long period an abscess may arise. Every case of severe contusion of the skull therefore should be watched for weeks, and even months, and at the first indication of any such symptoms, operative interference would be justified.

COMPRESSION OF THE BRAIN.

The treatment of this condition depends entirely upon its cause. If there has been an injury of the skull and the indications are those of intra-cranial hemorrhage, instant trephining should be done at the point or points and in the manner described under the head of intra-cranial hemorrhage (p. 981). If there is a depressed fracture, trephining should be done as indicated in the treatment of that subject, and the bone removed or lifted (p. 984). If there are foreign bodies in the brain they should be removed with the fragments as stated under that heading (p. 986). If from abscess of the brain, trephining should be done, the abscess evacuated and drained (p. 971). If the compression arises from internal hydrocephalus, the patient should be trephined and the exudate drained by puncture of the ventricles (p. 968). If the compression is caused by a tumor, the tumor should be removed if the case is one suitable for such operation (p. 976). The later treatment consists of rest, restricted diet, attention to the bowels and bladder, the absolute absence of visitors or anything that can cause mental or physical excitement, together with such general hygienic precautions as are suitable to the case.

SURGERY OF THE SPINAL CORD.

The progress which has been made in the surgical treatment of diseases of the spinal cord, especially in the operative treatment, within the last few years, is very remarkable. The various surgical procedures are summarized as follows:

SPINA BIFIDA. The treatment of spina bifida by the injection of Morton's fluid (iodine gr. x., iodide of potassium, gr. xxx., glycerin ℥j), has proved in very many cases effectual. In 1885 a committee of the Clinical Society of London made a thorough report on the methods of treatment of spina bifida. Only fourteen cases out of sixty were known to have been cured spontaneously. In seventy-one cases treated with Morton's fluid the mortality was shown to be 38 per cent. Morton, himself, in twenty-nine later cases, however, has reported a mortality of only six, or 20.7 per cent. Powers (*N. Y. Med. Journ.*, July 16, 1892, page 65), has collected fifteen more cases with four deaths, a mortality of 26.66 per cent.

The method of operating is as follows: After thorough disinfection of the skin about one drachm of the cerebro-spinal fluid is evacuated and a drachm of the solution injected. This injection should be made at the side through healthy skin and not through the membranous portion of the sac, and care should be taken not to wound the nerves. Leakage of the cerebro-spinal fluid should be prevented by sealing the opening by iodoform collodion and a gauze dressing secured also with collodion. Later injections, if needed, should be made at intervals of a week or ten days.

The treatment of this malformation by operation, however, has been winning its way greatly into favor, and as the technique improves, it is fairly certain that the percentage of recoveries after excision will increase and make this the method of choice. Powers (*loc. cit.*) has tabulated thirty cases of excision with twenty-four recoveries and seven deaths, a mortality of 22.58 per cent. The mortality, therefore, is about the same as that after using Morton's fluid, but will probably improve very much as our experience grows. Hildebrand (*Deutsch. Zeitschr. f. Chir.*, 1893, xxxvi., 515), has collected 87 operations with 23 deaths, a mortality of 26.4 per cent.

Of the three varieties of spina bifida, meningocele, meningo-myelocele, and hydromyelocele, only the first and second are amenable to treatment by excision. Even in these two forms, only those cases "in which the general condition of the patient is good, in which no paraplegia is present, in which the vertebral fissure is of moderate extent, and the communication between the sac and the canal is narrow" should be chosen for operation. (Powers.) In other cases less favorable for operation Morton's fluid should be used.

OPERATION IN MENINGOCELE. An elliptical incision is made leaving sufficient skin to cover in the defect. The sac is then dissected down to its base, the serous membrane at its neck being sutured with the two serous surfaces apposed, or if very small, the neck of the sac may be ligated, and the skin closed over it, and dressed as usual. It is very important, as far as possible, to prevent the escape of the cerebro-spinal fluid, partly on account of immediate danger to life, but especially to prevent a fistula, which may later become infected, and be followed by a septic meningitis.

OPERATION IN MENINGO-MYELOCELE. In this condition the nerves are commonly found attached to the posterior surface of the sac. Whether they are adherent or not can sometimes be determined in translucent tumors by transmitted light from a candle while the surgeon looks through a cylinder of paper applied at the opposite side. If present, the nerve-roots must be dissected loose from the sac and replaced in the canal. In other respects the operation is done as before.

Attempts have been made, however, within the last four years to close the bony opening in the spine and not merely the membranous opening. This has been effected by loosening the muscles on each side by using a strip of periosteum of the rabbit, the osteogenetic surface being turned undermost and the piece sutured in place to the periosteum, and by transplanting a piece of bone from the scapula of a young rabbit. The most important modifications, however, are as follows. Dollinger, with bone forceps divided the rudimentary portions of the arches of the fourth and fifth lumbar vertebræ close to their bases, and sutured them together in the middle line. Bobroff has chiselled from the iliac crest a piece of bone three centimetres wide, two long, and nearly one thick. The bone was not chiselled loose, but was turned over on a hinge, formed by the erector spinæ muscles, so that the periosteal surface was next the cord, the chiselled surface presenting posteriorly. The pieces of bone were then slid under the gluteus maximus, which had been dissected loose from the bone and were held in position by sutures through the bone. The patient was kept on the abdomen for two months, at the end of which time consolidation of the transplanted bone had taken place, and he was allowed to change his position. Five months after the operation the boy was in good condition, had recovered from his incontinence of urine and feces at night, and of feces during the day. In the dorsal region the same author proposes to obviate the difficulty by chiselling off the outer lamellæ of the adjacent ribs.

SPINAL DRAINAGE FOR TUBERCULAR MENINGITIS AND HYDROCEPHALUS. This has already been considered under the head of Hydrocephalus (page 968).

INTRA-SPINAL DIVISION OF THE NERVE-ROOTS IN INVETERATE NEURALGIA. This is considered on page 1002.

SPINAL HEMORRHAGE. It is doubtful whether in the present state of surgery any operation should be undertaken either in hæmato-myelia or hæmatorrhachis, though Mills has suggested the possibility of trephining the spine at two points to secure drainage. It is not impossible that, as our experience and technique become larger and better, this may be accomplished.

GUNSHOT WOUNDS OF THE SPINAL CORD. Many of these lesions are, of course, of so serious a character as to forbid any interference. But as Vincent has shown (*Rev. Chir.*, 1892, p. 89), first, in those cases in which the cord is compressed by extravasations of blood, fragments of bone, or by the projectile lying without the medullary canal, improvement may possibly be expected if we remove the source of the compression by operation; second, if the projectile has injured the cord, as the diagonsis of the extent of the injury cannot be made without an operation, exploration may be done, unless other fatal, especially visceral, lesions are present, and third, if the projectile lodge in the spinal canal an operation is certainly indicated, as fatal meningitis and myelitis with cystitis and bedsores will undoubtedly follow, unless it be removed.

LAMINECTOMY FOR POTT'S DISEASE. It is only since 1883 that such surgical interference in Pott's disease has been undertaken, and surgeons are not yet wholly in accord as to the indications for interference. "So long as we have reasonable or even moderate hope of recovery without operation, we do not believe it advisable or justifiable in the present condition of spinal surgery to perform so serious an operation as the resection of the laminæ." (Burrell and Bullard). "It is necessary before operating to have exhausted all other methods of treatment," says Kraske: "I would say interfere when the paralysis of the bladder is established. This is the one symptom which is so serious as to justify everything," to which dictum Lloyd adds "that the first sign of degeneration of the cord should indicate immediate operation." These opinions seem to me to indicate the proper medium between rashness and too great conservatism. Ordinary cases of mere curvature of the spine from Pott's disease are never to be operated upon, but only those in which paraplegia with paralysis of the bladder, cystitis, and bedsores have arisen should be considered as justifying operative interference. Of course, if there are other active tubercular complications, operation is absolutely contraindicated. The causes of pressure may be, first, masses of granulation tissue arising from the vertebræ within the spinal canal, or the thickening which results from pachymeningitis, occasionally amounting practically to a tumor, or, secondly, abscesses resulting from the breaking down of this granulation tissue. Either of these lesions, providing the conditions already stated exist, may be treated by laminectomy, with removal of the neoplasm, the granulation tissue, the pus, the carious bone, etc., by the usual operative means (*vide infra* Technique, page 993).

TREATMENT OF SPINAL ABSCESS. Spinal abscesses have been operated on for many years. Until within a few years, however, surgeons have been content to evacuate and drain such abscesses with or without curetting, or the injection of iodoform in ether or in olive oil. Mr. Treves (*Med. Chir. Trans.*, vol. lxvii., and *Manual of Operative Surgery*, ii. 731) has well described his improved method. The parts having been made aseptic, a vertical incision of two and a half inches is made upward between the crest of the ilium and the last rib, at about two and a half inches from the lumbar spines. The lumbar aponeurosis, with its attached muscular fibres, are divided the full length of the incision, exposing the erector spinæ, which is recognized by the vertical direction of its fibres. This muscle is drawn as far as possible toward the middle line, exposing the middle layer of the lumbar fascia. Through this the transverse processes of the lumbar vertebræ are sought for, especially the third, it being the most conspicuous and easily felt. This layer of the fascia is then divided to the transverse processes, exposing the thin quadratus lumborum muscle. This is divided close to the extremity of a transverse process, and the incision enlarged gently by the finger to the entire extent of the wound. Care must be taken not to wound the abdominal branches of the lumbar arteries. The psoas muscle, which overlaps the quadratus, is now discovered, the inter-muscular interval being marked by the thin but distinct anterior layer of the lumbar fascia. Some of the tendinous fibres of the psoas having been divided close to a transverse process, the finger is introduced beneath the muscle, following the process until the anterior aspect of the bodies of the vertebræ is reached. The abscess cavity will usually have been entered at some point, and should now be thoroughly opened and subjected to prolonged irrigation by a sublimate solution, 1-5000. During the irrigation the abscess may be gently curetted by the finger-nail or sharp spoon, care being taken not to break through the anterior wall of the abscess cavity. The cavity is then well scrubbed out, either with a fine sponge or with gauze sponges, every pocket and diverticulum being penetrated, the sponge being changed frequently, and the sublimate solution continuously used for flushing. When the abscess cavity appears to be clean, as shown by the sponge returning perfectly unsoiled, it should be dried and closed by silkworm-gut, the sutures being passed sufficiently deep to include the greater part of the soft tissues over it. It is closed without drainage. The after-treatment consists in absolute rest in the recumbent position for months, with attention to the favorable dietetic, hygienic, and climatic conditions as far as the means of the patient will allow. If suppuration recurs the wound should be reopened and the former treatment practically repeated, followed by free drainage.

Retro-pharyngeal abscesses, or tubercular abscesses, arising from the bodies of the cervical vertebræ, were formerly opened in the pharynx, but the methods proposed by Chiene and Burckhardt are better. These consist in an incision at the outer (Chiene) or inner (Burckhardt) border of the sterno-cleido-mastoid muscle; the bodies of the vertebræ are reached by a blunt dissection largely by the finger, the vessels in the neck being carefully avoided. When the cavity is reached it is to be treated as has been already described for lumbar or psoas abscess. The great advantage of these methods is that thorough antisepsis can be secured, while it is impossible to secure it through the mouth. The oral route, however, has generally a small mortality, and in the case of a timid or an inexperienced operator, may be chosen by preference.

FRACTURES AND DISLOCATIONS OF THE SPINE. This, again, is a subject in the treatment of which surgeons are by no means in accord, especially from the fact that it is so difficult to diagnosticate precisely the nature and extent of the lesion. It is argued on the one hand that in these destructive lesions, especially if they be total transverse lesions followed by entire paralysis both of motion and sensation, with complete abolition of the knee-jerks and other deep reflexes on both sides, no benefit whatever can be derived from operation. On the other hand, however, the evidences adduced, especially by the condition of the knee-jerk, it is argued, are insufficient to prove absolutely that the lesion is a total transverse lesion, entirely destroying the cord, and that operation is the only means of determining whether the lesion is complete; and that a few cases have been reported in which operation, even when total abolition of the knee-jerks exists, has been followed by partial, or in some cases by complete relief. A notable instance of this is given by Schede (*Annals of Surgery*, September, 1892, p. 231) in which all of the symptoms above described were present, and bedsores had begun to form the next day. Sixteen hours after the accident Schede operated, removing the bony fragments pressing on the cord, the dura being uninjured, but the cord being soft and fluctuating. The patient not only recovered the functions of the bladder and rectum, but two months after his discharge was in excellent health. These cases are, as a rule, so helpless without operation, that my own feeling is in favor of operation in suitable cases, especially as the mortality of the expectant plan is so great. Many surgeons have approved of the rule of Lauenstein, that if after from six to ten weeks there is incontinence of urine and feces with cystitis and bedsores, little is to be hoped for from nature's efforts, and an operation is justifiable. Horsley, however, is much more emphatic, and says "in all cases where displacement or crepitus indicates compression, and extension directly after the accident fails to reduce the displacement," we should operate. In view of the early degeneration of the cord, if the pressure be not relieved, of the great mortality in such cases when not operated on, and of the rare but undoubted instances of good results following early operation, the tendency of modern surgeons is toward earlier rather than later interference, if any interference is considered justifiable. In no case can we be sure that ultimate benefit cannot be obtained, until at least a year after the operation. For the technique of laminectomy in these cases, see page 993. The dangers of the operation as formulated by Mr. Horsley are hemorrhage, difficulty in clearing the neural canal, physical difficulties in treating the fractured vertebræ, damage to the spinal cord in many cases, and septic infection. To this White has properly added the danger of anæsthesia in the prone position, the abdominal muscles being paralyzed.

The results of the operative and non-operative treatment are strikingly in contrast. Thus Gurlt has tabulated two hundred and seventy non-operative cases with two hundred and seventeen deaths, a mortality of 80 per cent., while Thorburn, though he opposes the operation, has collected sixty-one operative cases in which only thirty-five died, a mortality of only 57 per cent.

In two cases of dislocation without extensive injury to the cord, Wilkins and Hadra have immobilized the vertebræ by silk or wire. This method possibly merits further consideration. In a recent case of paralysis from elongation of the cervical nerve-roots as a result of dislocation, Chipault has obtained a brilliant success by operation and wiring the spinous processes to retain the vertebræ in position.

TUMORS OF THE SPINAL CORD. Though operation was performed in 1887 by Johnson, the modern surgery of spinal tumors practically dates from the brilliant paper of Gowers and Horsley (*Med.-Chir. Trans.*, 1888). Minute care is necessary in the location of the tumor. As a rule, as has been shown by Horsley and Gowers, we are apt to attack the spine too low, and if the tumor be not found at the level at which it is presumed to exist, the cord should be exposed further upward rather than downward. The means for location of the tumor have been already described on pages 608, 616, and 617, attention being especially paid to the exact location of the upper border of even the slightest paræsthetic zone. The favorite position of such tumors is either below the middle of the cervical region, or at the two extremes of the dorsal region, occasionally in the cauda equina and elsewhere. In tumors involving the cauda equina, which consists practically of peripheral nerves, the prognosis is much better than in those tumors which affect the spine proper. The operation consists in a laminectomy (*vide infra* Technique) exposing the cord. If it is deemed advisable the dura is opened and the tumor treated in accordance with what is found. If the tumor be distinct from the cord it may be removed, but if it involve the cord itself it is usually inoperable. If opened, the dura is to be sutured and the wound treated as usual.

SYRINGOMYELIA. Abbe has reported the only case, so far as I know, in which an operation has been done in this form of glioma. I have myself operated on one, not yet reported, and a second which, I believe, was probably a syringomyelia. No benefit followed Abbe's operation, and but little my own second operation. My first case died in five days. It would seem best, therefore, not to operate in such cases.

TECHNIQUE OF LAMINECTOMY. Shaving if necessary, and extensive disinfection of the back should precede the operation. The ordinary antiseptic preparation, also, of the hands, instruments, and dressings, is, of course, to be insisted upon. About two dozen hæmostatic forceps, and various rongeur and other bone forceps and raspatories should be provided. Of the rongeur forceps, the double rongeur and the rongeur forceps, which I originally devised for linear craniotomy, and which I have found to be the best for removing the laminæ of the vertebræ, and the Hopkins rongeur forceps are among the most useful (see Figs. 311, 312, and 313, pp. 959 and 960). The patient is placed on the abdomen, the anæsthetic, therefore, being administered in the most unfavorable position. If the injury is in the upper spine, the muscles of the abdomen and chest are paralyzed, and no one should administer the anæsthetic but a reliable man, of large experience and good judgment. Strychnine, digitalis, and atropine are to be administered as needed. The patient should also be well protected from cold by blankets and hot water bottles. An incision is made in the middle line four inches or more in length, according to the extent of the spine to be attacked. Various other incisions, T, V, and H in shape, have been proposed. The H incision of Bullard and Burrell allows the posterior arches to be lifted with the muscles and replaced later. This, however, seems to me to be of no special advantage, and I have always operated by a single median incision which has proved quite satisfactory. If the edges cannot be separated widely enough a transverse incision may be made through the fascia. The laminæ on one side of the spine are bared partly by the knife and partly by the raspatory. So far as possible the muscles should be separated from the bone by the knife, blunt dissection being avoided, as it tears the fascia and muscles into fragments, which are apt later to undergo necrosis with probable suppuration. When the muscles have been cut away the raspatory is then used in order to expose the arches cleanly. As soon as one side has been cleared it is to be packed with sponges wrung out of water as hot as can be borne, to check the hemorrhage while the opposite side is being cleared. When this has been done a clean field is secured. The interspinous ligaments are then to be cut, care being taken especially in the cervical and lumbar regions not to penetrate the canal and possibly wound the dura and cord. By the double rongeur forceps one of the spines is gnawed away with a part of one of the arches; as soon as the lower blade of my rongeur forceps can be inserted under the lamina it can be bitten away rapidly, thus opening the spinal canal and exposing the fatty tissue between the theca and the bones. This fatty tissue is to be divided in the middle line and gently crowded to each side of the theca with small bits of gauze or sponge. This removes the fatty tissue mechanically, and at the same time checks its hemorrhage. It should be noticed then whether the dura pulsates or not. Often there are adhesions or other causes of interference in the continuity of the subdural space. The color of the dura may indicate the presence of blood or pus. Its tension will show whether there is an excess of cerebro-spinal fluid or not. This is determined by touch, but it must be remembered that the cord may be thickened by a syringomyelia, which will simulate very much the presence of fluid. A tumor or thickening of the dura itself can usually be appreciated, to some extent, by touch. If it be desired to examine the bodies of the vertebrae, the cord and its membranes can be carefully drawn to one side by means of an aneurism needle, so as to expose the bodies of the vertebrae for observation, and if need be, for operation. The nerves are sufficiently elastic to allow of moderate stretching. If a tumor of granulation tissue exist on the surface of the dura, it is now to be removed. Any dislocated or fractured arches of the vertebrae should be removed. The opening of the spinal dura is more serious than that of the brain. In the latter the opening can be closed without drainage, thus preventing the continuous escape of the cerebro-spinal fluid. In the spine, the wound through the thick muscles, which are often seriously injured in cleaning them from the bone, require drainage, and often will not unite by first intention. As long as the fluid escapes there is much danger of a fistula, which not only is a source of irritation to the skin and an annoyance from the constant dressing it requires, but is a source of serious danger from subsequent infection and meningitis. The mere escape of the fluid itself is not very dangerous. In spite of these objections, however, it is usually best to open the dura. After such treatment as is necessary it is to be closely sutured with a continuous catgut suture. When it has been opened the dural separator of Horsley (see Fig. 319, p. 962) or an ordinary probe bent at a suitable angle, should be inserted upward and downward to explore the condition of the subdural space. If a tumor of the cord has been suspected, but not found, it is to be again remembered that it is much more apt to be higher up than lower down. When found, if it be on the surface of the cord, it may be removed, not uncommonly, quite easily, but if it infiltrate the substance of the cord it is inadvisable to attempt its removal. If the cord has been injured or crushed little can be done beyond freeing it from pressure. Suture of the cord has been attempted but not successfully. The nerves have, however, been sutured by Tuffier.

Occasionally where it is desired to relieve pressure the dura may be left open. In one case of my own I left it open for the space of two inches. Horsley has left it unsutured for four inches. For the suture the finest semicircular Hagedorn needle and a needle holder are the best means. No drain should be inserted under the dura, but for twenty-four or forty-eight hours it is, as a rule, best to drain the muscular wound. The muscles should be approximated by buried catgut sutures, the skin being sutured by silkworm-gut and the usual dressings applied.

After-treatment. Within the first twenty-four hours the oozing, and the probable escape of some cerebro-spinal fluid will be so free as to require one or two dressings. The greatest care should be observed in these redressings, lest infection should occur. Especial care should be taken that this contamination does not arise from any bedsores, which are so often present, nor from the urine or feces if control of the bladder and bowels has been lost. During the operation any bedsores should be carefully covered by bichloride towels, and the wound should be dressed sufficiently frequent to prevent contamination through soiling of the dressings from any of these causes. Thorburn has proposed to drain the bladder by a supra-pubic cystotomy to prevent the constant wetting of the wound from the incontinence of the urine. I do not know of any case in which it has been done, though the suggestion seems reasonable. A marked improvement often takes place in the bedsores, not uncommonly even to complete cure. Scrupulous attention should be paid to the diet and the general hygienic conditions.

SURGERY OF THE NERVES.

The surgical treatment of diseases and injuries of the nerves concerns the various forms of neuralgia, such as tic douloureux, trifacial or trigeminal neuralgia, sciatica, the neuralgia of stumps and scars, local spasms, such as facial or spinal accessory spasm, tumors of the nerves, and finally wounds of the nerves.

The various forms of neuralgia, after all medical means have been exhausted, are treated surgically, either by nerve-stretching (neurectasy), by division of the nerves (neurotomy), or by exsection of the nerves (neurectomy). The methods of doing these various operations will be described later (p. 996).

TUMORS OF NERVES, OR NEUROMATA. These are not uncommon in stumps, inasmuch as the proximal end of a divided nerve generally undergoes a bulbous enlargement, which more commonly is a false neuroma, *i. e.*, it has no true nerve tubules, but consists generally of connective tissue. In a few cases, however, as Bowlby has shown, true nerve-fibres are found. Neuromata also occur in the continuity of nerves, and are usually made up of fibrous tissue, though they may be sarcomatous, carcinomatous, myxomatous, etc. (See page 740.) More commonly they are single, but occasionally they are multiple.

Should such neuromata produce no pain or other annoying symptoms, it is best not to interfere with them; but if they give rise to any serious pain, the tumor with a portion of the nerve in which it is developed should be removed. In a stump this can usually be done by dissecting down to them, generally best by an elliptical incision including the scar of the amputation, and exsecting the tissues, especially the nerves, at a point above their bulbous ends.

If the tumor exists in the continuity of the nerve it should be exsected, and the various procedures described under Nerve-suture or Nerve-grafting, should be employed (pp. 996 and 997).

PAINFUL SUBCUTANEOUS TUBERCLE. One peculiar form of very small tumor, usually a fibroma, is connected occasionally with subcutaneous nerves, and is known by the name of painful subcutaneous tubercle. From its excessive tenderness and pain, it is a source of great annoyance. The treatment is excision of the tumor, and a part of the nerve. The resulting anæsthesia is of small moment, and disappears with time.

WOUNDS OF NERVES. In every case in which a nerve is divided it cannot be too strongly insisted upon that nerve suture should be done *immediately*. By so doing, the nerve will be re-established in its integrity far better than by any secondary operation. The methods by which it is done are described under Nerve-suture (see below) and Nerve-grafting (p. 997). The tendons, which are frequently divided by the same accident, should also be sutured immediately, thus restoring the continuity of each muscle.

OPERATIONS ON NERVES.

I. *Nerve Suture.* This may be either primary or secondary. By primary suture is meant suture of the nerve at the same time when the wound itself is treated surgically, or very shortly afterward. If this is not done, the ends of the nerves are not only separated from each other, but may become attached to tendons, connective tissue or other structures, and are only found and dissected out with difficulty at a later operation. By secondary suture is meant dissection and suturing of the nerve at a period more or less remote from the primary injury. Primary suture is always to be preferred, if it is at all possible.

(*a*) *Primary Suture.* If the nerve is merely divided without any loss of substance, the ends should be approximated by two or three sutures passed not merely through the sheath of the nerve, but through its substance. The suturing material is best of fine silk or other absolutely aseptic and somewhat durable material. The limb is then to be placed on a splint at such an angle as to relax the nerve. Especial attention should be paid to the aseptic condition of the parts, as suppuration around the sutured ends may wholly defeat the object for which the operation has been done. If the ends are separated to any distance, one or both ends of the nerve should be stretched until they can be placed in contact and sutured. The results of primary suture have been very gratifying. In a few instances the function of the nerve seems to have been restored very quickly, but commonly a few weeks, and, possibly, even a few months elapse before the nerve resumes its function. In 81 cases of primary suture analyzed by Bowlby 32 were entirely successful, 34 were partially successful, and 14 were failures, the result in one not being recorded.

After-treatment. When the nerve has been sutured and the wound is healed, the after-treatment is of the utmost importance, and should be continued for months, or even for a year or more, before we despair of success. This after-treatment consists in the daily use of electricity, the faradic current if the muscles respond to that, or if they do not, the galvanic current until the faradic becomes practical. Massage, and the hot and cold douche, should be assiduously employed. The patient should also make a persistent attempt to use the muscles which have been paralyzed. For sensation the use of the electric brush to the well-dried skin will often be of service.

(*b*) *Secondary Suture.* Months and even years after an injury to the nerve, secondary suture has been followed so often by success that it should always be attempted. The nerve having been exposed, the two ends are to be loosened from their attachments. The proximal end, as a rule, is bulbous, and can be found with ease, whereas, the distal end is often found with considerable difficulty. When the latter cannot readily be found the nerve should be cut down upon beyond the site of the injury, where it lies in its normal relations, when the trunk can be followed up to the lower divided extremity.

The whole of the bulbous extremity of the proximal end should be removed, but only about a quarter of an inch of the distal end. The two ends should then be approximated by stretching, and sutured as above described. The later treatment is identical with that which should follow primary suture. In seventy-three cases of secondary suture, Bowlby's tables give as a result thirty-two successes, twenty-six partial successes, and fifteen failures.

II. *Nerve-grafting.* If the ends of the nerve after stretching cannot be brought into apposition, three different methods may be adopted.

(*a*) The nerve may be split a suitable distance above its end, the flap turned down and sutured to the distal end. The results of this method of treatment have not been very successful.

(*b*) A suitable nerve may be removed either from one of the lower animals or from a limb which has been amputated from the human subject at the time that the nerve-grafting is to be done, and a portion of the nerve transplanted. Whether the nerve is sensory, motor, or mixed seems to make no special difference. The results from this method also have been partially satisfactory; but in only a few cases has the method been tried.

(*c*) The gap between two ends of the nerve may be bridged by a few strands of catgut, either applied alone or in a cylinder of decalcified bone, in the hope that these will form a scaffolding on which the reconstruction of the nerves will take place. The results in the few reported cases have also been fairly satisfactory.

III. *Neurectasy or Nerve-stretching.* This operation was introduced by Nussbaum in 1872, and has given excellent results in a certain number of cases. It has been tried, but seems to have done little good, in ataxia, in paralysis agitans, epilepsy, tetanus, etc. In tic convulsif or spasm of the facial muscles, the facial nerve has been stretched in over a score of cases. The relief has usually continued over a few months, and in one case (Southam's) for five years. As a rule, however, relapse will follow, but the relief, even if temporary, justifies the operation. In a few cases of wry-neck or torticollis neurectasy of the spinal accessory nerve has been done with benefit, or even cure. In anæsthetic leprosy, also, neurectasy has given good results. Inasmuch as it is a less serious and less disabling operation than neurectomy, as a rule, it would be desirable to perform neurectasy before neurectomy is done. There are two modes of doing the operation. First, by the bloodless method, *i. e.*, without a cutting operation. This is only applicable to the great sciatic nerve. The patient being etherized, and the leg kept extended at the knee, the entire lower extremity is carried into forced flexion at the hip-joint. In a few cases this method has given good results. In adults it must be attended by considerable rupture of the bellies of the hamstring muscles. Two deaths have been reported. It is not, therefore, wholly without danger.

Secondly, by operation. The trunk of the nerve to be stretched is exposed and loosened from the surrounding parts. It may then be hooked up by the fingers, or, in a small nerve like the seventh, by an ordinary pocket button-hook or similar instrument, or, better for the larger nerves, by Horsley's saddle-shaped nerve hooks. The amount of force which can be applied to the different nerves varies of course. The following table from Marshall gives the breaking strain, in pounds, of human nerves as determined post mortem. Probably it would be somewhat greater in the living than in the dead subject.

The facial nerve, which does not appear in Marshall's table, will bear a strain of about 5 to 12 pounds, the latter being more than the weight of the head.

COHESION OF HUMAN NERVES AFTER DEATH, BREAKING STRAIN IN POUNDS.

Supraorbital	6	pounds.
Infraorbital	12	"
Mental	5½	"
Brachial plexus	50-64	"
Ulnar	58	"
Musculo-spiral	61	"
Median	84	"
Crural	83	"
Internal popliteal	114	"
Great Sciatic—Symington	86-176	"
" " Tillaux	118-127	"
" " Gillette	165	"
" " Trombetta	82-288	"
" " Ceccherelli	154-220	"

The great sciatic nerve, which is frequently stretched, has so high a breaking strain that it is generally a safe rule to lift the leg and pelvis by traction upon it. In a number of cases in which I have done it by means of a spring balance I have found that 25–30 pounds will nearly lift the pelvis from the table. Eleven deaths from lesions of the spinal cord have been recorded after nerve-stretching. As a rule, however, it is a very harmless operation. If it is done for pain, the nerve should be preferably drawn from the spine, if for spasm, the traction should be toward the spine.

IV. *Neurotomy and Neurectomy.* Neurotomy is now rarely done, inasmuch as its effect is very fugitive. As a rule, it is much better to do neurectomy. In cases where several nerves lie close together, as in the armpit, in order to exclude any doubt as to the proper nerve, the faradic battery should be resorted to. Neurectomy may be performed on the trunk or on the roots of nerves, or in certain cases, especially the fifth nerve, by the removal of the sensory ganglion. (Page 978.)

The various operations which have been above alluded to may be applied to any of the principal nerves of the body. The mode of access to each nerve will now be described, it being understood that the process is practically the same whether the nerve is to be sutured, grafted, stretched, divided, or resected.

SUPRAORBITAL NERVE. The point of emergence is through the supraorbital foramen or notch at the junction of the inner and middle thirds of the eyebrow. A curvilinear incision, about one and a half inches in length, in the eyebrow, so that it will be hidden by the hair, enables us to find the upper border of the orbit. The tissues should be now drawn upward and the nerve discovered in the orbit.

The *supratrochlear nerve,* lying at the inner end of the eyebrow, can be reached at the same time and by the same incision prolonged nearly to the nose.

THE INFRAORBITAL NERVE. A line from the supraorbital notch to the space between the two lower bicuspid teeth intersects the infraorbital and mental foramina from which the infraorbital and the mental nerves escape. The infraorbital nerve passes through the infraorbital canal and foramen. A curved incision one and a half inches long is made just below the inferior border of the orbit. The nerve lies deep, next the bone, under the levator labii superioris. The nerve having been found, a silk thread is passed under it for identification and traction. Having raised the upper border of the incision by a retractor, the periosteum covering the floor of the orbit is lifted by Allis' blunt dissector or other similar instrument, and held out of the way by a flat retractor. Too much traction is to be avoided lest the eye itself be injured. The canal for the nerve can be readily found in the floor of the orbit and broken in by pressure with a grooved director or the Allis' blunt dissector. By a small hook the nerve is lifted up from its bed and divided toward the back of the orbit by means of curved scissors. The entire nerve is then drawn out by traction on the anterior end. The bleeding is readily arrested by packing with a little gauze.

REMOVAL OF MECKEL'S GANGLION. This is done for tic douloureux. There are several methods of reaching the ganglion. I shall only mention one, Chavase's modification of Carnochan's method. A T-incision is made on the cheek, the horizontal portion extending from just below one canthus to a similar point below the other. The vertical portion of the incision reaches nearly to the mouth, but it should not enter the cavity of the mouth. The infra-orbital nerve being found as before, the anterior wall of the antrum is trephined by a ¾-inch trephine, or opened by a chisel, including the infra-orbital foramen, care being taken not to divide the nerve. The posterior wall of the antrum is then trephined by a ½-inch trephine or opened by a chisel, care being taken not to wound the internal maxillary artery just behind it. After breaking through the groove in the floor of the orbit the nerve is drawn downward and traced into the spheno-maxillary fossa. It is then to be divided close to the foramen rotundum, and Meckel's ganglion is to be isolated if possible; if not, the nerve with its branches and the ganglion are drawn out. The wound is then lightly packed with iodoform gauze. It need not be sutured until the gauze is removed, forty-eight hours later. No pain is caused, of course, by these sutures, the parts being now anæsthetic. A forehead electric light or a forehead mirror is of great service in illuminating the deep parts of the wound. Care should be taken to inflict no harm on the eyeball. In one of my cases the retina was detached by an effusion of blood, although the operation was conducted with the greatest care, and I have since seen a second case in which sight was destroyed.

THE INFERIOR DENTAL NERVE. Of the numerous methods for the resection of this nerve the best is as follows: An incision is made just underneath the lower border of the jaw from the angle nearly to the chin. This position is chosen in order subsequently to hide the scar. The upper border of the wound is then drawn upward and the bone is entirely exposed. A half-inch trephine is then applied one and a quarter inches above the angle of the jaw. This will disclose the nerve at its entrance into the inferior dental foramen. The dental nerve making its exit at the dental foramen is then exposed by blunt dissection, and a similar small trephine-opening is made just posterior to the foramen. Care must be taken not to go so deeply as to divide the nerve by the trephine or to penetrate the bone. If necessary, a third button may be removed half way between the two, or the outer border of the entire canal is chiselled away, the chisel being guided by the nerve. The nerve is now lifted from its bed and removed. It is often best to fill the two ends of the canal either by dental paste or by gold foil, so as to prevent any possible reunion. The hemorrhage from the inferior dental artery is usually easily controlled by packing and hot water; sometimes it will require to be ligated.

REMOVAL OF THE GASSERIAN GANGLION. (See page 978.)

RESECTION OF BOTH THE SECOND AND THIRD DIVISIONS OF THE FIFTH NERVE. Mixter, of Boston, has performed the following operation: A curved incision is made through the region of the temporal muscle, beginning and ending over the zygoma, which may be previously drilled for later wiring. The zygoma is sawn through at each end, care being taken not to involve the articulation, as would be done by going behind the tubercle. The temporal and pterygoid muscles being turned down with the zygoma, both nerves can be reached at the foramen rotundum and foramen ovale by blunt dissection.

AURICULO-TEMPORAL NERVE. This nerve lies immediately behind the temporal artery just in front of the ear. A slightly oblique incision discloses the artery, and back of it the nerve.

LINGUAL NERVE. This nerve is occasionally resected for the relief of pain in cancer of the tongue. The tongue is drawn forcibly toward the opposite side by means of a suture passed through its substance. This makes the lingual nerve tense, and it can generally be felt as a firm band beneath the mucous membrane in the floor of the mouth. An incision is made over it and the nerve secured by a hook. If the tongue is fixed by the growth the nerve may be exposed where it lies in contact with the lower jawbone just under the mucous membrane below the first molar tooth.

THE FACIAL NERVE. This nerve is occasionally stretched or resected in case of facial spasm or tic convulsif. The best method is that of Baum, as the scar is hidden by the ear, and the nerve is more readily found than by Hüter's method.

Baum's method. An incision is made behind the ear two and a half inches long. The posterior border of the parotid gland is first disclosed. Drawing this forward the shining aponeurosis of the sterno-cleido-mastoid at its insertion on the mastoid process is next seen. A blunt dissection is then made in the interspace between these two landmarks, when the prevertebral muscles and their fascial covering will be seen at a depth of one to one and a half inches. The nerve crosses the narrow interspace between the mastoid and vertical ramus of the jaw external to this fascia. A very weak electric current will disclose the exact point where the nerve crosses this space, but usually it is readily found without this. The wound should be very dry; a sponge electrode is placed on any indifferent point. To the other conductor a piece of disinfected copper wire is attached, and the tissues are touched at various points. An electric light is of great advantage.

Hüter's method. A vertical incision is made in front of the ear. The parotid gland, in which the nerve divides into its main branches, is exposed and one of the branches sought for. Any one of the latter followed backward will disclose the main nerve.

THE SPINAL ACCESSORY NERVE. This nerve has been operated on for spasmodic wryneck by stretching or excision. It enters the sterno-cleido-mastoid muscle on its inner surface from one to two inches below the lobe of the ear, and after leaving the muscle at its outer border passes to the trapezius. It may be reached by an incision along the anterior or posterior borders of the sterno-cleido-mastoid muscle.

Anterior Operation. An incision is made along the anterior border of the muscle from the lobule of the ear two to three inches downward. The muscle being exposed and everted, the nerve is discovered where it enters the muscle a little above the level of the hyoid bone. In a stout person this depth may be very considerable.

Posterior Operation. An incision two to three inches long is made along the posterior border of the muscle, the centre of the incision corresponding to the centre of the muscle lengthwise. The posterior border of the muscle is then turned inward, and the nerve which lies a little above the centre of the wound is traced to its point of emergency from the muscle and exsected.

RESECTION OF THE POSTERIOR CERVICAL NERVES. The posterior branches of the first three cervical nerves can be resected for spasmodic wryneck, involving not only the sterno-cleido-mastoid and trapezius, but also the posterior cervical muscles by a method which I described in 1891 and published in the *Annals of Surgery* for 1891, vol. xiii. p. 44. The first case in which I had the opportunity of doing the operation (one of Dr. Dercum's) was May 3, 1889. Since then I have performed the operation in three additional cases. Noble, Smith, Powers, Gardner, and others have also done the operation several times. The result in three of my four cases has been very good.

The nerves to be divided are the external branches of the posterior divisions of the second and third cervical nerves supplying the splenius; the sub-occipital from the first cervical supplying the rectus capitis posticus major, and a branch from the second cervical which, with the sub-occipital, supplies the obliquus inferior. The wound is always a deep one, especially in a patient with a very thick neck, and the operation is much facilitated by an electric forehead lamp. The steps of the operation are as follows:

1. After the usual shaving and disinfection, a transverse incision 2½ to 3 inches long is made, half an inch below the level of the lobule of the ear. This incision should be on the same side toward which the chin is turned, since the posterior muscles draw the head to their own side, in marked contrast to the sterno-cleido mastoid, which rotates the chin to the opposite side. If, therefore, the right spinal accessory nerve has been divided the posterior nerves of the left side should be divided, and *vice versa.* Noble Smith has substituted, he thinks with advantage, a vertical incision for this transverse one.

2. The trapezius is divided in line with the first transvere incision.

3. The trapezius is dissected on its anterior surface in order to find the occipitalis major nerve as it emerges from the complexus and enters the trapezius at a point between the intra-muscular aponeurosis of the complexus and the middle line about half an inch below the incision. This is a large nerve, the size of a stout piece of catgut, and is readily found.

4. The complexus is divided transversely at the level of the nerve by repeated small cuts, so made as not to divide the nerve, which is the guide. The nerve can then be dissected down to the point where it arises from the posterior division of the second cervical. The posterior division of the second cervical is then exsected at a point on the spinal side of the origin of the occipitalis major, so as to catch also the filament to the inferior oblique muscle.

5. Dissect upward toward the occiput and find the sub-occipital nerve, a nerve somewhat larger than the occipitalis major. The nerve passes immediately below the border of the inferior oblique muscle, running from the spinous process of the axis and passing almost horizontally outward, to be inserted into the transverse process of the atlas.

6. Recognize the sub-occipital triangle formed by the inferior oblique, the superior oblique, and the rectus captis posticus major. The sub-occipital nerve lies in this triangle close to the occiput, and is there to be exsected. Should there be any difficulty, as I found in one case, from the fact that the triangle was completely blocked up with some large veins, the nerve may be drawn out at the lower border of the inferior oblique and divided there.

7. An inch lower down than the occipitalis major, and under the complexus, is the external branch of the posterior division of the third cervical, supplying the splenius. This is a much smaller nerve than the other two, and is sometimes difficult to find, or it may be divided by careless dissection.

It should be exsected close to the bifurcation of the posterior division.

A drainage-tube is to be inserted in the wound, and the wound closed as usual by buried and superficial sutures. The drainage in the dorsal position is all that could be desired.

CERVICAL PLEXUS. This plexus is reached by an incision along the middle of the posterior border of the sterno-cleido-mastoid muscle in the interval between it and the trapezius. An incision having been made through the skin, superficial and deep fascia, the nerves are found by a blunt dissection, and such of them operated upon as is necessary. It should be remembered that the phrenic nerve arises from the third and fourth divisions, with a branch from the fifth. Stretching of these particular nerves, therefore, should be done with care; but they may be resected beyond the origin of the phrenic without difficulty.

BRACHIAL PLEXUS. This plexus is easily exposed just above the clavicle by a horizontal incision parallel to the clavicle and one inch above it. The skin, superficial and deep fascia are divided when a blunt dissection will readily expose the nerves. If it is desired either in the branchial or cervical plexus to differentiate the cords, it can be done by a battery. Care should be taken in operating on the brachial plexus to see that none of the cords are overlooked. The inner cord lies immediately external to the subclavian artery, and can be identified by this relation. It can be differentiated from the artery by the effect of pressure on the pulse.

AURICULARIS MAGNUS NERVE. The auricularis magnus emerges from behind the sterno-cleido-mastoid at the middle of its posterior border. An incision along the posterior border at its middle discloses the nerve passing upward toward the ear.

OCCIPITALIS MINOR NERVE. This also emerges from behind the posterior border of the sterno-cleido-mastoid a little above the middle of the muscle. An incision along the posterior border of the muscle somewhat above its middle, followed by a little blunt dissection, will show the nerve.

OCCIPITALIS MAJOR NERVE. This emerges from the trapezius near its outer border just below its insertion into the superior curved line of the occipital bone. The hair from the lower occiput should be shaved. A horizontal incision made near the centre of the inner border of the attachment to the trapezius muscle and just below the superior curved line will disclose the nerve. The occipital artery lies very near it externally.

INTRA-SPINAL DIVISION OF THE POSTERIOR OR SENSORY NERVE-ROOTS FOR INVETERATE NEURALGIA. In 1889 and again in 1890 and 1895 (*Annals of Surgery*, Jan., 1895) Abbe reported three cases in which he did this operation for neuralgia of the arm. Bennett in 1889 (*Med.-Chir. Trans.*, 1889, lxxii. p. 329) reported a fourth case. Bennett's case seems to have been relieved of his pain, but died suddenly on the eleventh day from cerebral apoplexy. Horsley (*Brit. Med. Journ.*, 1890, ii. p. 1289) has reported two cases, and McCosh (*Annals of Surgery*, Jan., 1895) has reported a seventh. All of the cases have been relieved, but apparently not absolutely cured. The operation, if done at all, should, I think, be done early rather than after other operations, especially amputation, inasmuch as it does not affect the motor roots of the nerves, and therefore if it relieves the pain it will leave a useful extremity. Often, however, patients will have insisted on other prior operations, including amputation. The operation is done as follows. A vertical incision is made at the desired point, and the posterior arches of the selected cervical or other vertebræ are removed, thus exposing the dura. This is then divided and held to one side by an aneurism needle or other similar retractor. The posterior nerve-roots are then identified. In order to determine which roots we are dealing with, it is always well to mark on the skin by a slight incision the level of a definite spine, for instance, that of the *vertebra prominens* or other vertebræ. This will enable us to identify exactly the nerve or nerves the roots of which are to be removed. The posterior roots having been exsected, the dura is sutured and the wound closed and treated as usual. The operation, of course, is serious, but not especially dangerous. None of the seven cases have died from the operation.

MEDIAN NERVE. This nerve can be reached in the axilla, the arm, the forearm, or the hand.

1. *The Axillary Operation.* A two-inch incision is made at the junction

of the anterior and middle thirds of the axilla, dividing the skin, superficial and deep fasciæ. The axillary nerves and artery are found by a blunt dissection. The median nerve is easily recognized either by the battery, which will cause the flexors and pronators of the hand to contract, or by the fact that it arises by two heads astride the axillary artery.

2. *The Operation in the Arm.* An incision is made at the inner border of the biceps at its middle. The nerve and the brachial artery are found just under the deep fascia. The nerve ordinarily crosses in front of the artery from without inward; occasionally, however, it passes behind it. The battery, if need be, may be used to determine whether it is the median nerve.

3. *Operation of the Forearm.* The nerve can readily be reached just above the wrist-joint by an incision two inches long at the inner border of the tendon of the palmaris longus. It lies immediately under the deep fascia.

4. *Operation in the Hand.* The branches to the thumb, fore- and middle-fingers may be reached by an incision along the internal border of the thenar eminence. The skin and superficial fascia being divided, the palmar fascia is disclosed. The nerve lies just under the edge of this latter fascia.

THE ULNAR NERVE. This nerve can be reached in the axilla, the arm, or the forearm.

1. *The Axillary Operation.* The same incision as that for the median nerve will disclose the ulnar, which is a large nerve lying to the inside of the median. The internal cutaneous usually lies between the median and the ulnar. The battery will differentiate it readily from the cutaneous nerve, and will cause the deep ulnar flexors and interosseous muscles to contract, thus flexing the fingers at the knuckles and extending them beyond the knuckles.

2. *Operation in the Arm.* A two-inch incision is made at the inner border of the biceps, but slightly further back than that for finding the median nerve. The nerve lies under the deep fascia posterior to the artery and the median nerve.

The ulnar nerve may also be exposed in the groove between the external condyle and the olecranon, but it is undesirable to cut away the tense fibres which hold the nerve to its groove. A few of the upper fibres may be divided if it is necessary, as I have had to do in one instance.

3. *Operation in the Forearm.* Just above the wrist the nerve can be found by an incision on the radial border of the tendon of flexor carpi ulnaris. The nerve at this point, it should be remembered, lies under a second layer of the deep fascia to the ulnar side of the ulnar artery.

MUSCULO-SPIRAL NERVE. This is best found by a nearly vertical incision three inches long along the outer border of the arm, beginning a little below the insertion of the deltoid. The nerve is found in the groove between the brachialis anticus and the supinator longus, or a little higher up between the internal and external heads of the triceps muscle. The deep fascia having been incised, the appropriate muscles are separated and the nerve disclosed. In the former position the branch to the supinator longus may first be encountered. A little deeper dissection following this branch down to the musculo-spiral groove of the bone will disclose the main trunk in the muscular interspace.

THE RADIAL NERVE. This is a branch of the musculo-spiral, and is easily found by a longitudinal incision along the outer border of the forearm, three inches above the wrist-joint, just as the nerve passes under the tendon of the supinator longus to the posterior surface of the forearm. The skin, superficial and deep fascia are divided, disclosing the tendon of the supinator longus, which is the guide to the nerve.

ANTERIOR CRURAL NERVE. The anterior crural nerve passes from the abdomen on to the thigh, underneath Poupart's ligament, in the groove between the psoas magnus and the iliacus, as they also emerge from the pelvis under Poupart's ligament. It lies at a slight distance external to the femoral artery. An incision just below Poupart's ligament, and parallel with it, the centre of the incision being a little external to the artery, will disclose the nerve (by a blunt dissection) just under the deep fascia. Of course, care should be taken in the inner part of the incision not to wound either the artery or the vein.

EXTERNAL CUTANEOUS NERVE. The external cutaneous nerve emerges from the abdomen, under Poupart's ligament, just internal to the anterior superior spine. An incision parallel with Poupart's ligament and just below it, the centre of which is slightly internal to the anterior superior spine, will disclose the nerve to the inner side of the tendon of origin of the sartorius muscle and just under the deep fascia of the thigh.

THE GREAT SCIATIC NERVE. The patient is placed upon the abdomen; an incision is made from three to four inches long in the middle line of the thigh, beginning at the gluteo-femoral crease. At the upper part of the incision the lower fibres of the glutæus maximus muscle will be found, and may be recognized by their almost transverse direction. Just below these fibres the bellies of the hamstring muscles run, the biceps being external. Tearing through the connective tissue at its outer border, by the finger, the great sciatic nerve can be readily found at a depth of about two inches.

THE POPLITEAL NERVES. The popliteal space is a lozenge formed by two long arms above, the semitendinosus to the inside and the biceps to the outside and by two short arms below, the two bellies of the gastrocnemius below. Near the upper angle of the lozenge the great sciatic divides into the external and internal popliteal nerves. The internal popliteal runs in the axis of the popliteal space from the upper to the lower angle. It lies immediately below the deep fascia. Immediately beneath it (in the prone position) lies the popliteal vein, and still further down, next the bone, lies the artery. The external popliteal nerve lies along the inner border of the biceps tendon, just underneath the deep fascia. The relation of these nerves is described, not so much because they are frequently operated on, but because in tenotomy of the hamstring tendons they are to be carefully avoided. The external popliteal lying so near the biceps is especially liable to be divided, unless the surgeon carefully remembers its position. Its division paralyzes the anterior muscles of the leg, causing foot-drop.

THE ANTERIOR TIBIAL NERVE. An incision three inches long is made at a slight angle to the course of the anterior tibial artery, which is in a line drawn from the mid-point between the external borders of the tubercle of the tibia and the head of the fibula, to a point midway between the two malleoli. The deep fascia having been exposed, the white line between the tibialis anticus and the extensor communis digitorium is found, and the fascia divided in this line. The connective tissue in the interspace between these two muscles is then loosened by the finger or a blunt dissector. Hidden toward the bottom of the interspace will be found the beginning of the extensor longus pollicis. The anterior tibial nerve lies to the fibular side of its artery, deep in the interspace, between the anterior tibial and extensor longus pollicis muscles.

The nerve may also be found just above the ankle by an incision in the middle line of the leg; the deep fascia having been divided, the tendon of the extensor longus pollicis is found. The nerve lies to the outer border of this tendon, between it and the tendons of the extensor longus digitorum.

POSTERIOR TIBIAL NERVE. By an incision three to four inches long at the inner border of the tibia, the skin, superficial and deep fascia, and the fibres of the soleus muscle arising from the tibia are divided. A second layer of the deep fascia is then disclosed, covering in the tibialis posticus muscle. The posterior tibial nerve lies underneath this fascia to the fibular side of the posterior tibial artery. The fascia is divided, the artery found, and by it as a guide the posterior tibial nerve.

MUSCULO-CUTANEOUS NERVE. The musculo-cutaneous nerve is a branch of the external popliteal, which pierces the deep fascia from one-half to two-thirds of the way down from the knee to the ankle. An incision obliquely to its course discloses the nerve in the superficial fascia.

INTERNAL SAPHENOUS NERVE. The internal saphenous nerve pierces the deep fascia to the inside of the knee in the interval between the tendons of the sartorius and gracillis, and then descends along the inner side of the leg just behind the internal border of the tibia. A slightly oblique incision at any part of its course below the knee will disclose the nerve in the superficial fascia.

EXTERNAL SAPHENOUS NERVE. This is a branch from the internal popliteal nerve, and pierces the deep fascia at the upper part of the calf. It then descends toward the outer side of the ankle-joint. An incision somewhat oblique to its course will reveal it in the superficial fascia.

PUDIC NERVE. The pudic nerve accompanies the pudic vessels upward and forward along the outer wall of the ischio-rectal fossa, and under the protection of the tuberosity and ramus of the ischium. It lies between the two layers of the obturator fascia. An incision in the perineum just to the inner border of the tuberosity of the ischium, and a blunt dissection toward the obturator fascia, with division of its outer layer, will disclose the vessels and nerve which lie about one and a quarter inches above the lower border of the tuberosity.

THE DIGITAL NERVES. The digital nerves of the fingers and toes run in a line corresponding to the junction of the palmar and lateral aspects upon each side of the fingers. Those to the thumb emerge from the palm at the border of the thenar eminence near the base of the first phalanx. All of them can be reached best by a lateral flap.

SURGICAL TREATMENT OF CONTRACTURES.

Most of these cases heretofore have been left untreated surgically, under the mistaken notion that no good could be done. But of late the surgeon has interfered, to the great advantage and often to the entire cure of the patient.

In cases in which the joints have become immobilized from rheumatic, gouty, or other forms of arthritis, if the case is seen early enough, the adhesions should be broken up under ether, repeatedly, if necessary, care being taken, of course, not to apply such a degree of force as will either be followed by serious inflammation of the joint, or fracture of the bone. Subsequent massage, Swedish movements, hot and cold douches, and electricity will often accomplish a great deal. If seen late, when firm anchylosis is established, it is doubtful whether in many cases any permanent good will result from such treatment, and I have seen very serious results follow, even to the point of imperilling life.

It is, however, in the muscular contractures proper following nervous lesions that I have been able to accomplish very much of late. Tenotomy with forced flexion and extension and persistent suitable after-treatment will sometimes restore even almost apparently helpless limbs to normal or nearly normal use. In contractures after cerebral lesions, attended with athetoid movements, it is doubtful whether any good result can be obtained, as, even if the limb is straightened, there is no such control of it as to make it useful. In spastic contractures of the arms or legs there is no such voluntary control of the muscles as to make us hopeful of benefit. But in hysterical contractures I have obtained unexpectedly good results, so that I am greatly encouraged, even in the most severe cases. In order to obtain the best results this tenotomy will generally have to be very extensive and thorough; all the contractured parts, whether they be tendon, contractured fascia or fibrous tissue, must be divided, even down to the bone. I have entirely given up for several years any attempt to treat such cases by subcutaneous division. Modern surgery enables us to effect the widest division of such parts by open wounds, with no resulting evils. Recently, in two cases kindly referred to me by Dr. S. Weir Mitchell, in which both thighs were flexed at a right angle on the trunk and strongly adducted against each other and both knees so flexed that the heels touched the buttocks, I divided the soft parts immediately below the anterior superior spine of the ilium, and the adductors on the upper inner aspect of the thigh, both down to the bone, by a wide open incision, and in the ham I divided the hamstring tendons, the popliteal fascia, and soft tissues, down to the bone, in all three places being watchful to avoid injury to the important nerves and bloodvessels. Forced extension at the hip and knee enabled me to overcome about two-thirds of the contracture, in doing which, of course, the wounds gaped open very widely. They were closed as far as possible by suture, and where this was impossible were packed with iodoform gauze. No reaction followed, and the operative gaps were gradually filled up by granulation tissue. The remaining third of the contracture was then overcome by the persistent use of splints and traction by means of weights and pulleys. This later use of splints, weight and pulley, with massage, electricity, and Swedish movements, is a most important adjuvant to the operative treatment, and must never be neglected. Several weeks, or even months, may be necessary to attain the end in view. In another, even more remarkable case, in which from excessive contracture the knee had been bent at a right angle for a long time, the patient insisted upon either a straight leg or amputation. After extensive tenotomy of all the structures in the ham, excepting the vessels and nerves, during the attempt to straighten the leg the posterior ligaments of the knee-joint suddenly gave way so widely that I could thrust three fingers into the knee-joint. I debated for some moments whether I should not amputate at once above the knee, but my confidence in modern antiseptic methods was such that I resolved to attempt the preservation of the leg. The wound was stuffed with iodoform gauze and placed upon a splint. The man got well without the slightest reaction, the wound filled up with granulation tissue, and I was gratified by seeing him go out of the hospital with a useful leg, though anchylosed in the straight position.

In some cases, instead of such simple tenotomy, the tendons may be lengthened or shortened by a definite amount, as I have described in a paper in the *Transactions of the College of Physicians*, Phila., 1891. The same prolonged after-treatment described above must follow the operation if any good is to be obtained.

CHAPTER XXXIV.

NEURO-ELECTROTHERAPEUTICS.

By GEORGE W. JACOBY, M.D.

ELECTROTHERAPY designates that part of therapeutics in which the electric current, galvanic, faradic, or static, is employed in the treatment of disease. Electrolysis and galvano-cautery, although properly included in this definition, will not be referred to here, and this chapter will be still further curtailed by limiting it to a consideration of the use of electricity in diseases of the nervous system. For this and other reasons the following chapter can make no claim to completeness. Much that may be looked for here will not be found, and for such information, as well as for the literature of the subject, the reader is referred to any of the large number of special treatises upon medical electricity which are in existence.

History. The somewhat mythical stories of the employment by the ancients of natural reservoirs of electricity, as bathing patients in water containing electric eels, can interest us only as historical curiosities. With the invention of the friction machine by von Guericke in 1663, the actual therapeutic employment of electricity was inaugurated, and for more than a century thereafter static electricity was alone employed. All that has been transmitted to us from that period is the claim of various electrotherapists of the eighteenth century (De Haven, Jallabert, Mauduyt, and others), that phenomenal successes had been attained by its use. The end of that century, bringing with it as it did the discoveries of Galvani and Volta, relegated to obscurity the applications of static electricity with their vaunted cures. Galvanism, in its turn, on account of the impractical apparatus which at that time was necessary for its production, and on account of its exaltation to a general panacea, was not able to maintain its position long. It was really not until Oersted discovered the force of magneto-electricity, and a few years later (1832), Faraday made his fundamental discovery of induced electricity, that, in consequence of the construction of the first induction coil, electrotheraphy escaped from the contumely with which it had, to a greater or less extent, been regarded. As the foremost representative of this new era, in fact, as the father of modern electro-therapeutics, we must regard Duchenne, of Boulogne. He it was who developed the method of localized electrization, and through his painstaking investigations gave a sound basis to our knowledge of electro-muscular contractility and electro-cutaneous sensation. Upon this foundation were built the works of Remak, Ziemssen, Brenner, and Erb. In 1881, at the International Congress in Paris, another impetus, one which may be looked upon as a turning point in its scientific career, was given to electrotherapy. This was the introduction of the absolute galvanometer. By this means it, for the first time, became possible to regulate the dosage of the current, and therefore to allow a certain predetermined amount of electricity to pass through a given part of the body.

Electrotherapy, in consequence of this and other scientific work, then constituted a science, *per se*, which by many was considered as finished and complete. Soon thereafter came the rejuvenation of static electricity in Paris. Its employment there, chiefly upon hysterical patients, coincided in point of time with the renewed attention bestowed upon hypnotism.

The instantaneous results obtained with both of these measures upon this class of patients could not fail to attract attention, and from this to the assumption that both agents acted through the same means, namely, by suggestion, was but one step. Furthermore, unscientific ultra-sanguinistic ideas had so obtruded themselves in the treatment of disease by electricity that its use was being advocated in nearly every disorder, no matter how slight, to which the nervous system was subject; many of its advocates, also, not only had no idea as to the mode of production of the cures supposed to have been obtained by its use, but were not even interested in seeking for such an explanation.

It may, therefore, be easily understood why observers scientifically schooled and accustomed to search for explanations of their observations should have generalized the deduction derived from the use of electricity in hysteria, and thus become ready recipients of the sweeping assertions which soon followed.

When, then, Moebius expressed the opinion that our knowledge of the nature of the curative action is *nil*, and that at least four-fifths of all electrical curative properties are of a pyschic character, it is not surprising that he soon received the support of men like Schultze, in Bonn; Bruns, in Hanover, and many others. The nihilistic tendencies which permeate medical therapeutics to the extent even that surgical operations have been accused of exerting their beneficial effects by means of suggestion have thus also included the action of electricity in their grasp, and its specific curative action has become the subject of one of the most debated questions of the day.

At present observers are divided into three camps, comprising those who admit that suggestion bears an important part in the production of cures by means of electricity, but who claim that the specific electrical action is equally, or, perhaps, more important; those who claim that suggestion as a factor does not enter the question at all; and lastly, those who claim that all that is achieved is done by means of suggestion, electricity itself has no more specific action than a magnet or a mustard plaster, and, instead of using electricity, you may just as well employ hypnotism in whatsoever form.

While it is very easy to make sweeping statements of this nature, it is an entirely different matter to disprove them.

It may as well be admitted here that our knowledge of the manner in which electricity acts upon the human organism is very elementary; that the various theories as to its electrolytic, cataphoric, and catalytic action are none of them proven, and that the therapeutic action of electricity may, as Erb has suggested, be due to an entirely different and uninvestigated series of phenomena, changes in assimilation and calorification, elementary affinities, osmotic processes, etc. No matter how indefinite this knowledge may be, we certainly have not yet arrived at a point where the assertion that all is hypnotism, all is suggestion, should be allowed to confuse and befog us. At the same time, the electrotherapy of to-day rests only upon an empirical basis, and until experimental investigations shall have shown us how electricity acts upon the living body, so long will we have to admit the existence of a certain strength in the position of the suggestionists. That suggestion plays its part in electro-therapeutics cannot be denied. Every capable physician recognizes the value of suggestion and makes use of it in some form or other. If, therefore, in electricity we possess a remedy which, in addition to specific action, is a potent vehicle for the employment of suggestion, all the more reason why it merits a prominent place in our therapeutic armamentarium. At the same time we must not deceive ourselves, and must endeavor to form in our mind a clear idea of the extent of this specific action and of the actual value of electricity in the treatment of disease. We must not be immoderate in our expectations, nor ask the impossible. That destruction of substance, degenerated nerve-fibres and ganglia cannot be restored by means of electricity should be very evident; but that we do possess in electricity a therapeutic measure which in many disorders of the nervous system produces as positive results as any other known remedy, is a fact which no unprejudiced observer can deny.

While we admit that our knowledge of the manner in which electricity acts as a remedial agent is very meagre, we must insist that only he will be able to obtain the best possible results from its use who possesses, in addition to thorough anatomical and physiological knowledge, a complete understanding of the physics and of the methods of the application of the electric current.

Three kinds of current are used for therapeutic purposes, constant or galvanic, faradic or induced, and frictional or static electricity. The ways of applying the constant current are known as the labile, stabile, and intermittent methods. In the stabile method the current is allowed to flow continuously and steadily; during the entire application the electrodes must be kept firmly applied; in the second method one electrode is fixed to a certain place, while the other is moved over, but not lifted from the body. The circuit is at no time actually broken, but the current strength necessarily changes according to the varying resistance of the skin and the pressure applied to the electrodes. The marked exciting action of this method is certainly not due to these variations in current strength, for these can with care be avoided, but is caused by the fact that the several parts are being successively exposed to the influence of the greatest current density, and thus are specially irritated.

In the interrupted method one pole is immovably fixed, while with the other the parts to be acted upon are repeatedly touched, or both electrodes are kept in contact with the body while interruptions are made in the metallic part of the circuit. Occasionally voltaic alternatives are used. These consist in the production of a reversal of the current in the fixed electrodes by making a pole change in the metallic circuit. Such interruptions and voltaic alternatives constitute a most decided method of nerve or muscle excitation, one which, in the severe forms of reaction of degeneration, is alone capable of producing any response to the current.

Two special methods of galvanization which are much employed are *central galvanization* and *galvanization of the sympathetic*, or, to employ a better term, subaural galvanization. The first consists in placing a large, flat electrode (kathode) upon the epigastrium, while the anode, formed by a large, round sponge-electrode, is placed upon the head and then moved along the spinal column. First the forehead is stroked from side to side with less than one milliampère of current; then the top of the head, the sides of the neck, and, finally, the spinal column are treated in the same way, except that over the spine a stronger current may be used. Thus, the entire cerebro-spinal axis is subjected to the action of the current. *Subaural galvanization* is carried out as follows: One pole, consisting of a medium-sized electrode, is applied to the angle of the lower jaw next to the hyoid bones and pressed backward and upward toward the spinal column. The other pole, a large electrode, is placed upon the opposite side of the neck next to the fifth, sixth, and seventh cervical spinous processes. Usually the kathode is placed at the first-described place. Weak, stabile currents, 2 to 5 milliampères, should be used and care taken not to cause any interruptions of the current or variations in its density. The duration of each application should also be short, lasting from one to three minutes.

Faradization may be divided into two categories, according to whether the skin or the underlying tissues are to be acted upon.

Faradization of the skin requires the use of a secondary coil wound with long, thin wire. The skin must be thoroughly dried and the exciting electrode, which should be connected with the negative pole of the secondary coil, must also be dry. For this purpose either a perfectly dry sponge-electrode or a wire brush should be employed. The positive pole is connected with a moist electrode and placed upon some non-muscular part of the body, as upon the sternum or nape of the neck; then, with the other dry electrode, the skin over the entire area which it is desired to excite is to be touched or brushed, thus producing a passage of sparks between the skin and the electrode.

If very sensitive parts are to be thus treated it may be better for the operator to make use of his own hand as an exciting electrode. By this means the painfulness of the current is constantly controlled and can easily be regulated by lessening or increasing the amount of contact between subject and operator. In this method the moist positive electrode is again placed upon an indifferent point, while a moist negative electrode is held tightly in one hand of the operator, who, with the back of the other hand, more or less gently strokes the parts to be acted upon.

For *faradization of muscles and nerves* a secondary coil wound with short, thick wire is employed. A large, well-moistened electrode is placed upon an indifferent point, and the other moist but smaller electrode is passed over the body of the muscle. In many muscles certain points will be found from which they can be more easily excited to contraction. These are the motor points and correspond to the entrance of the nerves into the muscles.

If not only a single muscle is to be acted upon but an entire muscular group made to contract, then *faradization of the nerve-trunks* is employed. The method is the same as that described for faradization of the muscles; the small electrode, however, being placed over the motor point of the nerve.

General faradization consists in passing one electrode over the whole or greater part of the body, the other being fastened at some indifferent part (sternum, sacrum, soles of the feet). Full stimulation and good contractions of the muscles are to be produced with as little pain as possible.

Galvano-faradization consists in uniting the secondary coil of the induction apparatus and the galvanic current into one circuit and applying both currents through one set of electrodes. The negative pole of the coil is connected with the positive pole of the galvanic current, and the electrodes are attached to the unconnected poles. A good rule to follow in the regulation of this current is to use the same amount of galvanic current as we would were we using this current alone, and then to regulate the faradic current according to the amount of skin or muscle action that we desire to produce. In this combined current we have a summation of the exciting action of the galvanic kathode to that of the faradic kathode, and thus the exciting action of certain current strengths is materially increased.

This may be the place to speak of another method of general electrization which certainly has its value and indications. I refer to the hydro-electric bath. According to which current is used we speak of a faradic, a galvanic, or a galvano-faradic bath. Two forms of hydro-electric baths may be used, the monopolar and the dipolar. In the monopolar bath the tub is connected with one pole of the battery directly if made of metal, indirectly by means of the water if of wood, while the other pole is attached to a metal rod which is suspended from above the tub; the free end of this rod is covered by a sponge or other material for the patient to take hold of.

If the tub is of metal, care must be taken by means of a net or other arrangement to prevent direct contact between the body of the patient and the tub. This form of bath is called monopolar, because the patient's body is surrounded by water charged with electricity from a single pole.

In the bipolar bath, both poles are placed in the tub, the simplest manner being to cover the metallic plates, which constitute the electrodes, with perforated wood or hard rubber, and then to place them in the water at the ends or at the sides of the tub, according to whether it is desired to have the current traverse the body longitudinally or transversely. Very complicated and expensive tubs have been constructed for the administration of electric baths, but the simple methods here described will answer in all cases.

Franklinization. For the application of the static or frictional current, the patient is placed upon an isolating platform and the machine then set in motion. Knowing which is the positive and which the negative pole ot the machine, the negative terminal is to be connected by means of a metallic conductor with the isolating platform, and the positive one is connected by means of a chain with an insulated electrode. The shape of this electrode determines the form of the current obtained, a ball electrode giving sparks, while a crown or pointed electrode produces a spray. Now the terminals of the machine which have thus far been in contact are widely separated so that no spark passes between them, and then the insulated electrode held by the operator is approached to the body of the patient, whose clothes need not have been removed.

In order to diminish the action of the current the negative pole, instead of being connected with the insulating platform, may be directly connected with the earth by means of any water or gas-pipe.

Therapeutically the various currents may be applied to the seat of disease if it is desired to obtain a direct influence, and at some distant point if indirect or reflex action is desired. This desire to obtain a direct or an indirect action of the current will not only govern our choice of the *place of application* of the electrodes, but will also, to a certain extent, determine the *size of the electrodes* to be used. Our choice of electrodes will, however, depend to a greater extent upon the dosage (density) of the current desired. As we will show presently, the smaller the electrode the greater is the density of a given current, and its action is directly proportionate to its density; therefore, in the majority of instances one large and one small electrode are chosen, the latter being placed over that part upon which it is desired to concentrate the action of the current; if a deeper lying organ is to be reached we must endeavor to include the organ between the electrodes, both of which should be of large size.

Quite as important as the determination of the size of the electrodes and their place of application is correct *current dosage.*

As regards dosage, galvanism possesses a distinct advantage over the other forms of current, since we are able by means of the absolute galvanometer, and by attention to the size of the electrodes, to regulate precisely the strength and the density of the current. The regulation of both strength and density of current is the fundamental factor of the dosage of electricity.

The size of the electrode determines the density of the current; therefore, to speak of current strength without giving the size of the electrodes employed has no value whatever, as a current of 10 milliampères strength passed through an electrode of large surface is a weak current, whereas the same strength of current passed through a small electrode may be very strong. In noting the strength and density of a given current it is well to make use of some practical formula which may be universally understood.

As current density is equal to the strength of the current divided by the diameter of the electrodes, we can describe the density by a fraction whose numerator is expressed by milliampères and whose denominator gives us the size of the exciting electrode.

If round electrodes are used it is convenient to follow the advice of Stintzing and note their diameter, while in the case of rectangular electrodes the two side-lengths are to be mentioned. If then we desire to express that in a given case a current of 6 milliampères has been applied through an exciting electrode, 12x6 c.cm. in size, the formula employed would be $D = \frac{6}{12x6}$. If to this the length of application is added, then every person will know precisely how much current the patient has received.

Being able to accurately determine the density of the current which is being administered, it might properly be asked what current density should be used in the treatment of disease? This is dependent upon so many varying factors that no definite answer can be given.

For general use a density of $\frac{1}{15}$ to $\frac{1}{20}$ (15 to 20 square centimetres of electrode for each milliampère of current) appears to be about correct, but currents of less or more density may naturally be indicated in individual cases. In galvanization of the brain the density should not be more than $\frac{1}{100}$, and in subaural galvanization not more than $\frac{1}{50}$ to $\frac{1}{30}$.

An attempt has been made to introduce infinitesimal doses in electrotherapy. as $\frac{0.10}{50}$ to $\frac{0.5}{50}$, in the treatment of peripheral paralysis, but proofs of such efficacy are totally insufficient to convince any but a strongly partisan observer.

The dosage of the current should be regulated in every galvanic application.

No absolute measure exists for the strength of the faradic current, and no practical apparatus for measuring it has yet been invented. All that we can do is to give the distance to which the secondary coil has been drawn out, and even then the results obtained from each coil are only comparable among themselves, as the length and thickness of the wire with which the coils are wound influence the action of current. We are thus enabled to speak of the strength of an induced current only in very vague terms, referring to it as weak, medium, or strong.

The dosage of static electricity is even a more difficult matter than that of the faradic, for, although we can recognize the tension of a current from the size of the spark, there is no practical way of estimating its strength (intensity.)

Other factors besides dosage must, of course, also receive consideration in electrotherapy. The choice of the proper current, the number of applications, as well as their duration, are important questions, which, however, can be specifically settled only for each individual case.

The *choice of the proper current* is not always easy, and frequently cannot be determined. In such case the selection will, to a great extent, have to be made empirically.

The *duration* of each application has been shortened with the introduction of the galvanic current into therapeutics; formerly applications lasting from a half to one hour were recommended, but to-day the consensus of opinion tends toward the opposite direction; applications to a single part are to occupy from one to ten minutes, while generally an entire treatment, embracing the application to various parts, should not exceed fifteen to twenty minutes.

The frequency of applications to be made will depend upon the character of the disease to be treated. Chronic cases will require treatment once or twice a week, while acute cases should be treated daily, or under exceptional circumstances even twice daily.

PRACTICAL POINTS FOR THE APPLICATION OF ELECTRICITY.

In addition to the necessary knowledge of the technique of electrical application, of the physics of electricity and of the anatomical relationship of the underlying parts to the surface of the body, every electrotherapist should be the possessor of an ample instrumentarium, and should always be sure that this apparatus is in thoroughly good working order. The necessary apparatus consists of:

1. An induction coil.
2. A galvanic battery of sufficient electromotive force (at least thirty cells), which is supplied with (*a*) a selector and (*b*) a pole-changer.
3. A rheostat.
4. A milliampèremeter.
5. Electrodes of a certain known surface.
6. Conducting cords.
7. An interrupting handle.

The certainty that the apparatus to be used is in good condition should be the first rule for every electro-therapeutical application. Everything should be carefully inspected each time it is used, and we must be sure that the current is turned off before the electrodes are applied to the body. The causation of pain, unnecessary excitation, current interruptions, or voltaic alternatives are to be studiously avoided. The electrodes must be thoroughly moistened, steadily and quickly applied, and the current very gradually introduced and removed. It is best to increase and decrease the strength of the current by means of the rheostat, and during the entire application the needle of the milliampèremeter should be carefully watched. The electrodes should be applied and removed only when the needle of the galvanometer indicates the absence of all current; one hand of the operator should always be upon the excitation electrode and the other upon the rheostat, the indifferent electrode having been given to the patient to hold, or, better still, fastened by means of any mechanical contrivance. In no other way can changes in the density of the current, especially in labile applications, where the resistance varies in the different parts over which the electrode is applied, be controlled. This gradual introduction and removal of the galvanic current is perhaps the most important point in the technique of its application, and I will even go so far as to say that he who has not completely mastered these procedures should not be intrusted with the application of galvanism. In electrotherapy every success depends upon the method and the manner in which the current is employed, and attention must be paid to a great many details which can be most surely and quickly mastered by personal experience. Applications to one's own body will quickly bring to light any defects which may exist in the apparatus, and will give a better knowledge of the variations of sensibility in the different parts of the body, of the situation of the motor points, of the amount of moisture and pressure to be used, and of the current action and current strength, than hours spent in the study of these questions.

In all applications it should never be forgotten that harm may be done by the use of electricity. Not alone that its employment is often positively contraindicated, or that dizziness, flashes of light, buzzing in the ears, cough, vomiting, drowsiness, and other occurrences due to faulty method, may be produced, but certain persons appear to have an idiosyncrasy against the use of electricity in whatsoever form it may be applied. Such persons react badly to even a minimum amount of current; another class of patients always react badly when the negative pole is applied to the head. This fact may be noted, not only in the use of the galvanic current, but also in applications of static electricity. Here a negative charge of the body will produce restlessness and disagreeable sensations, while a positive charge will calm and soothe them. This purely clinical experience has its analogue in the action of the constant current upon certain lower animal (electrotopism). Finally, it will be found that many hypochondriacs, neurasthenics, tremors of certain kinds, and vertigoes are badly influenced by electricity in any form.

A certain definite plan of treatment should be mapped out for each case and given a fair trial; if the results are unsatisfactory other methods may be employed, but a certain method should be adhered to for a length of time at least sufficient to clearly demonstrate its efficacy or uselessness.

THE THERAPEUTIC USE OF ELECTRICITY IN DISEASES OF THE CENTRAL NERVOUS SYSTEM.

There can be no doubt that we are able to reach the brain, and perhaps also the spinal cord, by electric currents, notwithstanding their bony capsules; but whether the current is able to influence processes of disease in these parts is a question which is open to considerable discussion. It has been assumed that nerve elements which for one reason or another were in a process of atrophy could, by means of electric excitation, be incited to regeneration, or at any rate arrested in their further degeneration, and that excitation of the trophic centres would here produce an increased development and thus exert an influence over those parts of the nervous system which were still unaffected by the disease. Also, it was thought that the galvanic current could produce vasomotor changes, which, in their turn, would promote retrogression of the pathological process (absorption of exudations, new growths, etc.).

That we have in the galvanic current a means by which changes in the circulation of the blood in the brain can be affected may be experimentally demonstrated by ophthalmoscopic examination of the retina.

The normal functioning of the central nervous system depends to a great degree upon a well-regulated circulation; disorder of the circulation will, if temporary, produce functional disturbances; if repeated or constant, it seems probable that permanent changes, degenerations, may result. Under these circumstances it cannot be denied that, possessing in electricity a remedy which can influence the circulation in the central nervous system, we are warranted in making use of this agent in the treatment of certain diseases of these parts.

It is, however, improbable that any direct influence can be exerted upon the ganglion cells by means of the electric current, and thus far no proof of any kind has been furnished that such a specific action exists. Under these circumstances, we would hardly expect to obtain any beneficial results in a large number of diseases of the brain and spinal cord. Clinically, this assumption is fully corroborated, for all organic diseases of the nervous system in which the motor cells are primarily affected can in no way be permanently influenced by electrical treatment. At the same time, not all the symptoms occurring in such disease are due to the organic changes in the ganglia. Symptoms must be produced by the functional disturbances which precede the organic changes, and that these early disorders may be indirectly influenced and beneficially stimulated by acting upon their blood-supply cannot be denied.

In *electrization of the brain* stabile galvanization is used; medium-sized or large electrodes should be employed, and so placed as to obtain the greatest density of current over the diseased focus. The currents should be weak, from one to five milliampères, and the duration short, two to five minutes. If the brain is to be traversed by the current longitudinally, a large flexible electrode is to placed upon the forehead and a smaller one on the occipital region; if transverse electrization is desired, a medium or large electrode is applied to each mastoid region.

Considering the progress made by neuropathologists in topical brain diagnosis, it would not seem impracticable to endeavor to localize the action of the current to the actual seat of disease. For this purpose a small electrode is to placed over the *locus morbi*, and the other large electrode on the opposite side of the head or neck. It is as yet entirely impossible to determine with intact skull which parts of the brain in particular are traversed by the current, so that this method should still be received with a certain amount of reserve.

In addition to the direct electrization of the brain, great importance has been attachéd in the treatment of diseases of this organ by prominent electrotherapists to subaural galvanization; this has already been described.

The electrical treatment of *cerebral hemorrhage and its dependent disorders* consists chiefly of galvanization of the brain and faradization or galvano-faradization of the paralyzed muscles. Considering the frequency of cerebral hemorrhage and the prolonged disability due to the resultant paralysis, it is important to know when to begin electrical treatment, and to appreciate how far any result may be expected from such applications.

It should be self-evident, and not require special mention, that the use of electricity during the apoplectic attack is absolutely contraindicated. There seems to be a general agreement that in these cases electricity should not be used earlier than four to six weeks after the attack. This is certainly proper, for nothing is to be gained by its earlier use, and harm may be done. The manner of applying the current is that already described; each application should be short, and attention should be called before the treatment is begun to the possible occurrence of dizziness, nausea, and headache, so that the patient experience no alarm should they occur. The treatment is to be repeated every second day, and continued for about a month. If any ill effects of an alarming nature are observed, the direct treatment of the brain should be at once discontinued. The occurrence of *late rigidity*, of *œdema* of the extremities, of *glossy skin*, or *joint affections*, cannot be prevented, nor once present can they be benefited by the use of electricity.

Aphasia may be treated by localized galvanization, the anode being placed upon the previously shaved scalp over the left lower frontal or first temporal convolution, and the kathode on the nape of the neck.

The treatment of the *paralyzed limbs* must not be neglected. Labile applications of the kathode, the anode being placed on some indifferent point, or faradization of the nerves and muscles are here indicated.

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orders not ending in suppuration, the *cerebral palsies of childhood*, the athetosic movements resulting therefrom, hardly deserve to be considered as subjects for electrical treatment. Also, in *mental diseases*, we would hardly expect to attain beneficial results from any known electro-therapeutic procedure, nor should we forget that fresh delusions may easily be implanted upon such a psychically disordered brain.

In certain conditions of *stupor*, however, energetic use of the faradic brush, excitations of the skin by the static spark, often by their reflex modification of the cerebral circulations, produce a decided improvement in the mental condition of the patient.

The *spinal cord* may be treated electrically in a direct and an indirect manner. For direct electrization of the cord the galvanic current alone is available, and, even with this, it is a question whether we are able to reach the spinal cord through its bony envelope; certainly actual proof of such a possibility does not exist; clinically, however, we do see an improvement in certain symptoms follow the application of galvanism to the cord, so that, inasmuch as no harm can be caused by such treatment, it is our duty to make use of it in this class of diseases which in general is so barren of therapeutic results.

The current is to be applied with large electrodes, and the labile method used if the entire length of the cord is to be influenced, the stabile method if it is desired to act upon any particular segment. Under all circumstances, on account of the deep situation of the cord, strong currents must be employed.

In *affections of the conducting tracts*, whether primary or secondary, one pole may be placed upon the sternum and the other successively passed over all the parts of the spinal column, or the stabile method may be made use of by placing one pole on the cervical and the other on the lumbar region.

In any *transverse focal* affection or disorder at a certain level of the cord we should attempt to directly transverse the focus with the current by placing a large electrode on the spine over the diseased segment, and the other electrode upon the ventral surface of the body, so that the affected part lies in a direct line between the two electrodes. As a matter of general agreement it is well to use the anode only in irritable conditions and the kathode in atonic ones, as the active pole.

Besides this direct electrization of the cord, we may also act upon it indirectly by means of peripheral faradization. Here either the skin over the spinal column may be faradized with a dry electrode or with a wire brush, or the skin of the extremities thus treated until a marked reddening of the surface takes place.

Hemorrhages into the spinal cord are of so little practical importance on account of their rare occurrence that only a few words need be said about their electrical treatment. If it is admitted that the cord can be reached by the current, and provided that we can localize the seat of the hemorrhage, there is no reason why resorption of the clot and nutritional changes may not be effected through galvanization.

As *inflammatory processes of the cord* those disorders are here to be considered in which there is a destruction of nerve elements and a secondary increase of connective tissue, involving the gray as well as the white substance of the cord and not confined to any system of fibres. They may be summed up under the generic name of myelitis. Only in the chronic forms is any hope of electrotherapeutical success warranted, and even here the prognosis will naturally vary according to the special characteristics of each individual case. The prime indication in the electrical treatment of chronic myelitis is the localization of the lesion; this having been accomplished, the cord is to be treated in the manner already described. The results of the cord disease, the paralyses of the extremities, the disturbances of bladder will have to be treated symptomatically. For the former galvanization of the nerves and muscles is to be combined with the galvanization of the lesion ; for the latter affection the method employed is to apply the anode to the lumbar enlargement, the kathode over the bladder, and to allow a current of medium strength to pass for several minutes. Voltaic alternatives may then be used, and if necessary intra-vesical applications or excitations of the sphincter of the bladder, according to whether the condition here is one of spasm or of paralysis. If intra-urethral or intra-vesical applications are to be made, the strictest antiseptic methods must be employed. Faradization may also be used either externally or internally. The results attained by the use of electricity alone are usually not of a satisfactory nature.

In *poliomyelitis anterior acuta* the treatment should be directed to the spinal cord in the manner described according to the seat of the lesion and also to the degenerated muscles. The latter is the most important, and should be carefully and energetically carried out and continued for a year or even more, from the onset of the disease, if there is the slightest evidence of improvement.

Even later it is of advantage to institute a fresh course of treatment covering a period of from eight to ten weeks, twice or three times a year. In cases which have apparently come to a standstill, an improvement will frequently be noted after each course of treatment. A large electrode is to be applied over the diseased portion of the cord, and with a medium-sized exciting electrode all the affected muscles are to be made to contract. The skin is to be markedly irritated during this procedure, as we thus obtain a reflex action upon the cord. The galvano-faradic current here merits the preference, but the induced current alone may be employed. A thorough electro-massage of the muscles is also of advantage. Daily applications of from five to ten minutes' duration are indicated; electricity should here not be used to the exclusion of other methods, as a great deal can be done for these children in other ways with energy and perseverance.

Subacute and chronic poliomyelitis, whether occurring in children or in adults, should be treated upon the same principles. Here it is again necessary to emphasize the fact that improvements and recoveries of the lost functions may occur after the lapse of a very long period of time, so that it is essential to keep up the nutrition of the muscles and to persevere in the electrical treatment for many months. The treatment should be begun as early as possible, but of course not until all general symptoms have passed away.

In *progressive muscular atrophy* the treatment is to be carried out in much the same manner as in poliomyelitis, galvanization of the cord and strong galvanic faradization of the affected muscles; the results of electrical applications are decidedly unsatisfactory in this affection, and, while it will rarely be possible to forego the use of this remedy at some period of the disease, it is well to be perfectly frank with ourselves and to acknowledge the futility of such treatment.

The same may be said of other more systematized diseases of the spinal cord. In *spastic spinal paralysis, ataxic paraplegia, and amyotrophic lateral sclerosis*, electricity can only serve as a means for keeping up the hopes of the patient.

In the treatment of *locomotor ataxia* (tabes dorsalis) electricity still occupies an important position in the opinion of many prominent neurologists. Undoubtedly a large number of the indisputable improvements in some of the symptoms which occur soon after the beginning of electrical treatment in cases of tabes are dependent upon psychic influences, but the fact that certain other symptoms are always improved in the same sequence goes far to sustain the supposition that electricity possesses a specific influence upon them. If we consider that the anatomical changes in tabes are not limited to the spinal cord, but that the peripheral sensory nerves are usually invaded by the degenerative process, and in many cases are the primary seat of disease, it is evident that taken very early such cases must be susceptible of cure or of arrest.

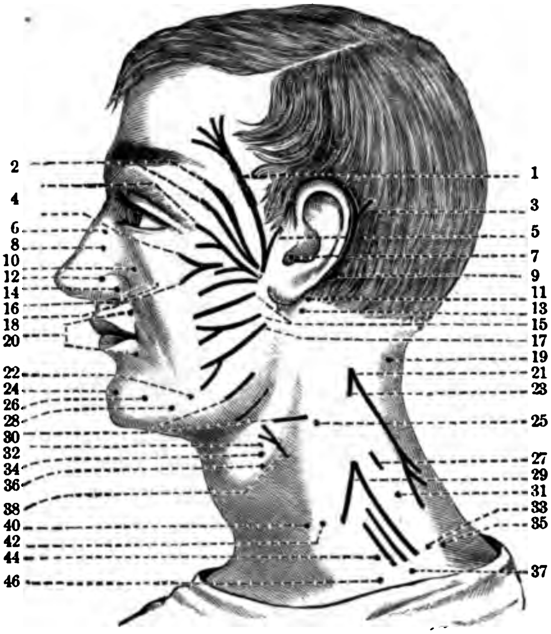
It is all a question of early diagnosis, and how early the diagnosis of tabes can be made with certainty is a point which can hardly be decided. That the tabic process cannot be influenced by electricity after the gross anatomical changes, which we are accustomed to find in the cord, have taken place, is certain, and it seems probable that all improvements which may occur after the earliest initial stage has passed occur in the natural course of the disease, and are not due to any remedy which may have been employed. Unfortunately tabic patients are subjected to electrical treatment only after a great deal of valuable time has been lost by attempts to influence the disease through internal medication. Electricity should be used in the very early stages and not as a last resort; then and then only can its proper remedial position in this disease be ascertained. It is my personal opinion, gained of course only by clinical observation, that cases showing sensory disturbances, transitory attacks of diplopia, which on this account were diagnosticated as tabes incipiens, have been prevented from growing worse through systematic and prolonged courses of electrical treatment.

The treatment in the early stages should be directed to the peripheral parts. General galvano-faradization of the skin, muscles, and nerves of the entire body, with the exception of neck, head, and face, is here indicated. Such daily applications, covering a period of four to six weeks, are often followed by the most satisfactory results. Should this not be the case, or should the patients be first seen after symptoms of anatomical cord changes have presented themselves, then the direct galvanization of the cord is to be employed in addition to general farado-cutaneous brushing. In the fully developed stage of the disease electricity in any form is useless.

There can only be one opinion concerning the action of electricity upon *diseases of the peripheral nerves*, motor as well as sensory, and that is that such treatment is frequently followed by good results. This statement can hardly be disputed by the most intense suggestionist, as it allows amply for, the natural tendency to recovery as well as for any effect which the patients psychic state may have upon the acceleration of such recovery.

Disorders of the *motor nerves* may be divided into two kinds, the paralytic and the spasmodic. While practically the benefits derived from the use of electricity in the paralytic form of the motor nerve disorder have been demonstrated in manifold instances, we are theoretically far from understanding the manner in which these results are brought about. It is obvious that where complete and irreparable destruction of the nerve-tissue exist, electricity as well as every other method will fail to re-establish conduction. On the other hand, it should be clear that the remarkable improvements which are frequently observed to follow upon a single application of electricity are entirely dependent upon suggestion, and not due to any specific action of the current. At the same time, if we are able, through causing a functionally disordered group of muscles to contract energetically, to produce upon the centres which preside over this group a perception of motion corresponding to this muscular contraction, we certainly convey to these centres the suggestion that the muscles can be freely used. That we do possess in electricity an unequalled and incomparable method of producing such a suggestion is manifest. The fact, therefore, that in certain cases of paralysis the action of electricity is a psychic one does not in the least invalidate its curative properties; in addition to this psychic action it must also be admitted that the electrical excitation to contraction of those muscular fibres, which still react mechanically, aids in producing a better nutrition, and thus an increased growth of these fibres.

FIG. 332.



A diagram of the motor points of the face, showing the position of the electrodes during electrization of special muscles and nerves. The anode is supposed to be placed in the mastoid fossa, and the kathode in the part indicated upon the diagram. (From VON ZIEMSSEN.)

1. Occipito-frontalis (ant. belly). 2. Corrugator supercilii. 3. Occipito-frontalis (post. belly). 4. Orbicularis palpebrarum. 5. Retrahens et attollens aurem. 6. Pyramidalis nasi. 7. Facial nerve. 8. Lev. lab. sup. et alæ nasi. 9. Deep posterior auricular branch of facial nerve. 10. Lev. lab. sup. propr. 11. Stylo-hyoid. 12. Dilator naris ant. 13. Digastric. 14. Dilator naris post. 15. Buccal branches of facial nerve. 16. Zygomat. minor. 17. Subcutaneous branch of inferior maxillary nerve. 18. Zygomat. major. 19. Splenius capitis. 20. Orbicularis oris. 21. External branch of spinal accessory nerve. 22. Branch of levator menti and dep. ang. oris. 23. Sterno-mastoid. 24. Levator menti. 25. Sterno-mastoid. 26. Dep. lab. infer. 27. Levator anguli scapulæ. 28. Dep. ang. oris 29. Phrenic nerve. 30. Subcutaneous nerves of neck. 31. Posterior thoracic nerve to rhomboid muscles. 32. Sterno-hyoid. 33. Circumflex nerve. 34. Omo-hyoid. 35. Posterior thoracic nerve to serratus magnus. 36. Sterno-thyroid. 37. Branch of brachial plexus. 38. Branch for platysma. 40. Sterno-hyoid. 42. Omo-hyoid. 44, 46. Nerves to pectoral muscles.

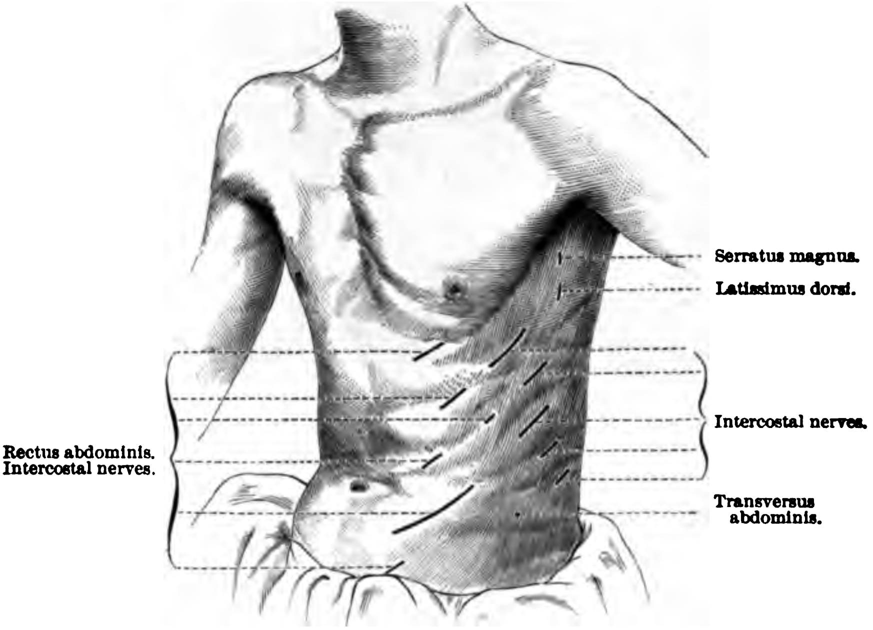
Whether electricity has any action whatsoever upon the arrest of degeneration or promotion of the regeneration in a diseased nerve is an undecided question. The treatment must here, as in other affections, be divided into a peripheral and a central one, the peripheral treatment consisting in electrization of the muscles and the central one in treating the diseased nerve above or at the seat of lesion.

In direct excitation of the muscles the faradic current is to be used; if the excitability to the faradic current is lost, labile and interrupted galvanic currents should be employed, and only if the galvanic excitability is markedly reduced are voltaic alternatives indicated. In excitation of a muscle from the nerve either the faradic or galvanic current may be employed.

As regards the curative action of static electricity upon paralysis we still possess very few data. That the action of the static and faradic current is not absolutely identical is shown by cases of peripheral paralysis in which the muscles fail to respond to the faradic current, but react well to static excitation.

In the treatment of *peripheral paralysis* we should not fail to classify the cases according to the severity of the nerve affection, as determined by electrical examination.

FIG. 833.



Motor points of the trunk. (From VON ZIEMSSEN.)

In light cases, which get well in from four to six weeks, it must have become apparent to every observer that a slight increase in voluntary control is produced after each application of electricity. In medium cases, which improve up to a certain point and then seem to remain stationary, rapid progress toward complete recovery may be inaugurated by the use of electricity. The severe cases, with well-marked reaction of degeneration, run their usual course, entirely uninfluenced by electrical treatment.

The paralysis due to peripheral nerve disorder to which electrical treatment may be directed are:

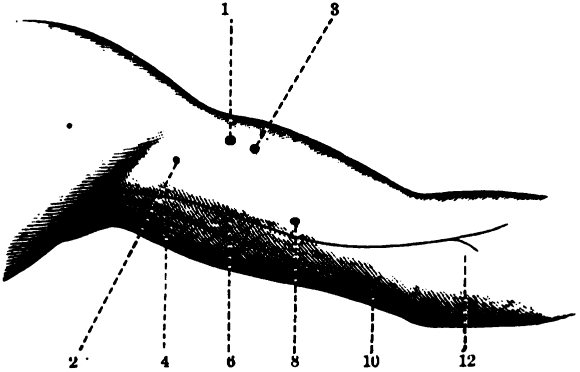
Paralysis of the motor nerves of the ocular muscles. In paralysis of the third, fourth, and sixth pair of cerebral nerves the direct treatment of the muscles is, on account of their situation, impracticable. Nevertheless an attempt should be made to produce indirect kathodic excitation of these paralyzed muscles, by applying the small kathode to the closed lids over the insertion of the muscle to be acted upon, the eye at the same time being turned in the opposite direction. Whether any decided excitation of the muscle is thus produced is questionable, although the possibility of such production cannot be denied. The faradic current applied through the closed lids is certainly entirely useless.

The insertions of the paralyzed muscles in the conjunctiva may be directly excited, the pain being lessened by an application of cocaine. A galvanic current sufficiently strong to stimulate the muscles is, on account of the close proximity of the retina and the delicate structure of the conjunctiva, scarcely safe.

The indirect method recommended by Benedikt consists in placing one pole (anode) upon the forehead, the other (kathode) on the margin of the orbit, near the affected muscle. Either the faradic or the stabile or labile galvanic current may be thus used. All in all, it is questionable whether, besides an increase of power observed after the current has been applied for a few minutes and which is quickly lost, we are able to accomplish anything by the electrical treatment of these nerves.

Facial paralysis, if of peripheral origin, whether due to a lesion of the nerve after its exit from the stylo-mastoid foramen, or whether the lesion affects the nerve in its course from the pons to the Fallopian canal, may be beneficially influenced by electrical treatment. The action of electricity in the first instance, however, is a direct one, while in the second case a centripetal excitation of the facial trunk is produced reflexly through the trigeminus. The lighter cases, as determined by the electrical reactions, may be treated with the faradic current alone or in conjunction with a labile galvanization of the muscles supplied by the nerve, the indifferent electrode (anode) being well pressed in behind and below the ear upon the point of emergence of the nerves.

FIG. 334.

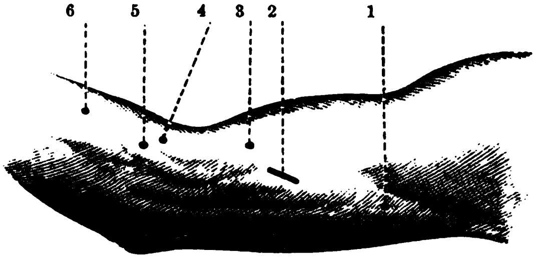


Motor points of the arm, under side. (From VON ZIEMSSEN.)

1. Musculo-cutaneous nerve. 2. Musculo-cutaneous nerve. 3. Biceps. 4. Internal nerve of triceps. 6. Median nerve, 8. Brachialis anticus. 10. Ulnar nerve. 12. Branch of median nerve to the pronator teres.

In severe cases, first the kathode and then the anode should be used in the same manner; then a series of closure and opening contractions should be obtained; and, finally, galvano-faradization of the entire territory carried out. The seat of the lesion can, of course, not be reached directly. Each application should last about five minutes and be repeated three to four times a week. The duration of such treatment will depend entirely upon the severity of the affection, but averages from three weeks to six months. If, after six months, no marked improvement has taken place, the treatment should be determined. It is, however, well in all cases of old, long-standing facial paralysis to renew the electrical treatment from time to time, as fresh improvements may thus be produced.

FIG. 335.



Motor points of the arm, outer side. (From VON ZIEMSSEN.)

1. External head of triceps. 2. Musculo-spiral nerve. 3. Brachialis anticus. 4. Supinator longus. 5. Extensor carpi radialis longior. 6. Extensor carpi radialis brevior.

Pareses of the muscles of the larynx frequently necessitate the application of electricity. Intra-laryngeal electrization is, on account of its difficulty and unpleasantness, to be avoided, particularly as it presents no advantages over the extra-laryngeal method. A large electrode is to be fastened over the nape of the neck, and labile galvanization of the skin over the larynx and trachea carried out. Voltaic alternatives and galvano-faradization are often of benefit.

Electrization of the *phrenic nerves* is chiefly made use of in paresis of the diaphragm, if artificial repiration is to be produced. The best plan is to press a very small round electrode well in at the posterior border of the sterno-mastoid above the lower end of the scalenus anticus. A strong faradic current is to be used, and is to be applied until respiration is re-established. With the first sign of returning respiration the current is to be discontinued.

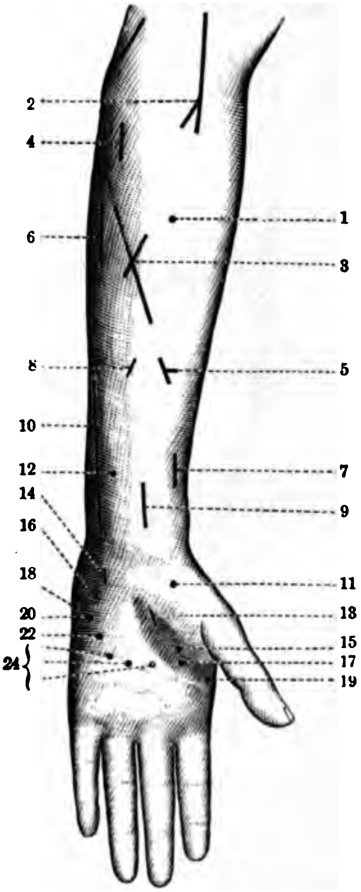
The paralyses affecting the *nerves of the arm and leg* are to be treated electrically upon the general principles already set down; labile galvanization and galvano-faradization of the entire territory innervated by the affected nerve, the kathode and anode being used successively, and the other pole being placed over the seat of lesion or at some indifferent point.

For the treatment of *spasmodic affections* of motor nerves only general rules can be given, and these must be modified to meet the exigencies of the various forms and localizations.

The main indication in the treatment of spasms will be to discover and remove their cause and to modify the functional or molecular disorder, which probably exists in the motor nerves and their centres. Whether the latter indications can be met by the use of electricity, can be decided only empirically, and it seems that some cases and forms of spasm are benefited by its use.

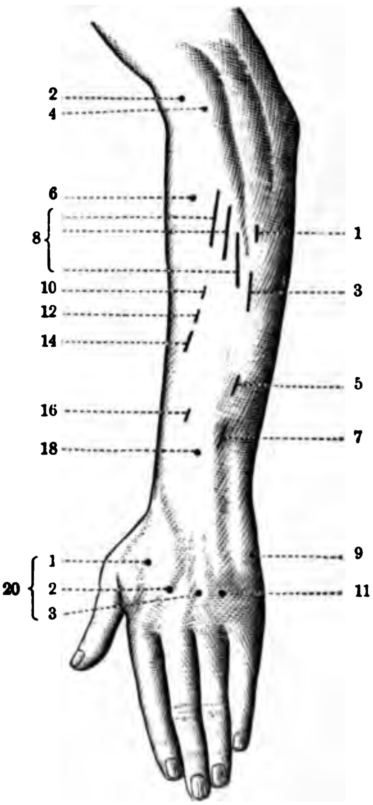
In the electrical treatment galvanism alone is indicated, and, inasmuch as it is always a question of reducing the irritability of the motor apparatus, the anode will be employed; it is also assumed that through the modifying and catalytic effects of the current pathological irritations in the vicinity of the nerves may be removed; if this is so, we also have in galvanism a remedy which will act upon the cause of the spasm in some cases. The single forms of spasm to which galvanism is specially applicable are facial spasm, with its offspring, blepharospasm, torticollis tonic and clonic, and the various spasms or cramps occurring in the muscles of the hand and arm in consequence of overexertion.

Fig. 336.



Motor points of forearm, inner surface.

Fig. 337.



Motor points of forearm, outer surface.

(From Von Ziemssen.)

Fig. 336.—1. Flexor carpi radialis. 2. Branch of the median nerve for the pronator teres. 3. Flexor profundus digitorum. 4. Palmaris longus. 5. Flexor sublimis digitorum. 6. Flexor carpi ulnaris. 7. Flexor longus pollicis. 8. Flexor sublimis digitorum (middle and ring fingers). 9. Median nerve. 10. Ulnar nerve. 11. Abductor pollicis. 12. Flexor sublimis digitorum (index and little finger). 13. Opponens pollicis. 14. Deep branch of ulnar nerve. 15. Flexor brevis pollicis. 16. Palmaris brevis. 17. Adductor pollicis. 18. Adductor minimi digiti. 19. Lumbricalis (first). 20. Flexor brevis minimi digiti. 22. Opponens minimi digiti. 24. Lumbricales (second, third, and fourth).

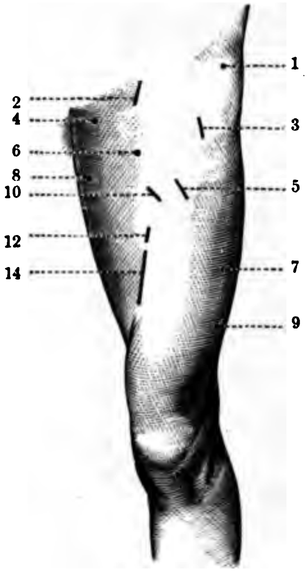
Fig. 337.—Extensor carpi ulnaris. 2. Supinator longus. 3. Extensor minimi digiti. 4. Extensor carpi radialis longior. 5. Extensor indicis. 6. Extensor carpi radialis brevior. 7. Extensor secundi internodii pollicis. 8. Extensor communis digitorum. 9. Abductor minimi digiti. 10. Extensor indicis. 11. Dorsal interosseus (fourth). 12. Extensor indicis and extensor ossis metacarpi pollicis. 14. Extensor ossis metacarpi pollicis. 16. Extensor primi internodii pollicis. 18. Flexor longus pollicis. 20. Dorsal interossei.

Of the disordered conditions to which the *sensory nerves* are subject, anæsthesia and pain are the two which chiefly call for electrical treatment.

Paræsthesias and other sensory disturbances very rarely, *per se*, require treatment of any kind, as they are usually simply concomitants of other more serious troubles.

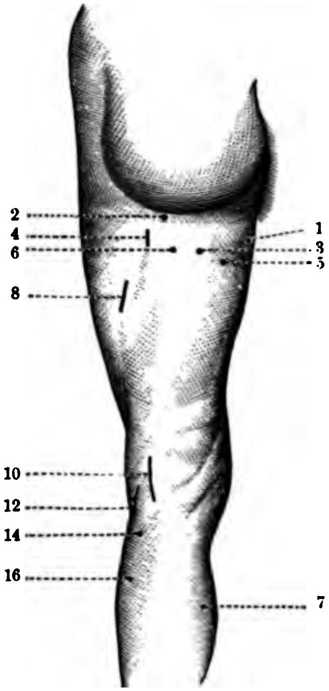
Anæsthesia will have to be treated upon the same general principles as paralysis, directing the treatment to the seat of the disease, when this can be localized, as well as to the seat of the symptoms. Anæsthesia due to central disorder must be carefully differentiated from that due to peripheral causes, for in the former the good results of electrical treatment will be sought for in vain, while in the latter thorough faradization with a dry electrode or with a faradic brush will be found to exert a beneficial influence in very many cases.

Fig. 338.



Motor points of thigh, anterior surface.

Fig. 339.



Motor points of thigh, posterior surface.

(From Von Ziemssen.)

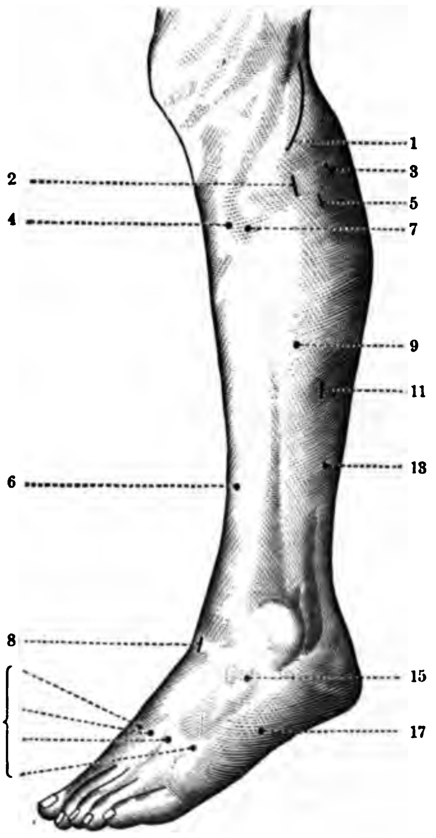
Fig. 338.—1. Tensor vaginæ femoris (branch of superior gluteal nerve). 2. Anterior crural nerve. 3. Tensor vaginæ femoris (branch of crural nerve). 4. Obturator nerve 5. Rectus femoris. 6. Sartorius. 7. Vastus externus. 8. Adductor longus. 9. Vastus externus. 10. Branch of crural nerve to quadriceps extensor cruris. 12. Crureus. 14. Branch of crural nerve to vastus externus.

Fig. 339.—1. Adductor magnus. 2. Inferior gluteal nerve for gluteus maximus. 3. Semitendinosus. 4. Great sciatic nerve. 5. Semi-membranosus. 6. Long head of biceps. 7. Gastrocnemius (internal head). 8. Short head of biceps. 10. Posterior tibial nerve. 12. Peroneal nerve. 14. Gastrocnemius (external head). 16. Soleus.

In the treatment of *neuralgias* the specific curative action of electricity has been generally acknowledged; but whether this action is dependent upon the production of a change in the excitation or nutrition of the hyperæsthetic nerve, or whether direct anæsthesia is produced by means of strong counter-irritation, or whether the action is not a purely mechanical one, producing a molecular change in the nerve itself, cannot be stated.

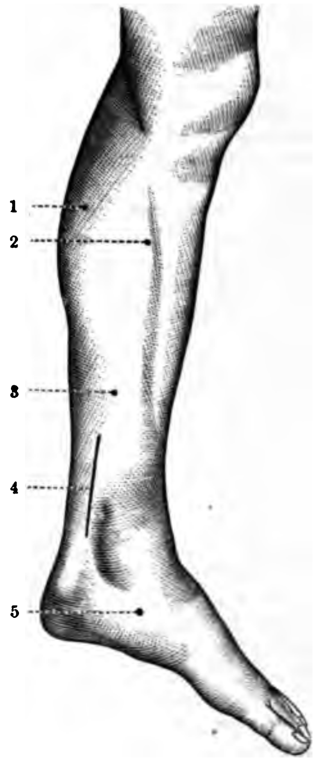
This much is certain, that whatever may be the cause of the beneficial influence, idiopathic neuralgias offer a more fruitful field for electrical treatment than do any other affections of the nervous system. In accordance herewith, the differentiation between the idiopathic and symptomatic neuralgias is of prime importance; if the neuralgia is found to be a symptom of some local or general affection, other remedies than electricity will have to be employed for the removal of this cause, but aid may be sought in the use of general faradization or galvano-faradization. If, on the other hand, the

FIG. 340.



Motor points of the leg, outer side.

FIG. 341.



Motor points of the leg, inner side.

(From VON ZIEMSSEN.)

FIG. 340.—1. Peroneal nerve. 2. Peroneus longus. 3. Gastrocnemius (external head). 4. Tibialis anticus. 5. Soleus. 6. Extensor longus pollicis. 7. Extensor communis digitorum longus. 8. Branch of peroneal nerve for extensor brevis digitorum. 9. Peroneal brevis. 10. Dorsal interossei. 11. Soleus. 13. Flexor longus pollicis. 15. Extensor brevis digitorum. 17. Abductor minimi digiti.

FIG. 341.—1. Gastrocnemius (internal head). 2. Soleus. 3. Flexor communis digitorum longus. 4. Posterior tibial nerve. 5. Abductor pollicis.

neuralgia is as far as can be determined not dependent upon any such local or general disease, then, as stated, great service may be expected from the direct use of electricity. In this direct treatment all three currents may be employed, but it is well always to begin with the galvanic anode on account of its analgesic action. The method of application is usually a stabile one, the kathode being placed at some indifferent point and the anode upon the place of exit of the nerve or upon a particularly painful point. The size of the electrode to be used will be governed here, not only by the general principles already laid down, but by the extent of the pain. A diffuse pain would indicate the employment of a broad electrode, while a circumscribed pain requires the use of a small one. During this application variations in the current density are to be carefully guarded against, and interruptions under all circumstances to be avoided.

If we have satisfied ourselves that the galvanic current gives no relief, the faradic current may be employed, either for its direct or for its indirect action. If the former is desired, a weak current is passed directly through the nerve; if this current is well borne, it may be slowly and carefully increased, and then again decreased. Indirectly the faradic current acts purely as a counter-irritant, and nothing can serve the purpose of such application better than the metallic brush. An increased counter-irritant action can, if desired, be obtained by attaching the brush to the galvanic kathode; after application of the electric brush remarkable and continued improvements are often noted, so that this mode of procedure should always be tried if the stabile application of galvanism has failed.

In some cases in which both the galvanic and faradic current have failed to produce good results static electricity seems to give evidences of analgesic properties.

All depends here upon the method of application and the carefully graduated discharge of the current, so as to avoid giving shocks to the patient. It would almost seem as though, in consequence of the enormous tension of this current and of the great deviations which take place during every discharge, that molecular alterations must be produced in the nerves.

A matter of general experience in the electrotherapy of neuralgias is that certain nerves are more easily and more quickly influenced by this agent than by others; this difference is also noted among various branches of the same nerve.

The neuralgias which we are most frequently called upon to treat are those of the trigeminus and of the sciatic nerves. As regards the method of treatment of *trigeminal neuralgias*, there is not much more to be said than that which has already been stated in the general remarks; the painful points, the supra-orbital, infra-orbital, and that of the chin, will be treated anodally, according to which branch is particularly affected. The current should be gradually increased up to the desired point, and then again equally gradually decreased. Each application should last from three to five minutes, and in acute cases one or two daily applications are indicated. Great care must be exercised in the electrical treatment of the affections of this nerve, as not infrequently exacerbations of the pain occur as a direct result of such treatment. Purely symptomatically, the use of medicinal cataphoresis will temporarily serve to alleviate the sufferings of the patient.

In the treatment of *sciatica* by electricity a great diversity of opinion exists as to the methods to be employed, as well as to the results which may be attained. This is certainly due to the fact that we do not exercise sufficient care in properly differentiating the various cases and separating the neuritic sciaticas from the neuralgic ones. The first form—and to this the majority of cases belong—are certainly but little helped by the use of electricity, while in the second category we very often attain good results. It is essential in this form to subject the entire nerve to the action of the current. For this purpose a large plate electrode is placed over the sacrum or over the sciatic notch, while the other electrode is passed over the entire course of the nerves. Strong currents may be used, and each application be made to last from ten to fifteen minutes. Galvano-faradization of the entire limb and faradic brushing of the skin are also occasionally of benefit.

Of other neuralgias, the *cervico-occipital* and those of the *radial* and *ulnar* territories are frequently quickly and permanently relieved by a few applications of electricity.

Of *intercostal* neuralgias the same may be said as of sciatic :that they are often due to inflammatory disorders of the nerve, and are then hardly susceptible of improvement by means of electricity. In idiopathic intercostal neuralgias relief is obtained by the usual methods. These remarks apply in their entirety to the form known as *mastodynia* and to the "*functional neuroses*," from the fact that disorders of the nervous system, without known pathological basis, may produce local as well as general symptoms. We can understand that their electrical treatment may similarly be divided into a local and a general one. According to the nature of the case, the relief of simple symptoms and the restoration of the disturbed nervous parts to their normal condition will be the results aimed at. The first of these indications may often be met by local electrical treatment alone, but in the second it is always necessary to subject the entire central nervous system to the modifying treatment, and here electricity should be regarded simply as an adjuvant, a very valuable one, it is true, to other remedies.

Among the functional neuroses, in the treatment of which electricity has been at various times highly recommended, is *chorea minor*. The failure of all the usual remedies occasionally makes it necessary that electricity should be tried in this affection.

The only current which could possibly have any influence upon this disorder is the galvanic, and galvanization of the spine or of the brain has been recommended, according to whether the advocate was an adherent of the spinal or cerebral theory of its causation. Temporary results are occasionally obtained by either method, but I have never seen any evidence of permanent benefit; on the other hand, I have repeatedly seen the choreic movements decidedly increased through unskilled electrical applications. Everything which excites or frightens the patient is to be strenuously avoided in the treatment of chorea; hence the use of the faradic current, of the electric brush, of strong galvanic currents, as has been advocated, cannot be too strongly discountenanced.

Cerebral galvanization has also been recommended in *epilepsy*, notwithstanding that neither our knowledge of the pathogeny of this obscure disease nor clinical experience furnishes a single argument in its favor. The current recommended has naturally been galvanic, and the methods those of central and subaural galvanization. If made use of at all, extraordinary care should be exercised in these applications, for the occurrence of an epileptic attack during the passage of the current would certainly be attributed to this procedure.

All in all, even the reports of the advocates of the use of electricity in the treatment of epilepsy are not such as to warrant us in expecting as good results from it as we do from the administration of certain internal remedies.

Paralysis Agitans. The electrical treatment of this disease, galvanic or static, occupies the same place as treatment of other nature. Various writers have recommended this or that electrical method as palliative of certain symptoms, or even as curative of the disease, but every conscientious observer who follows these recommendations must arrive at the conclusion that all forms and methods of electrical treatment are worse than useless in this affection.

Exophthalmic goitre is one of the "neuroses" in which galvanism is undoubtedly of a great deal of benefit. That the affection is ever arrested in its course, or entirely cured by the use of electricity alone, is improbable, but that single symptoms often disappear, and that the entire condition of the patient is improved by its use, is quite generally acknowledged.

The method which is usually advocated is that of subaural galvanization. In many instances a lowering of the pulse-rate of from 15 to 20 beats per minute may be obtained by each application, and exceptionally even a decrease in the size of the thyroid may be noted. Unfortunately this effect is usually transient, but repeated applications may produce permanent effects. A large number of other methods, including general faradization and galvanization of the brain, have also been recommended, and it is certainly difficult from the various descriptions to decide upon the best method of treating this disease.

In my experience two principles should form the basis of every treatment, viz., the use of weak currents and their very frequent applications. The frequency of the applications (several times a day) necessitates self-administration by the patient, and the plan recommended by Cardero answers all requirements. The galvanic current alone is to be used, the current strength not to exceed two milliampères, and each application to last from five to ten minutes. "The anode is to be placed on the nape of the neck, the centre of its lower border corresponding to the seventh cervical spinous process, and be firmly held in that position during the application. The electrode should be moved up and down the side of the neck from the mastoid process along the course of the great occipital nerve."

A battery containing a sufficient number of dry or wet cells—*i. e.*, of sufficient voltage to give the desired current (the resistance of the skin or electrodes, of course, being considered)—together with electrodes of a fixed diameter, may be given to the patient with the necessary instructions. No selector, rheostat, nor galvanometer is necessary, as the current cannot exceed the desired amount, and, with attention to details (wetting the electrodes, etc.), can fall but little below it. According to the voltage of the cell used, and assuming the resistance of the parts to which the current is to be applied, when thoroughly moistened, to be from 2000 to 3000 ohms, from three to six cells will be required. The battery should be inspected at regular intervals, so as to be sure that everything is in working order.

The local treatment of the struma or exophthalmus either by galvanic or static currents is irrational and not productive of favorable results. Everything should be done to improve the general nutrition of the patient, and with this in view general faradization or galvano-faradization is often of value.

A number of cases of exophthalmic goitre will be encountered with undeveloped or not well-defined symptoms. Such cases, showing only one of the classical triad of symptoms, with or without some of the other less important phenomena, such as tremor, etc., are frequently indistinguishable from cases of neurasthenia. They will do well if treated according to the authority laid down in the following paragraphs.

NEURASTHENIA. If there is any affection of the nervous system in which physical methods alone deserve prime consideration, it is the disorder known as neurasthenia. Among these physical methods no remedy plays a more important part than electricity. Electricity may here form part and parcel of other treatment—drugs, rest, nutrition, massage, hydrotherapy—or may be used alone. In either case general faradization or galvanization may be used. It is my opinion that strong currents are to be avoided, and that a moderate excitation of the peripheral nerves is all that is necessary. Neurasthenics require very circumspect treatment, and the symptoms of cerebral or spinal irritation are very often increased by the use of strong currents; each application should also be short—five to ten minutes—at any rate at first, and later the time may be increased.

The results of general electrization are often directly apparent in the relief of pain, fatigue, and regulation of the pulse.

Besides general electrization, central galvanization is of benefit in the treatment of neurasthenics whose general nutrition seems to be good.

Also in the treatment of single symptoms the use of electricity is of value. Thus headache, sleeplessness, palpitation, etc., may often be relieved by cerebral galvanization. Particularly against the insomnia of neurasthenia has galvanization of the brain and the static head-douche been recommended. Personally, I have often found the use of the positive static charge of very great benefit in combating this symptom, having been able to produce sleep by this means after all other physical methods had failed and a resort to the use of drugs being undesirable. In many cases of neurasthenia, as already stated, electricity in any form does harm instead of good; it is not possible to determine without a trial which cases will be benefited and which ones made worse by the use of electricity. If patients after each application complain of being excited, of tremor, dizziness, headache, tendency to faint, and sleeplessness, there is either something wrong in the method of application or the use of electricity is contraindicated.

HYSTERIA. All doubts which have been expressed concerning the specific curative action of electricity in organic diseases of the nervous system deserve to be much more strongly emphasized in considering the treatment of hysteria. Many of the symptoms met with in this affection are the result of auto-suggestion, and these same symptoms can often be made to disappear by an allo-suggestion transmitted through the porper channels. Whatever vehicle may have been employed for this transmission, we should be clear upon the point that it is the suggestion and not the vehicle which has affected the cure. In the great majority of cases when electricity is of service in dispelling certain predominant symptoms of hysteria it is as a vehicle for carrying suggestion that its action must be sought; and as such a psychic remedy its value in the treatment of hysterical symptoms is very great. The firmest believer in the actual therapeutic value of electricity will hardly contend that the disease "hysteria" can be cured by this remedy. The removal of certain symptoms is an important part of the treatment of this disease, and any remedy which will aid us in effecting such a result, immaterial in what way, must be considered a valuable acquisition.

In every case a certain plan of treatment must be laid out and followed, but not blindly; changes and modifications will be necessitated by the course and character of the symptoms in different patients, and nowhere is it more important than here to remember that we are treating individuals and not diseases.

As part and parcel of the Weir Mitchell treatment, or whenever it is deemed necessary to increase nutrition and to promote assimilation, general electrization will be found of value.

Local symptoms, paralysis, contractures, neuralgias, hyperæsthesias, and anæsthesias should be treated upon the principles already laid down for the application of electricity in these disorders.

DISEASES OF THE MUSCLES. Certain *diseases of the muscles*, in the treatment of which the use of electricity is of value, must be referred to before closing this chapter; these are muscular rheumatism, arthritic atrophy, and the primary myopathies. The affection known as muscular rheumatism, notwithstanding our indefinite knowledge as to its actual character, is frequently very much benefited by the use of electricity. The localization of the affection has more influence upon the amount of benefit to be expected than the intensity, and the most striking results may be obtained in severe torticollis or lumbago, while myalgia of the pectoral muscles or of those in the vicinity of joints is often but slightly influenced. The treatment should consist in the production of thorough contractions of the affected muscles and excitation of the overlying skin. At best this treatment is very painful, but milder measures, as the use of the stabile galvanic current, are of avail only in such cases in which a superficial massage would bring about quicker results. Faradization, galvano-faradization, the use of the faradic brush or of the static spark are here the sovereign remedies.

Fresh cases may thus often be cured by a few applications, while chronic cases will require several weeks of daily treatment.

In the treatment of the *muscular atrophy* occurring in consequence of joint-lesions electricity is an important agent. By means of galvanic and faradic excitation the nutrition of the muscles is to be bettered, and thus an increase in their volume attained. The use of continuous weak galvanic currents is said to be of value, particularly in the treatment of these atrophies. The results of all treatment here will depend upon the condition of the joint; until the primary joint-lesion is cured little is to be expected from any treatment directed to the muscles themselves.

In the treatment of the *primary myopathies* as little has been attained by the use of electricity as by any other means; at the same time the systematic employment of this remedy in the shape of galvano-faradization of the muscles and of the motor points may aid in promoting the nutrition of the parts, and thus be instrumental in delaying their involvement by the atrophic process.

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